Retrospective Cohort Mortality Study of Dry Cleaner Workers Using Perchloroethylene

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To evaluate the carcinogenic potential from occupational exposure to perchloroethylene (PCE), a retrospective cohort mortality study of workers employed in the dry cleaning industry was conducted among 1,690 workers from four labor unions. The majority of the cohort had potential exposure to petroleum solvents as well as to PCE while working in the dry cleaning industry. Mortality from primary cancer of the liver was of particular interest, due to the findings of excess liver cancer in mice exposed to PCE. Other sites of cancer were also of interest.

A total of 493 deaths were observed, whereas 575.5 were expected based on US mortality rates. Mortality from all cancers combined was greater than expected (142 observed v 122.9 expected). No deaths due to liver cancer were observed. Urinary tract cancer was the only specific site where there was a statistically significant excess in observed deaths (12 observed v 4.7 expected). There was some consistency in these findings across the four individual unions and across race/sex groups. A subcohort of workers who were employed only in dry cleaning shops that used PCE as their primary solvent was identified from the union records. There was only one death from urinary tract cancer, whereas 1.3 deaths were expected in this subcohort.

Perchloroethylene (PCE), also known as tetrachloroethylene, is a solvent used commonly for cleaning fabric (dry cleaning) and for degreasing metals. It has been estimated that at least 1.6 million workers in the United States are potentially exposed to PCE.[1] The possibility that PCE could pose a significant occupational health risk to these workers was raised after a National Cancer Institute (NCI) bioassay indicated that PCE induced liver tumors in exposed mice.[2] In addition, toxic tubular nephropathy was observed in the treated mice.

In an unpublished study conducted by The Dow Chemical Company in 1977, there was no evidence of a tumorogenic response in rats exposed by inhalation to PCE. There was increased mortality among the rats in the high-dose group.

The National Toxicology Program (NTP)[3] recently completed a study where F344/N rats and BGC3F, mice were exposed to PCE by inhalation. PCE produced renal tubular cell karyomegaly, which is an abnormal enlargement of the cell nucleus, and renal tubular cell hyperplasia in rats. It also increased the incidence of renal tubular cell adenomas or adenocarcinomas in male rats. Both low and high doses of PCE were associated with an increased incidence of mononuclear cell leukemia in male rats and in female rats the low dose increased the incidence of leukemia. In mice there was a dose-related increase in the incidence of hepatocellular neoplasms. PCE also produced renal tubular cell karyomegaly in mice.

Due to the potential carcinogenic effect of PCE as demonstrated by the animal studies and due to the widespread use of PCE in the workplace, an epidemiologic retrospective cohort mortality study was conducted to examine the effects on exposed workers, particularly the risk of mortality from cancer. After evaluating the numerous occupational groups potentially exposed to PCE, the dry cleaning industry was chosen for the epidemiologic study, because PCE had been used as a cleaning solvent in this industry for at least 30 years.

PCE was introduced into the dry cleaning industry in the late 1930s but did not replace other synthetic sol-

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vents such as carbon tetrachloride until shortly after world War II. During this same period petroleum derivatives (primarily various types of Stoddard solvents) were the predominant solvents used in dry cleaning. A gradual shift from petroleum derivatives to PCE began in the late 1940s. This shift in solvents increased in the 1950s and early 1960s. However, in the period before 1960, petroleum derivatives were still the dominant solvents. By 1977, the industry estimated that approximately 74% of commercial dry cleaning shops used PCE, about 24% used petroleum solvents, and the remainder used fluorocarbons (personal communication from William Fisher, International Fabricare, 1985).

An exposure evaluation survey, which included a random sample of the facilities (still in business) in the epidemiologic study, was conducted from 1977 to 1979.[4] There were 44 commercial dry cleaning shops included in the survey. Time-weighted average (TWA) and peak exposures to PCE were determined by collecting personal air samples. Other solvents used for spot removal were also sampled. The operator or dry cleaner had exposures significantly higher than the other workers (geometric mean TWA of 22 ppm *v* approximately 3.0 ppm, respectively). Based on historical exposure data some of which date back to 1956, the levels of exposure to PCE in commercial dry cleaning shops have remained fairly constant since its introduction into the industry. The survey also revealed that there was consistency in the level of exposures by geographic location. The only substance detected in the air samples during the survey was PCE. Therefore, even though other solvents were used for spot removal, their airborne concentrations were nondetectable.

