Exhibit 218

Updated and Expanded Swedish Cohort Study on Trichloroethylene and Cancer Risk

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There is limited evidence for mutagenicity and carcinogenicity of trichloroethylene (TRI) in experimental test systems. Whether TRI is a human carcinogen is unclear, however. This paper presents an update and extension of a previously reported cohort of workers exposed to TRI, in total 1670 persons. Among men (n = 1421), the overall standardized mortality ratio (SMR) and cancer morbidity ratio (SIR) were close to the expected, with SMR, 0.97; 95% confidence interval (CI), 0.86 to 1.10; and SIR, 0.96; 95% CI, 0.80 to 1.16, respectively. The cancer mortality was significantly lower than expected (SMR, 0.65; 95% CI, 0.47 to 0.89), whereas an increased mortality from circulatory disorders (cardiovascular, cerebrovascular) was of borderline significance (SMR, 1.17; 95% CI, 1.00 to 1.37). No significant increase of cancer of any specific site was observed, except for a doubled incidence of nonmelanocytic skin cancer without correlation with the exposure categories. In the small female subcohort (n = 249), a nonsignificant increase of cancer and circulatory deaths was observed (SMR, 1.53 and 2.02, respectively). For both genders, however, excess risks were largely confined to groups of workers with lower exposure levels or short duration of exposure or both. It is concluded that this study provides no evidence that TRI is a human carcinogen, ie, when the exposure is as low as for this study population.

Trichloroethylene (TRI) is a widely used solvent that may be found in degreasing processes in the metal manufacturing industry, in the production of rubber and plastics, sometimes in dry-cleaning, and as a solvent in glues and other products. Moreover, TRI has been used in anesthesia for surgery and obstetrics. Therefore, epidemiologic studies were called for when TRI in the mid-1970s was found to be mutagenic in bacterial test systems[1] and to cause an increase in the spontaneous rate of hepatocellular carcinoma especially in male mice, whereas no effect was seen in rats.[2] In subsequent studies with experimental test systems some additional but as yet inconclusive evidence for the mutagenicity and carcinogenicity of TRI has been obtained.[3,4]

A first cohort study of 518 men with exposure to TRI appeared from Sweden in 1978[5] and did not show any excess of cancer, nor did a subsequent and somewhat larger cohort of 2084 persons from Finland indicate any clear risk.[6] An update and expansion of the Swedish study through 1979 was presented in 1984, although in abstract form only.[7] Applying a 10-year latency requirement, this update showed some excess of incident urinary tract cancers (11 cases observed vs 4.85 expected) and hematolymphatic malignancies (5 vs 1.20), but there was a deficit in total cancer mortality with 22 observed cases versus 36.9 expected.

In a cohort study of 2646 employees from a manufacturing plant using TRI, there were fewer deaths than expected from heart disease, cancer, and trauma.[8] Loss to follow-up was less than 2%. With regard to cancer, there were only 21 cancer deaths observed

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versus 36.7 expected. No specific exposure data were available. The assemblers, who were assumed to have had the greatest opportunity for exposure to TRI, conformed generally to the expected deaths for all causes.

More recently, a cohort of 14,457 workers at an aircraft maintenance facility has been evaluated with regard to mortality. Of these employees, 6,929 had been exposed to TRI during the 1950s and 1960s.[9] No association was found between several measures of exposure to TRI and any clear excess of cancer. However, nonsignificant excess risks were noted for cancers of the biliary passages and liver among men, whereas in women nonsignificantly raised risk estimates were obtained for cancers of the cervix and the hematolymphatic system, particularly for non-Hodgkin's lymphoma. More specifically, women employed in departments where fabric cleaning and parachute repair operations were performed had more deaths than expected from multiple myeloma and non-Hodgkin's lymphoma, although this excess was not clearly related to TRI exposure.

