MORTALITY IN DRY-CLEANING WORKERS: An Update

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Abstract

Background: A cohort of 1,708 dry-cleaning workers identified from union records, was exposed to perchloroethylene (PCE), a known animal carcinogen and probable human carcinogen, for at least one year before 1960. Many workers also had exposure to Stoddard solvent, a petroleum-based dry-cleaning solvent.

Methods: Vital status was updated through 1996 and life table analyses conducted.

Results: The cohort had excess cancer mortality (271 deaths, standardized mortality ratio [SMR] 1.25, 95% confidence interval [CI] 1.11-1.41). Elevated SMRs for tongue, bladder, esophagus, intestine, lung, and cervical cancer, pneumonia, and diseases of the stomach and duodenum were statistically significant.

Conclusion: The current study confirms findings of prior updates and other studies that dry cleaning workers have excess cancer mortality at several sites. Although important lifestyle and socioeconomic risk factors exist for both cervical and esophageal cancer mortality, excesses of these sites in the PCE only subcohort and among workers with longer duration of PCE exposure suggest an association with PCE exposure.

Key Words: solvents, occupational exposure, tetrachloroethylene, cohort studies, cancer mortality

Background

Human and animal studies of the effects of exposure to PCE and to Stoddard solvent have been documented and were reviewed in the original mortality study (Brown and Kaplan, 1987) and our earlier update (Ruder et al., 1994). More recently, there have been studies of possible PCE neurotoxicity (Cavalleri et al., 1994; Echeverria et al., 1995), kidney damage (Verplanke et al., 1999) and association with spontaneous abortion (Doyle et al., 1997). Possible associations of PCE with cancers of the kidney (Mandel et al., 1995; Schlehofer et al., 1995; McLaughlin and Bolt, 1997), liver (Lynge, 1994), and breast (Aschengrau et al., 1998) have also appeared in the scientific literature. The International Agency for Research on Cancer has classified PCE as a probable human carcinogen (IARC, 1995). There have been no completed bioassays for the carcinogenicity of Stoddard solvent to date, but a two-year test is currently underway in the National Toxicology Program at the National Institute for Environmental Health Sciences (NTP, 1998).

The original NIOSH study (of which this is the second update) investigated mortality through 1982 among 1,708 dry-cleaning workers (65% female, 52% nonwhite) with PCE and other solvent exposures, including about one-third exposed only to PCE (Brown and Kaplan, 1987). Although overall mortality was less than expected, cancer mortality was increased (142 deaths, SMR 1.16, CI 0.97-1.36), and the increase in bladder cancer mortality was statistically significant (8 deaths, SMR 2.96, CI 1.28-5.86). Two deaths due to urinary calculi were observed in the PCE-only subcohort (SMR 6.67, CI 0.73-21.9). Our 1994 analysis updated vital status through 1990 and found the excesses for esophageal and intestinal cancer had become statistically significant. Among workers exposed only to PCE, those with 5 or more years of employment and 20 or more years since first exposure had a statistically significant increased risk of esophageal cancer (Ruder et al., 1994).

Information on the dry-cleaning industry and on current regulations can be found in appendix I.

For the current update our objective was to document mortality in this cohort and to investigate possible associations with occupational exposure.

Methods

Dry-cleaning union records in four areas (San Francisco-Oakland, California; Chicago, Illinois; Detroit, Michigan, and New York, New York) were searched for individuals not known to ever have been exposed to carbon tetrachloride who had worked for at least one year prior to 1960 at a shop using PCE as the cleaning solvent (in every dry-cleaning shop, small amounts of a variety of solvents, are present, used for spotting stains). Cohort members could be actively employed, retired, deceased, or lost to followup in 1960.

Where possible, shops were visited to verify solvent use history. A subcohort of 625 workers worked only in a shop or shops where PCE was the cleaning solvent (PCE-only subcohort). For the other 1083 workers (PCE-plus), periods of work at shops where records were inadequate to confirm PCE use, or another solvent (mostly Stoddard solvent or other petroleum solvents), was known or thought to have been used as the cleaning solvent, were included in work histories coded as "other solvent."