Methods

The study cohort was defined to include workers exposed to PCE for a minimum of 1 year prior to 1960, and with no known previous occupational exposure (in the dry cleaning industry) to carbon tetrachloride or trichloroethylene.

Records maintained by four local unions were used to identify dry cleaner workers who met the definition of the study cohort. The majority, if not all, of the dry cleaning shops were commercial as opposed to industrial cleaners. Workers were chosen only when there was documentation that they were employed for at least 1 year prior to 1960 at a shop where PCE was the primary solvent. Some and/or a complete solvent history was available for approximately half of the shops employing union members. If no solvent history was available for a particular shop, employment in that shop was not considered in determining the eligibility of union members. For each eligible worker a history of employment in PCE as well as non-PCE dry cleaner shops was coded. If solvent history was unknown prior to 1960, it was assumed to be non-PCE inasmuch as most shops used petroleum solvents prior to this date.

In an attempt to restrict an analysis to a cohort of workers primarily exposed to PCE with no confounding exposure to petroleum solvents, a subcohort of workers who were known to be employed only in shops where PCE was the primary solvent was identified.

The vital status of cohort members was determined as of Dec 31, 1982. For those identified as deceased, copies of their death certificates were obtained and the underlying cause of death was coded by a trained nosologist according to the Revision of the International Classification of Diseases (ICD) in effect at the time of death. Those lost to follow-up (unknown vital status) and those who died subsequent to the closing date of the study, ie, Dec 31, 1982, were considered alive for purposes of analysis.

Person-years at risk (PYAR) were calculated for each worker starting after 1 year of employment in a PCE shop(s) and ending at the date of death or the closing date of the study, whichever occurred first. Using a life-table analysis system,[5] the PYAR for each worker were combined into 5-year calendar time periods and 5-year age groups. PYAR were additionally distributed by length of employment and by time since first employment in PCE shops (latency). Employment in unknown or petroleum solvent shops was not used in the calculations of length of employment or latency.

The PYAR stratified into age and calendar time periods were multiplied by the corresponding US mortality rates to yield expected numbers of deaths. At the time of this study, the life-table analysis system only maintained US mortality rates through 1978, the end of the eighth revision of the ICD. To calculate expected deaths through 1982 for this study the death rates for the interval 1975 to 1979 were based on US deaths occurring through 1978, and the death rates for the interval 1980 to 1982 were assumed to be identical to the previous time period (1975 to 1979).

The observed and expected cause-specific deaths were compared and differences were tested assuming the Poisson distribution.[6] The risk is reported as a standardized mortality ratio (SMR), defined as observed/expected deaths × 100.

Because each of the unions included in the study were located in large metropolitan cities where the mortality rates from cancer are generally higher than those of the total US, state mortality rates corresponding to the location of each union were also used in calculating expected cancer deaths. The state mortality rates more closely estimate the rates of the cities.

Results

The cohort totaled 1,690 workers with 493 deaths and contributed 42,267 PYAR to the analysis. The vital status follow-up through Dec 31, 1982, was successful for 93% (ie, 7% lost to follow-up) of the cohort. The follow-up for females (92%) was much less complete than for males (97%). This is primarily due to name changes and women dropping out of the work force at an earlier age. Therefore, they are less likely to be listed in the Social Security Administration records.

The subcohort of workers employed only in shops

where PCE was the primary solvent totaled 615: 113 white males, 94 non-white males, 195 white females and 213 nonwhite females. There were a total of 137 deaths identified in this subcohort.

Although all sites of cancer and particularly cancer of the liver were the causes of greatest interest, multiple causes of death were also examined. Table 1 lists the observed and expected deaths for most major diseases and for specific sites of cancer. As seen in most cohort studies of workers, the observed number of deaths for all causes is less than expected (493 observed *v* 575.5 expected; SMR = 86) with statistically significant deficits in diseases of the circulatory system and diseases of the nervous system (primarily stroke), demonstrating the healthy worker effect. The number of observed deaths for all neoplasms is higher than expected (142 observed *v* 122.9 expected; SMR = 116). No deaths from liver cancer were observed, whereas 3.5 were expected. Only urinary tract cancer showed a statistically significant excess in observed deaths (12 observed *v* 4.7 expected; SMR = 255). Within the urinary tract cancer category, both kidney and bladder cancer were elevated with bladder cancer being statistically significant (8 observed *v* 2.7 expected; SMR = 296).