There are some studies on liver cancer and exposure to TRI that have not indicated any association in this respect.[10–12] On the other hand, both a study of workers in the metal polishing and plating industry[13] and a study of jewelry workers[14] showed an excess of liver cancer, which was discussed as possibly due to TRI exposure. No supportive, specific exposure data seem to have been available, however. None of these studies, therefore, can be taken as indicative of any risk from exposure specifically to TRI.

In this paper we report an update and further expansion of the Swedish cohort study of workers exposed to TRI as presented in 1978 and 1984.[5,7] The expansion adds power to the present update, in particular because it includes more people who have reached a reasonable latency time since first exposure.

Materials and Methods

Technical Background

From 1930 to 1986, only one plant in central Sweden produced TRI for the domestic market. Originally, acetylene was used as raw material, but with the construction of new production facilities in 1970, it was substituted by ethene, which was considered to reduce impurities in the end product. TRI is sensitive to photochemical degradation, and therefore various stabilizers were added in the commercial product. The Swedish producer provided two slightly different TRI formulas on the market, "standard TRI" and "TRI-plus." "Standard TRI," the bulk TRI product, was stabilized with diisopropylamine and thymol, whereas "TRI plus," which was intended for degreasing of aluminum, contained butylene oxides and epichlorohydrin (until 1975) as stabilizers (R. Wettström, personal communication, 1988). With regard to the present study, it may be noted, however, that no information was available on the relative use of the different TRI formulas among the companies involved, but it is likely that standard TRI by far predominated the exposure of those involved in the cohort.

Cohort Ascertainment and Follow-Up

The general background of the present cohort has been presented in our first report on this issue.[5] It may be recalled here, however, that the Swedish producer offered the customers free surveillance of their exposed workers by analyses for a metabolite of TRI in urine, ie, trichloroacetic acid (U-TCA). The producer's files with data from this biologic monitoring constitute the basis for the cohort, but as reported in the 1978 paper, part of the earlier files had been destroyed. With the lapse of time, however, it has become interesting to expand the cohort by enrolling more people from the laboratory files, as they now have achieved a reasonable latency time from first exposure. To establish the new expanded cohort, 1727 exposed individuals were drawn from 115 different customer companies, which had used the surveillance service at least once between 1955 and 1975.

Because the laboratory files contained rather incomplete information for identification of the subjects, the employer was unable to identify 23 subjects and another four persons were not found in the population registers. Thirty people were found to have emigrated, so that the cohort finally encompassed 1670 persons: 1421 men and 249 women, ie, 96.7% of the subjects as available in the laboratory files.

Hence, with the losses mentioned above, the unique 10-digit personal identification number assigned to each resident in Sweden was obtained from the employer of each person who had undergone a U-TCA analysis. The identification numbers were checked in various population registers, corrected if necessary, and finally matched with the Swedish cause-of-death and cancer registers, respectively.

On an individual basis, the number and timing of the U-TCA analyses varied considerably, and two different summary exposure indices were primarily considered for each individual: the mean U-TCA value for all available urine samples and, alternatively, the highest mean U-TCA value for any consecutive 3-year period during 1955 to 1975. In the present study, the first-mentioned kind of exposure index was used. Also exposure time was considered and taken as the time interval from the year of the first found urine sample until the end of employment or 1979, whichever came first. The overwhelming majority of male subjects in the cohort had experienced a quite low TRI exposure with a mean U-TCA level below 50 mg/L in 81% of the cases, roughly corresponding to an average exposure level of 20 ppm or 110 mg/m^3 in the air.

The cohort obtained on the basis of the urine samples is of an oblique character,[15] the persons entering at different points in time between 1955 and 1975 but all with follow-up through 1986 regarding mortality. For cancer morbidity, the follow-up started with the cancer registry in 1958 and continued through 1987. For the analysis, the EPILIN software[16] was used. Observed events (deaths and incident cases of cancer) for men and women were compared with the expected number of outcome as derived from a computation of the number of person-years under observation and cause specific national

rates with regard to 5-year age groups and calendar years. Risk estimates were calculated as standardized mortality and cancer incidence ratios (SMRs and SIRs, respectively), and the corresponding 95% confidence intervals (CI) were obtained by assuming a Poisson distribution of the events. In the analysis, only the person-years and events before the age of 80 were accounted for because higher age groups were considered to entail less accurate diagnoses.