We used the National Death Index to ascertain mortality through December 31, 1996. Analytic methods were those used in the original and followup investigations (Brown and Kaplan, 1987; Ruder et al., 1994). Age-, sex- and calendar-time-specific SMRs were derived using the modified life-table analysis system (LTAS) developed by NIOSH (Waxweiler et al., 1983; Steenland et al., 1990) to calculate person-years at risk (PYAR) and the expected number of deaths, using national rates except when analyzing the four geographic subcohorts. For each worker in our study, the period of observation started January 1, 1940 (rate files are not available for earlier years), or after one year of employment in a unionized PCE plant, whichever was later, and ended at the date lost to followup, date of death, or December 31, 1996, whichever was earlier. We calculated SMRs for the entire cohort and for the two exposure subcohorts by duration of employment in dry-cleaning shops using PCE (1-5 years, 5+ years) as a surrogate for PCE exposure and by latency periods (less than 20 years, 20+ years). Analyses for the four geographic subcohorts used county rate files for 1960-1996 (these are not available for earlier years) and the national rate file for 1960-1996. Additional analyses used the multiple-cause mortality rates and programs (Steenland et al., 1992). For nonmalignant chronic diseases, such as renal disease, an outcome of a priori interest in this cohort, a multiple-cause analysis (MCOD) could reveal excesses not observed in the analysis of underlying causes of death (UCOD) (Steenland et al., 1992). All analyses were done using the PC version of the LTAS (Steenland et al., 1998,

http://www.cdc.gov/niosh/ltindex.html). In our tables we present the number of deaths observed, the SMR, and the 95% confidence interval (two-sided) for the SMR. Testing for heterogenity and trend in the SMRs and calculation of approximate confidence intervals used the methods of Breslow and Day (1987).

Results

The current composition and status of the cohort and the subcohorts with PCE-only and PCE-plus exposures are shown in Table I. During the six additional years of followup 226 members of the cohort died.

Cancer Deaths

As in the previous analyses, there was a statistically significant increase in cancer deaths overall (Table II), with the excess mainly in nonwhites. Among white women the rate was elevated; among white men there was no increased cancer risk overall.

Three sites (esophagus, intestine, and bladder) had a statistically significant excess of cancer deaths through 1990, and continue to have elevated SMRs. Esophageal cancer is in non-statistically significant excess in all four gender-race categories. Intestinal cancer is in twofold excess for nonwhite men (8 deaths, SMR 2.30, CI 1.00-4.53) and white women (15 deaths, SMR 2.06, CI 1.15-3.40), increased for nonwhite women and reduced in white men. Bladder cancer deaths were confined to men and white women, with a statistically significant fourfold increased risk for nonwhite men (4 deaths, SMR 4.19, CI 1.14-10.7). (A table of SMRs by gender-race categories is available from the first author).

In this update, the elevated SMRs for cancer of the tongue, lung, and cervix were statistically significant. Nonwhite workers experienced all the tongue cancers (2 deaths among women, SMR 10.4, CI 1.25-37.4; 3 deaths among men, SMR 8.92, CI 1.84-26.1) and the statistically significant excess of lung cancer (16 deaths among women, SMR 1.88, CI 1.07-3.05; 25 deaths among men, SMR 1.52, CI 1.05-2.39). The cervical cancer SMRs were elevated, but not statistically significant, for both nonwhite and white women.