Other interesting results among the malignant neoplasms include cancer of the cervix uteri which shows an elevated SMR (10 observed *v* 5.1 expected; SMR = 196) and cancer of the breast which is slightly lower than expected (12 observed *v* 13.8 expected; SMR = 87). These results probably reflect the socioeconomic status of the cohort which is generally lower income. This type of pattern has been shown in other studies.[7]

In the analysis of cancer mortality based on death rates of the state where each union was located (rather than the US death rates), there are 140.2 expected deaths from all cancer sites which is closer to the 142 deaths observed in the study population. However, except for pancreatic cancer (11 observed *v* 10.7 expected), there was very little change in the expected number of deaths from the specific sites of interest. For urinary tract cancer the expected number of deaths based on state rates is 4.9, *v* 4.6 based on US rates. Therefore, all remaining results in this manuscript are based on expected deaths calculated using US death rates.

Mortality from all major organ systems (other than cancer) was lower than expected, except for a slight excess in diseases of the digestive system (22 observed *v* 18.8 expected; SMR = 117). Mortality from all accidents is significantly lower than expected (3 observed *v* 23.9 expected; (SMR = 13). Almost all categories of accidents showed a deficit in mortality, with transportation accidents accounting for the largest portion of the overall category (0 observed *v* 10.7 expected).

Mortality by race and sex group was examined separately. For all groups, overall mortality was lower than expected, and cancer mortality was higher than expected. In nonwhite males the excess in cancer mortality was statistically significant. In three out of the four race/sex groups mortality from bladder and kidney cancer was elevated, although these were based on small numbers of observed and expected deaths. For bladder cancer a statistically significant excess was observed in nonwhite males (3 observed *v* 0.6 expected; SMR = 500). Breast cancer was lower in both racial groups and cervix uteri cancer was higher than expected. In all four groups mortality from accidents was significantly lower than expected. In white males there was an un-

TABLE 1
Cause-specific Mortality of Dry Cleaner Workers From All Four Unions Included in Cohort (All Race and Sex Groups)*

Cause	Observed	Expected	SMR	95% CI
All malignant neoplasms (MN)	142	122.9	116	97–136
MN of buccal cavity and pharynx	3	2.9	103	21–302
MN of digestive organs and peritoneum	38	35.5	107	76–147
MN of intestine except rectum	16	11.8	136	78–220
MN of liver	0	3.5	—	—
MN of pancreas	11	6.4	172	86–310
MN of respiratory system	29	25.4	114	76–164
MN of breast	12	13.8	87	45–152
MN of female genital organs	16	12.7	126	72–204
MN of cervix uteri	10	5.1	196	95–363
MN of male genital organs	8	5.5	145	63–286
MN of urinary organs	12	4.7	255	132–450
MN of kidney	4	2.0	200	55–517
MN of bladder	8	2.7	296	128–586
MN of other and unspecified sites	20	13.1	153	93–236
MN of lymphatic and hematopoietic tissue	4	9.3	43	12–110
Diseases of the nervous system	44	60.5	73	53–98
Diseases of the circulatory system	163	232.0	70	60–82
Diseases of the respiratory system	23	28.7	80	51–120
Diseases of the digestive system	22	18.8	117	73–177
Cirrhosis of the liver	14	12.9	109	60–183
Diseases of the genitourinary system	10	10.2	98	47–180
Calculi of the urinary system	2	0.3	667	73–2186
Accidents	3	23.9	13	3–37
Violence	10	12.4	81	39–149
All deaths	493	575.5	86	78–94

* Abbreviations used are: SMR, standardized mortality ratio, CI, confidence interval.

usual finding within diseases of the genitourinary system; a statistically significant excess in mortality from calculi of the urinary system was found (2 observed *v* 0.09 expected).

Mortality was also examined across individual unions which represent totally independent cohorts. Consistent with the findings of the combined cohort, a deficit in overall mortality and an excess in cancer mortality was noted across all four unions. There was also a deficit in accidents in all four unions. In three of the four unions there was an excess in bladder cancer, two of which were statistically significant. Two of the four unions had an excess in kidney cancer. All unions had an increase in cancer of the intestine. The SMRs by race and sex groups and by union for selected causes of death are summarized in Tables 2 and 3.