A remark may be justified here also, namely that the Swedish cancer register has used a coding to the 7th Revision of the International Classification of Diseases (ICD 7), but in 1975, all lymphohematopoietic cancers were reclassified to meet the standards of the 8th revision of the ICD. The cause-of-death register, on the other hand, was coded to the ICD 6, 7, or 8 during subsequent phases of the observation period (1955 to 1986). The coding used for the individual cases by the Swedish cancer registry and by the National Central Bureau of Statistics was obtained and applied in the study so as to provide for accurate comparability with the national rates.

Results

Male Mortality

The overall male cohort provided 22,446.5 person-years at observation and the total mortality was close to the expected with an SMR of 0.97 (95% CI, 0.86 to 1.10), whereas the total cancer mortality was significantly lower than expected (SMR, 0.65; 95% CI, 0.47 to 0.89). The mortality from diseases of the circulatory system, on the other hand, was slightly increased, reaching borderline statistical significance (SMR, 1.17; 95% CI, 1.00 to 1.37). To obtain indications of any dose-response pattern within the cohort, if present, three subgroups were constructed according to the overall mean U-TCA (Table 1). The most notable finding was the very low cancer mortality in the two lowest exposure groups, whereas in the "100+" group, the SMR for cancer was slightly increased. However, the point estimate (SMR 1.05) was based on only 1763.5 person-years under observation. The circulatory mortality was of a similar magnitude regardless of the average TRI exposure level as reflected by the U-TCA values.

Considering exposure levels in terms of mean U-TCA as well as exposure time, as defined above, it appeared that the subcohort with an exposure time of less than 2 years had slightly higher overall mortality than those with 2 years or more of TRI exposure (Table 2). An excess mortality was particularly evident among those with the shortest exposure time and lowest mean U-TCA level (−49 mg/L) who had an overall SMR of 1.49 (95% CI, 1.13 to 1.97) mainly caused by a significant increase of deaths from diseases of the circulatory system (SMR, 1.79; 95% CI, 1.23 to 2.57). Except for the group of subjects with a mean U-TCA of 50 to 99 mg/L, the cancer mortality for those with short exposure time was about as high as for those with at least 2 years of TRI exposure. Applying a latency time requirement of 10 years to the subcohort with at least 2 years of exposure resulted in slightly higher point estimates for overall mortality, cancer mortality, and circulatory mortality for those with 100+ mg/L U-TCA than in the lower exposure groups. The figures were based on small numbers of observed events, however, and the deviations from unity were not statistically significant.

Male Cancer Morbidity

The overall cancer morbidity among the men of the TRI cohort was slightly lower than expected from national incidence rates (107 observed cases; SIR, 0.96; 95% CI, 0.80 to 1.16; Table 3). Regarding specific cancer sites, a statistically significant increase of malignant skin tumors (all were squamous cell carcinomas) was found with eight observed cases versus 3.4 expected. There was also some excess of cancer of the liver, the larynx, the prostate and of non-Hodgkin lymphoma but for all sites the confidence intervals of the SIR included unity.