An analysis of multiple causes of death (MCOD) included all causes of death shown on the death certificate (Steenland et al., 1992). For several causes of death which did not have a statistically significant elevated SMR in the underlying cause of death (UCOD) analysis, the MCOD analysis showed a statistically significant excess. The UCOD analysis includes for each decedent that single cause which ultimately led to the death. This cause often is not the immediate cause of death. For pancreatic cancer the UCOD SMR was 1.53 (18 deaths, CI 0.91-2.42); the MCOD SMR was 1.66 (20 deaths, CI 1.02-2.57). There were 29 deaths due to cancer of other and unspecified sites in the UCOD analysis (SMR 1.25, CI 0.83- 1.79); 176 deaths in the MCOD analysis (SMR 1.52, CI 1.31-1.77). There were no additional esophageal or bladder cancer deaths in the MCOD analysis and one additional cervical cancer death (13 deaths, SMR 2.32, CI 1.23-3.96). An analysis was also conducted using rate files which have 99 causes of death (instead of 92) because these include non-Hodgkins lymphoma (NHL), which was found to have a positive association with PCE exposure in a recent IARC review (IARC, 1995). In our cohort there were 7 NHL deaths from 1960-96 (SMR 1.39, CI 0.56-2.86).

Table III presents cancer SMRs by time since first exposure and duration of exposure. For esophageal and bladder cancer, and cancer overall, there is a clear pattern of increased risk with longer time since first exposure and with longer duration of exposure. The tongue, intestinal, and lung cancer SMRs show a statistically significant association with time since first exposure; the cervical cancer SMRs show a statistically significant association with duration of employment.

The mortality experiences of the two subcohorts, PCE-only and PCE-plus, are presented in Table IV. Among the PCE-only subcohort, deaths from tongue cancer were in statistically significant excess. As reported previously, those with 5 or more years of work with PCE and 20 or more years of latency since first exposure were at statistically significant increased risk of esophageal cancer. Among the PCE-plus subcohort, deaths from esophageal, intestinal, pancreatic, lung and bladder cancer were in statistically significant excess. In the multiple cause of death analysis, there was also an increased risk for rectal cancer mortality in this subcohort (UCOD analysis, 7 deaths, SMR 2.16, CI 0.86-4.45; MCOD analysis, 9 deaths, SMR 2.64, CI 1.21-5.02).

Mortality in the four geographic subcohorts was analyzed, using local (county) mortality rates as well as U.S. rates (1960-96) for comparison. In general, the results for the geographical subcohorts are consistent with those in the overall cohort. In addition, SMRs calculated based on county rates are generally similar to those based on U.S. rates (table available from first author upon request). The county-rate-based SMR for cirrhosis is 0.89, while the U.S.-rate-based SMR is 1.42, but the confidence intervals do overlap. The results generally suggest that geographical variation in mortality does not account for the observed excesses. Additional analyses (not shown) demonstrate that cancer mortality SMRs have remained elevated through the 1990s. (Table available from first author upon request.)

Heart and circulatory system disease

Heart disease SMRs were not elevated or reduced in the cohort as a whole (Table II). However, among the PCE-only subcohort, deaths from ischemic heart disease (IHD) were in statistically significant excess and there was a statistically significant deficit of deaths from circulatory system disease (Table IV). In the multiple causes of death analysis, there were excess deaths in the cohort as a whole due to other diseases of the heart (UCOD analysis, 57 deaths, SMR 0.94, CI 0.71-1.22; MCOD analysis, 367 deaths, SMR 1.12, CI 1.01-1.24) and to hypertension without heart disease (UCOD analysis, 22 deaths, SMR 0.92, CI 0.58-1.39; MCOD analysis, 65 deaths, SMR 1.59, CI 1.22-2.02).

Respiratory disease

There were 43 pneumonia deaths observed and 28.1 expected (SMR 1.53, CI 1.07-2.06). Although seven of the pneumonia deaths occurred in individuals age 64 or younger, 8.6 deaths were expected (SMR 0.82) in that age group versus 36 deaths and 19.5 expected (SMR 1.85, CI 1.30-2.57) among those 65 or older. No association between pneumonia SMR and duration of employment was seen. Among nonwhite men there was a statistically significant increase for pneumonia (14 deaths, SMR 2.02, CI 1.10-3.39) and rates were elevated for women, but not for white men. Women also experienced a greater than threefold increased risk of death from emphysema; for white women the elevation was statistically significant (6 deaths, SMR 3.81, CI 1.39-8.30).