Analyses for cancer of the intestine and bladder by length of employment in PCE shops (a surrogate of exposure) and by time since first exposure (latency) were conducted to aid in the interpretation of the results. These analyses are given in Tables 4 and 5. There appears to be a positive trend of increasing risk with an increase in latency and exposure for both cancer sites. Separate analyses for kidney cancer are not presented because of the small numbers.

In the subcohort of workers who were employed only in shops where PCE was the primary solvent, there was only one death from urinary tract cancer, whereas 1.3 were expected. Both of the deaths from renal calculi were included in this subcohort, whereas only 0.09 deaths were expected.

An attempt was made to confirm the cause of death for each of the bladder and kidney cancer cases. Medical histories and pathology reports were requested from the hospital where the death occurred. Information was obtained for six of the eight bladder cancers and one of the four kidney cancers. The cause of death from the death certificate was confirmed in each case where information was available from the hospital.

Discussion

The diseases of concern in this study were all sites of cancer, especially cancer of the liver. Liver cancer was of particular interest because of the NCI bioassay and the recent NTP study which demonstrated that mice exposed to PCE developed liver tumors. The NTP study also demonstrated the PCE may be associated with renal carcinogenicity and leukemia in rats. Because it is not clear how an animal carcinogen might express itself as a human carcinogen, all other sites of cancer were of interest as well.

Mortality from all causes was found to be less than expected, which is probably due to the "healthy worker effect." Mortality from all cancers was higher than expected, almost reaching statistical significance—95% confidence interval for the SMR was 97 to 136. This excess in all cancers was a consistent finding across all race and sex groups and all unions; however, this excess was reduced when state mortality rates were used for calculating expected deaths.

Urinary tract cancer was the only specific site found to have a statistically significant excess in observed deaths. This excess was primarily due to bladder cancer; however, kidney cancer was also found in excess. There was some consistency in this finding in that bladder cancer was elevated in three out of the four race/sex groups, and in three out of the four unions, although

TABLE 3
Standardized Mortality Ratios for Selected Causes by Union

Cause of Death	Union 1	Union 2	Union 3	Union 4
All causes	71	83	84	96
All malignant neoplasms	132	124	103	106
MN of intestine	167	115	136	156
MN of pancreas	167	214	83	167
MN of kidney	—†	222	—	333
MN of bladder	1,000*	416*	200	—
Accidents	—	10*	—	26*

* $P < .05$.
† — = no observed deaths.

TABLE 4
Mortality From Cancer of the Intestine (Except Rectum) and Cancer of the Bladder by Time Since First Employment (Latency) in Perchloroethylene Shops*

Latency (yr)	MN of Intestine Observed/Expected (SMR)	MN of Bladder Observed/Expected (SMR)
<10	0/1.8 —	0/0.4 —
10–19	6/3.9 (154)	0/0.9 —
20–29	10/4.9 (204)	5/1.1 (455)
≥30	0/1.2 —	3/0.3 (1,000)
Total	**16/11.8 (136)**	**8/2.7 (296)**

* Abbreviations used are: MN, malignant neoplasm; SMR, standardized mortality ratio.

TABLE 2
Standardized Mortality Ratios for Selected Causes by Race and Sex Groups of Dry Cleaner Workers

Cause of Death	White Males	White Females	Nonwhite Males	Nonwhite Females
All causes	85	96	88	75
All malignant neoplasms	107	107	145*	110
MN of intestine	66	136	313	111
MN of pancreas	111	167	143	286
MN of kidney	143	333	—†	333
MN of bladder	273	400	500*	—
MN of breast	—	89	—	88
MN of cervix	—	188	—	206
Accidents	—	—	36*	—

* $P < .05$.
† — = no observed deaths.