The excess of liver, skin, and pros-

TABLE 1
Mortality from Selected Causes 1955–1986 in TRI-exposed Men ≤ 79 y by Mean U-TCA

Cause	U-TCA Mean (mg/L)							
	−49		50–99		100+		All	
	Obs	SMR*	Obs	SMR	Obs	SMR	Obs	SMR
All	170	0.98 (0.85–1.13)	40	0.93 (0.68–1.27)	19	0.96 (0.59–1.49)	229	0.97 (0.86–1.10)
Malignant tumors	28	0.67 (0.46–0.96)	4	0.39 (0.11–0.99)	5	1.05 (0.34–2.44)	37	0.65 (0.47–0.89)
Circulatory system	102	1.18 (0.98–1.43)	24	1.09 (0.72–1.62)	12	1.22 (0.63–2.12)	138	1.17 (1.00–1.37)
Respiratory system	6	0.76 (0.28–1.65)	2	0.97 (0.12–3.50)	0	– (0.00–4.07)	8	0.74 (0.32–1.45)
Digestive system	7	0.98 (0.39–2.01)	2	1.16 (0.14–4.19)	0	– (0.00–4.47)	9	0.93 (0.42–1.76)
Violence + intoxications	20	1.12 (0.70–1.73)	6	1.70 (0.62–3.68)	2	1.08 (0.13–3.87)	28	1.21 (0.82–1.74)
Person-years		17,466		3,217		1,763.5		22,446.5

* Standardized mortality ratio (95% confidence interval).

TABLE 2
Mortality from Selected Causes 1955–1986 in TRI-exposed Men ≤ 79 y by Exposure Time and Mean U-TCA Level

Cause	Exposure Time (y)																	
	<2							≥2										
	0–49		50–99		100+		All		–49		50–99		100+		All			
	Obs	SMR*	Obs	SMR	Obs	SMR	Obs	SMR	Obs	SMR	Obs	SMR	Obs	SMR	Obs	SMR		
All	49	1.49 (1.13–1.97)	11	1.13 (0.56–2.09)	7	1.02 (0.41–2.09)	67	1.36 (1.08–1.72)	121	0.86 (0.73–1.02)	29	0.88 (0.60–1.25)	12	0.93 (0.48–1.61)	162	0.87 (0.75–1.01)		
Malignant tumors	8	1.04 (0.45–2.04)	0	– (0.00–1.59)	2	1.21 (0.15–4.37)	10	0.86 (0.41–1.57)	20	0.56 (0.37–0.90)	4	0.50 (0.14–1.27)	3	0.96 (0.20–2.80)	27	0.60 (0.40–0.86)		
Circulatory system	29	1.79 (1.23–2.57)	7	1.42 (0.57–2.92)	5	1.50 (0.49–3.49)	41	1.68 (1.24–2.27)	73	1.04 (0.83–1.30)	17	1.00 (0.59–1.59)	7	1.07 (0.43–2.20)	97	1.03 (0.86–1.26)		
Respiratory system	4	2.62 (0.71–6.70)	2	4.29 (0.52–15.48)	0	– (0.00–12.08)	6	2.61 (0.96–5.67)	2	0.32 (0.04–1.13)	0	– (0.00–2.31)	0	– (0.00–6.13)	2	0.24 (0.03–0.84)		
Digestive system	1	0.76 (0.02–4.22)	0	– (0.00–9.40)	0	– (0.00–12.51)	1	0.50 (0.01–2.77)	6	1.04 (0.38–2.23)	2	1.51 (0.18–5.42)	0	– (0.00–6.97)	8	1.04 (0.45–2.04)		
Violence + intoxications	3	0.80 (0.16–2.33)	2	2.24 (0.27–8.09)	0	– (0.00–5.03)	5	0.93 (0.30–2.17)	17	1.21 (0.72–1.93)	4	1.51 (0.41–3.86)	2	1.77 (0.21–6.37)	23	1.29 (0.84–1.93)		
Person-years		3,851.5		850.5		704.5		5,406.5		13,614.5		2,366.5		1,059		17,040		

* Standardized mortality ratio (95% confidence interval).

tatic cancers, as well as of lymphomas, occurred essentially in the low exposure group (U-TCA −49 mg/L). Considering exposure time (Table 4), this excess was found among those with a minimum of 2 years of TRI exposure, a finding that remained also when 10 years of latency from first exposure was required (Table 5). This was most evident for the skin cancers (SIR, 3.65; 95% CI, 1.19 to 8.52).