TABLE 5
Mortality From Cancer of the Intestine (Except Rectum) and Cancer of the Bladder by Length of Employment in Perchloroethylene Shops*

Length of Employment (yr)	MN of Intestine Observed/Expected (SMR)	MN of Bladder Observed/Expected (SMR)
1–4	6/6.1 (98)	1/1.4 (71)
5–9	5/3.1 (161)	4/0.7 (571)
10–14	5/1.7 (294)	2/0.4 (500)
≥15	0/0.9 —	1/0.2 (500)
Total	**16/11.8 (136)**	**8/2.7 (296)**

* Abbreviations used are: MN, malignant neoplasm; SMR, standardized mortality ratio.

these were generally based on small numbers of observed and expected deaths. Kidney cancer was also elevated in three out of the four race/sex groups and two out of the four unions. It is interesting that both kidney and bladder cancer were found in excess. It can only be speculated that these two cancer sites are related to a common etiology. In studies of cigarette smokers,[8] and of workers exposed to benzidine[9], both sites were elevated.

When mortality from bladder cancer was examined by latency and exposure (based on employment in PCE facilities) a pattern consistent with an occupational etiology was found. The increased risk for bladder cancer mortality occurred after 20 years of latency which is similar to other studies of known bladder carcinogens.[10]

In this study the calculation of both exposure and latency associated with PCE are only estimates, because it was assumed that petroleum solvents were used during time periods of unknown solvent use and these time periods were not used in calculating exposure or latency. For some plants this assumption may be incorrect and PCE rather than petroleum solvents could have been used. This would tend to increase latency and exposure for the cohort. However, the bias that this creates depends on whether the bladder cancer deaths are affected more or less than others in the cohort.

In the analysis of workers employed only in shops where PCE was the primary solvent, there was no excess risk in mortality from bladder or kidney cancer. Therefore, the excess risk occurred in workers with a potential for mixed exposures to PCE and petroleum solvents. This finding does not preclude PCE as the exposure associated with the excess in urinary tract cancer but certainly weakens the possibility of an association. In addition, there is experimental evidence that kidney cancer may be related to exposure from petroleum solvents. Recently, in a study by Kitchen,[11] vaporized, unleaded gasoline induced kidney cancer in exposed Fisher 344/N rats. The chemical structure and toxicologic properties of Stoddard solvent, the petroleum solvent used in dry cleaning, is similar to those of gasoline.[12]

Cigarette smoking has also been associated with an excess risk of developing bladder cancer. Most studies have shown a twofold to fourfold excess risk of bladder cancer in male smokers compared to that in nonsmokers.[10] The role of cigarette smoking in the excess risks observed in this study cannot be determined quantitatively because no data were available on the smoking habits of the study cohort. However, according to Axelson,[13] smoking is a weak confounder unless it is strongly associated with the disease and unless smoking habits between exposed and nonexposed workers differ drastically. The possible effects from smoking on the risk for bladder cancer in this population were calculated based on the method described by Axelson.[13] Based on these calculations, it can be concluded that smoking cannot account for the threefold excess seen in this cohort. In fact, if 100% of the population were heavy smokers, this would account for only a 56% increase in the risk.

The most striking finding in the study was the overwhelming deficit in mortality due to accidents. This may be due to the demographics of the study population, which is primarily lower socioeconomic and inner city (Chicago, New York City, Detroit, and Oakland, California) workers. Therefore, it is possible that these workers did not own automobiles and relied heavily on inner city transportation systems which would account for the deficit in transportation accidents.

The two deaths from renal calculi, both of which were found in the subcohort of workers employed only in PCE shops was an unexpected finding that may have been a chance occurrence. However, it is possible that exposure to PCE could be related to this finding. Although we know of no reports describing nephrotoxicity in humans exposed to PCE, renal effects have been observed in experimental animal studies.[2,3] In addition, there have been reports in the literature describing the occurrence of urinary calculi due to chemical exposure. A person developed renal tubular acidosis and urinary calculi after persistent toluene sniffing,[14] and ethylene glycol has been associated with the production of calcium oxalate in renal tubules leading to formation of calculi in rats.[15] The role PCE might play in these types of mechanisms which lead to urinary calculi is unknown.

Several other epidemiologic studies of dry cleaner and laundry workers have been conducted. In a study by Blair et al[16] a proportionate mortality ratio (PMR) analysis was performed on 330 death certificates obtained from two local laundry and dry cleaning unions. These deaths only represented a sample of the total number of deaths that occurred between 1957 and 1977 among the union members. The sample of deaths was not necessarily a probabilistic sample but included deaths that had been identified by the researchers. Workers were included regardless of the solvent used in the dry cleaning shop which employed the worker; therefore, it was not specifically a study of PCE expo-

sure. The risk of mortality from all cancers combined was found to be higher than expected (87 observed *v* 67.9 expected; $P < .05$). Among the cancer deaths, lung, cervix uteri, and skin cancers were elevated at a statistically significant level. Other malignant neoplasms found in excess were intestine, liver and leukemia. Risks for bladder, kidney and pancreatic cancer were not elevated, and breast cancer risk was lower than expected.