Female Mortality and Cancer Morbidity

The female section of the study included only 249 women, consequently the informative value was considerably less than for the men. The overall female mortality (Table 6), however, was significantly increased (in total 24 observed cases; SMR, 1.55; 95% CI, 1.02 to 2.31). The excess was due to a nonsignificant increase of deaths in malignant tumors (SMR, 1.53) and circulatory diseases (SMR, 2.02). The excess of cancer deaths occurred particularly in the exposure group with U-TCA 50–99 mg/L (4 observed cases; SMR, 3.79, 95% CI, 1.03 to 9.68). However, the excess risk was confined to a very small group of nine women with less than 2 years of known TRI exposure (3 observed cases; SMR, 11.21; 95% CI, 2.42 to 34.21). The increase of circulatory mortality was particularly prominent in the group with the lowest TRI exposure (mean U-TCA −49 mg/L) (9 observed cases; SMR, 2.42, 95% CI, 1.10 to 4.58). Most of these cases (7 out of 9) had an exposure time of at least 2 years; with 10 years of latency the SMR dropped to 1.63 (5 observed cases; 95% CI, 0.53 to 3.79).

The overall female cancer morbidity was slightly higher than expected, but the excess risk was not statistically significant (22 observed cases; SIR, 1.32; 95% CI, 0.85 to 1.99). Half the cases were tumors of the breast and genital organs and most of the other cancers (8 of 11) were located in the gastrointestinal tract. There were no cases of liver cancer, nor were there any cases of skin cancer, lymphoma, or leukemia among the women.

The excess of female cancer cases

TABLE 3
Overall Cancer Morbidity 1958–1987 in TRI-exposed Men ≤ 79 y

ICD-7	Site	Observed	Expected	SIR*
140–209	All	107	111.9	0.96 (0.80–1.16)
151	Stomach	5	7.2	0.70 (0.23–1.62)
153	Colon	8	7.8	1.04 (0.44–2.03)
155	Liver	4	2.8	1.41 (0.38–3.60)
157	Pancreas	1	4.1	0.25 (0.01–1.38)
161	Larynx	2	1.4	1.39 (0.17–5.00)
162	Lung	9	13.2	0.69 (0.31–1.30)
177	Prostate	26	20.7	1.25 (0.84–1.84)
178	Testis	2	1.0	2.03 (0.25–7.31)
180	Kidney	6	5.2	1.16 (0.42–2.52)
181	Bladder	8	7.9	1.02 (0.44–2.00)
191	Skin	8	3.4	2.36 (1.02–4.65)
200	Non-Hodgkin's lymphoma	5	3.2	1.56 (0.51–3.64)
201	Hodgkin's lymphoma	1	0.9	1.07 (0.03–5.95)
202	Other lymphoma	0	0.1	– (0.00–33.72)
203	Multiple myeloma	1	1.8	0.57 (0.01–3.17)
	Other	21	31.2	0.67 (0.43–1.03)
Person-years			23,516.5	

* Standardized incidence ratio (95% confidence interval).

was confined to the group with less than 2 years of known exposure to TRI (9 observed cases; SIR, 2.55; 95% CI, 1.16 to 4.84) and these cases were equally distributed between the two lowest exposure categories (U-TCA −49 mg/L and 50 to 99 mg/L, respectively).

Discussion

The results obtained in the present study do not seem to indicate any increased cancer risk from exposure to TRI per se. An excess of cancer was noted for some sites, but was associated with low grade and short time exposure rather than with more heavy exposure, which would have been suggestive of a causal relationship. The reason for the significant excess of skin cancer among males with relatively low exposure is unclear, but it is not unlikely that some workers working with degreasing also could have had exposure to mineral oils and therefore might have developed skin cancer. If such exposures have occurred and to what extent there could possibly be an interaction with TRI exposure cannot be evaluated from the available information, however.