Katz[17] studied the mortality of female laundry and dry cleaner workers in Wisconsin by identifying 671 deceased workers from the occupational statement listed on death certificates. He tested for associations between occupation and cause of death by calculating specific PMRs for 25 causes of death. As in Blair's study the workers did not necessarily work at shops using PCE. In contrast to Blair's study, the risk of death for all cancer, liver cancer, colon cancer, lung cancer and leukemia was not elevated. Statistically significant excess risks were found for cervix uteri, genital (unspecified), and kidney cancer (7 observed *v* 2.7 expected). Bladder cancer risk was also elevated (5 observed *v* 2.6 expected). Breast cancer risk was less than expected.

A study using similar methods was conducted by Duh and Asal[18] in Oklahoma, where deaths from 440 laundry and dry cleaner workers were analyzed. Again, no identification of solvent use was attempted; however, the use of petroleum solvents is more common among the dry cleaning shops in Oklahoma where more than 50% of the shops use this solvent, whereas the remaining use PCE. Duh and Asal found a statistically significant excess risk for lung and kidney cancer. Other elevated risks were found for cervix uteri, other female genital organs, and skin cancer. The risks for liver, pancreas, and bladder cancer were lower than expected.

The mortality risks for these previous studies of dry cleaner workers along with those of the present study have been summarized in Table 6. There were several causes that exhibited consistent excess risks, including cancer of the cervix uteri and kidney. A deficit was observed in all studies for breast cancer. As discussed earlier, the results for cervix uteri and breast are probably related to the socioeconomic status of these occupational groups.

TABLE 6
Comparison of Cause-Specific Risks Among Mortality Studies of Dry Cleaner (and Laundry) Workers*

Cause of Death	Blair et al[16] (PMR)	Katz[17] (PMR)	Duh and Asal[18] (SMOR)	Brown (SMR)
All cancer	128†	96	90	116
Intestine	152	103	60	136
Liver	235	89	50	—‡
Pancreas	129	117	50	172
Lung	170†	98	170	114
Skin	429†	207	150	—
Breast	69	72	10	87
Cervix uteri	208†	195†	130	196
Bladder	83	189	40	296†
Kidney	200	257	380	200
Leukemia	227	67	—	—

* Abbreviations used are: PMR, proportionate mortality ratio; SMOR, standardized mortality odds ratio; SMR, standardized mortality ratio.
† $P < .05$.
‡ — = not calculable—no observed deaths.

Conclusion

The excess risk for urinary tract cancer in this study was somewhat unexpected. Because multiple causes of death were examined and this cause of death was not part of an a priori hypothesis, the finding may be due to chance. However, it was the only cause found to have a statistically significant excess in mortality and the excess risk was found in three out of the four race/sex groups and in three out of the four unions. The magnitude of the SMR and the pattern by latency and exposure to PCE was consistent with an occupational carcinogen. However, because of the limitation in the records used to identify the cohort, it is not possible to analyze the data by specific job or estimate of exposure to PCE. Therefore, a dose-response analysis for urinary tract cancer, other than by length of employment, is not possible. Even the analysis by length of employment is limited as a surrogate of exposure because of missing data on solvent use. Inasmuch as many of the workers could have had ccnfounding exposures to petroleum solvents in dry cleaning shops, those with employment in PCE shops only were analyzed separately. In this "PCE only" subcohort there was no excess risk for urinary tract cancer. Therefore, the confounding exposure to petroleum solvents complicates any conclusions regarding the association between PCE exposure and cancer of the urinary tract.

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References

1. *National Occupational Hazard Survey, vol 3: Survey Analysis and Supplemental Tables.* Dept of Health, Education and Welfare, National Institute for Occupational Safety and Health, publication No. 78-114, 1977 (with data base update as of Aug 6, 1980).

2. *Bioassay of Tetrachloroethylene for Possible Carcinogenicity.* Carcinogenesis Technical Report Series, No. 13. Dept of Health, Education and Welfare, National Cancer Institute, publication No. (NIH) 77-8N, 1977.

3. *National Toxicology Program Technical Report on the Toxicology and Carcinogenesis Studies of Tetrachloroethylene (Perchloroethylene) in F344/N Rats and B6C3F Mice.* National Institutes of Health, publication No. 85-2567, August 1985 (board draft).

4. Ludwig HR, Meister MV, Roberts DR, et al: Worker exposure to perchloroethylene in the commercial dry cleaning industry. *Am Ind Hyg Assoc J* 1983;44:600–605.

5. Waxweiler RJ, Beaumont JJ, Henry JA, et al: A modified life-

table analysis system for cohort studies *J Occup Med* 1983;25:115–124.

6. Breslow NE, Lubin JH, Langholz B: Multiplicative models and cohort analysis. *J Am Stat Assoc* 1983;78:1–12.

7. Hoover R, Mason TJ, Mckay FW, et al: Geographic patterns of cancer mortality in the United States, in Fraumeni JF (ed): *Persons at High Risk of Cancer: An Approach to Cancer Etiology and Control.* New York; Academic Press, 1975, pp 343–360.

8. Hammond EC: Smoking in relation to the death rates of one-million men and women, in Haenzel W (ed): *Epidemiological Approaches to the Study of Cancer and Other Chronic Diseases.* US Dept of Health, Education and Welfare, Public Health Service, National Cancer Institute Monograph 19, 1975, pp 127–204.

9. Zavon MR, Hoegg U, Bingham E: Benzidine exposure as a cause of bladder tumors. *Arch Environ Health* 1973;27:1–7.

10. Matanowski GM, Elliott EA: Bladder cancer epidemiology. *Epidemiol Rev* 1981;3:203–229.

11. Kitchen DN: Neoplastic renal effects of unleaded gasoline in Fischer 344 rats, in Mehlman MA (ed): *Renal Effects of Petroleum Hydrocarbons: Advances in Modern Environmental Toxicology.* Princeton, NJ, Princeton Scientific Publishers Inc, 1984, vol 7, pp 65–71.

12. Sandmeyer EE: Aromatic hydrocarbons, in Clayton GD (ed): *Patty's Industrial Hygiene and Toxicology*, ed 3. New York, John Wiley and Sons, 1981, vol 2B, pp 3253–3433.

13. Axelson O: Aspects of confounding in occupational health epidemiology (letter). *Scand J Work Environ Health* 1978;4:98–102.

14. Kroger RM, Moore RJ, Lehman TH, et al: Recurrent urinary calculi associated with toluene sniffing. *J Urol* 1980;124:89–91.

15. Williams HE, Smith LH: Disorders of oxalate metabolism. *Am J Med* 1968;45:715–735.

16. Blair A, Decoufle P, Grauman D: Causes of death among laundry and dry cleaning workers. *Am J Public Health* 1968;69:508–511.

17. Katz RM, Jowett D: Female laundry and dry cleaning workers in Wisconsin: A mortality analysis. *Am J Public Health* 1981;71:305–307.

18. Duh RW, Asal NR: Mortality among laundry and dry cleaning workers in Oklahoma. *Am J Public Health* 1984;74:1278–1280.

The Artist's Dream

By artist I mean . . . everyone who has tried to create something which was not here before him, with no other tools and material than the uncommerciable ones of the human spirit; who has tried to carve, no matter how crudely, on the wall of that final oblivion, in the tongue of the human spirit, "Kilroy was here."

That is primarily . . . all . . . we ever really tried to do. And I believe we will all agree that we failed. That what we made never quite matched and never will match the shape, the dream of perfection which we inherited and which drove us and will continue to drive us, even after each failure, until anguish frees us and the hand falls still at last.

Maybe it's just as well that we are doomed to fail, since, as long as we do fail and the hand continues to hold blood, we will try again; where, if we ever did attain the dream, match the shape, scale that ultimate peak of perfection, nothing would remain but to jump off the other side of it into suicide. . . .

—Comments by William Faulkner upon receipt of National Book Award for fiction in 1955. From "'Three Cheers for Good Marks': Writers on Their Prizes" in *The New York Times Book Review*, Nov 16, 1986