The women of the cohort experienced a generally more unfavorable health outcome than the men. With regard to the cancer panorama, however, there was little congruity between the genders, making an influence from their TRI exposure less likely. Similarly as among the men, the most unfavorable results were obtained among those women who had either low-level or short-time TRI exposure or both, which would suggest other risk factors than TRI to operate, possibly of socioeconomic nature.

In view of the nonpositive result obtained some considerations would be justified regarding such biases that could mask an increased risk. Hence, selectional phenomena may sometimes occur and be of such character that an effect is overlooked, eg, if deceased workers would be sorted out of the records used for establishing a cohort. Nothing such is likely to have occurred in this context, however, and it should be emphasized that the sorting out and destruction of some of the early laboratory files had no relationship to health events or to degree of exposure in terms of U-TCA analyses.

It is worth noting that the cohort members had to be followed from first found urine sample rather than from first employment. If we had started the enrollment of subjects from the date of first employment, this would have meant that only subjects surviving until urine sampling would have been included. Other exposed employees, who might have died or left the job because of disease, would not have contributed the expected numbers; even more important, they would not have appeared among the observed cases. Nor can it be known whether there were more exposed employees or not in the consumer companies. Consequently, the best approach was to establish the cohort on the basis of those workers for whom the records had survived and to follow them only forward from the point in time when first known to have had exposure.

One might also consider the possibility of some negatively confounding factor, that could eliminate an effect. There are no obvious such factors, however, which also would have to be fairly strong in order to eliminate an effect if actually present; the confounding effect from even rather strong factors tends to be quite modest in studies of this character.[17] Another remark in this context might be that the so called healthy worker effect is relatively weak in this study with regard to total mortality. A weak effect in this respect is not unusual in Swedish cohort studies, however, and the traditionally low unemployment rate

TABLE 4
Cancer Morbidity of Selected Sites 1958–1987 in TRI-exposed Men ≤ 79 y by Exposure Time and Mean U-TCA Level

Cause	Exposure Time (y)																	
	<2							≥2										
	0–49		50–99		100+		All	0–49		50–99		100+		All				
	Obs	SIR*	Obs	SIR	Obs	SIR	Obs	SIR	Obs	Obs	SIR	Obs	SIR	Obs	SIR	Obs	SIR	Obs	SIR
All	15	0.99 (0.57–1.64)	2	0.47 (0.06–1.68)	6	1.89 (0.69–4.11)	23	1.02 (0.66–1.53)	62	0.90 (0.71–1.16)	16	1.07 (0.62–1.73)	6	1.05 (0.39–2.28)	84	0.94 (0.77–1.16)		
Liver	0	– (0.00–9.67)	0	– (0.00–33.09)	1	12.73 (0.33–70.92)	1	1.75 (0.05–9.75)	3	1.74 (0.36–5.04)	0	– (0.00–9.41)	0	– (0.00–25.21)	3	1.32 (0.27–3.85)		
Prostate	4	1.43 (0.39–3.66)	0	– (0.00–4.39)	1	1.79 (0.05–9.94)	5	1.19 (0.39–2.78)	16	1.28 (0.75–2.09)	3	1.02 (0.21–2.96)	2	1.81 (0.22–6.54)	21	1.27 (0.81–1.94)		
Skin	1	2.19 (0.06–12.16)	0	– (0.00–27.40)	1	10.51 (0.27–58.55)	2	2.91 (0.35–10.50)	6	2.91 (1.07–6.34)	0	– (0.00–7.93)	0	– (0.00–21.43)	6	2.22 (0.82–4.84)		
Non-Hodgkin's lymphoma (ICD 200, 202)	1	2.17 (0.06–12.11)	0	– (0.00–28.38)	0	– (0.00–33.54)	1	1.47 (0.04–8.19)	3	1.44 (0.30–4.20)	0	– (0.00–8.58)	1	6.25 (0.16–34.83)	4	1.50 (0.41–3.85)		
Person-years		4,050.5		882		724.5		5,657		14,328		2,442		1,089.5		17,859.5		

* Standardized incidence ratio (95% confidence interval).

in Sweden, in the past usually around 2%, might be the reason for this, ie, also people with a less favorable life prognosis have been employed to a great extent. In the context of a non-positive study, there is always the concern about the healthy worker effect as masking a risk. This effect is less pronounced with regard to cancer and therefore hardly any pertinent argument when considering the results of this study.

The main limitation of this study may be found in the relatively low average exposure of the subjects involved. The exposure is likely to have occurred mainly through inhalation but some skin contact might have taken place as well. Regarding the results of the analyses, it seems quite reasonable that the rather low exposure (<20 ppm) for most of the workers included in the cohort should not be expected to bring about much of a visible cancer risk, even if there would be a hazard in this respect. The data presented here certainly do not rule out a cancer effect from TRI in humans. However, a reasonable conclusion, based on the present study as well as the investigations referred to in the Introduction, seems to be that the cancer risk to humans from TRI exposure is rather small, if any, under the circumstances that have prevailed when using TRI.

The traditionally low Swedish standard for TRI exposure at workplaces (30 ppm), as implemented on technical grounds (and by now even further lowered to 10 ppm or 50 mg/m^3), has obviously been achievable in view of the low exposure levels indicated for most persons in this cohort by the U-TCA monitoring. Indeed, a recent cross-sectional study of TRI exposure during metal degreasing indicates that the vast majority of modern metal manufacturing shops in Sweden has no problem to comply even with the present, quite low standard for TRI in this country.[18]

From the point of view that no pertinent excess of cancer has been detected in this material, nor in any other study, it seems as if TRI in the future could be a reasonably safe choice from among the various chlorinated solvents, especially if suitable

TABLE 5
Cancer Morbidity of Selected Sites 1958–1987 in TRI-exposed Men ≤ 79 y with ≥ 2 y of Exposure and 10 y of Latency by Mean U-TCA

Cause	U-TCA Mean (mg/L)							
	−49		50–99		100+		All	
	Obs	SIR*	Obs	SIR	Obs	SIR	Obs	SIR
All	41	1.02 (0.75–1.38)	11	1.10 (0.55–1.96)	6	1.54 (0.56–3.35)	58	1.07 (0.84–1.38)
Liver	2	1.89 (0.23–6.83)	0	– (0.00–13.52)	0	– (0.00–35.52)	2	1.40 (0.17–5.04)
Prostate	11	1.30 (0.65–2.33)	2	0.91 (0.11–3.28)	2	2.40 (0.29–8.67)	15	1.31 (0.74–2.16)
Skin	5	3.65 (1.19–8.52)	0	– (0.00–10.73)	0	– (0.09–28.69)	5	2.72 (0.88–6.34)
Non-Hodgkin's lymphoma (ICD 200, 220)	2	1.64 (0.20–5.92)	0	1.16 (0.00–12.72)	1	8.33 (0.22–46.43)	3	1.85 (0.38–5.41)
Person-years		5,696.5		1,193.5		562.5		7,452.5

* Standardized incidence ratio (95% confidence interval).

TABLE 6
Overall Mortality 1955–1986 in TRI-exposed Women ≤ 79 y

	Cause	Observed	Expected	SMR*
000–999	All	24	15.4	1.55 (1.02–2.31)
140–209	Malignant tumors	10	6.6	1.53 (0.73–2.81)
390–458	Circulatory system	10	5.0	2.02 (0.97–3.71)
	Other	4	4.0	1.01 (0.28–2.59)
Person-years		3,691.5		

* Standardized mortality ratio (95% confidence interval).

alternatives are lacking. The relative instability of the chemical might also favor its use in view of the adverse effect on the ozone layer of some of the more stable organochlorine compounds.

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