Digestive system disease

The statistically significant increase in deaths from diseases of the stomach and duodenum (Table II) was not

associated with duration of employment. Rates were increased for all but nonwhite men, with a statistically significant threefold increase for white men (5 deaths, SMR 3.25, CI 1.05-7.59). There was also a statistically significant increase for those in the PCE-plus subcohort (Table IV). White women were the only race-gender group at increased risk of cirrhosis (8 deaths, SMR 2.51, CI 1.08-4.95).

Genitourinary system disease

Two deaths due to urinary calculi were previously reported for this cohort (both white males exposed only to PCE). No additional deaths from this cause were observed. In this update, there was some indication of excess deaths due to acute glomerulonephritis, nephrotic syndrome, and acute renal failure (Table II).

Deaths from other causes

There was a statistically significant deficit of deaths from accidents (Table II).

Discussion

Cancer mortality

Excesses of cancer in dry-cleaning workers have been reported in many cohort and case-control studies (Vaughan et al., 1997; Mandel et al., 1995; Schlehofer et al., 1995; Lynge, 1994; Blair et al., 1990; Lynge and Thygesen, 1990; Asal et al., 1988; Duh and Asal, 1984; Schoenberg et al., 1984; Katz and Jowett, 1981; Lin and Kessler, 1981). A recent IARC review (IARC, 1995), which included the original report and previous update on this cohort, found evidence for consistently positive associations between exposure to PCE and risks for esophageal and cervical cancer and non-Hodgkins lymphoma. In the present update the evidence for cervical and esophageal cancer has become stronger. Mortality from non-Hodgkins lymphoma is elevated, but the SMR is not statistically significant.

Several of the cancers found to be in excess in this cohort are known to be smoking-related, and the observed elevations in both lung cancer and emphysema could be related to an increased prevalence of smoking in the study cohort compared to the referent population. However, the magnitude of the SMRs for for several smoking related cancers are greater than could be explained by smoking alone, and several of these are also related to longer duration of employment in shops using PCE. There is very little in the literature on the prevalence of smoking among dry

cleaners. An analysis of National Health Interview Survey data by Nelson et al. (1994) presented rates of 32.1% ± 7.3% for "laundry and dry cleaning operators" and 47.6% ±14.3% for "pressing machine operators"; the overall blue collar and service prevalences were 39.2% and 34.5%, respectively. Both job categories include workers in the laundry and dry-cleaning industries; dry-cleaning-specific rates were not shown. Vaughan and colleagues (1997) commented that the dry cleaners in their case-control study smoked and drank less than other blue collar workers, but there were only 24 dry-cleaning workers among the 1844 participants.

The statistically significant twofold excess of cervical cancer found in the NIOSH and other (Blair et al., 1990; Katz and Jowett, 1981) dry-cleaning cohorts may reflect socioeconomic factors, as nonwhite race, lower income, and less education have been associated with both greater risk of cervical cancer (Fasal et al., 1981; Devesa, 1984) and with increased odds of having invasive vs. in-situ cancer at diagnosis (Mandelblatt et al., 1991). However, Savitz et al. (1995) in a case-control analysis adjusting for some known risk factors for cervical cancer (but not human papilloma virus, the major causal agent for cervical cancer [Bosch et al., 1995; Schiffman et al., 1993]), found a fourfold increased risk for invasive cervical cancer among "maids and cleaners." Berlin et al. (1995) found a fourfold increased cervical cancer incidence among solvent-exposed workers.

The difference in cancer mortality rates between the PCE-only and PCE-plus subcohorts (Table IV) could be due to differences in type of exposure. Through 1977, both subcohorts had a mean of about six years of PCE exposure (Table I), and the PCE-plus subcohort had a mean of about five years of additional solvent exposure (to unidentified-as-such PCE and to other solvents). Because both subcohorts had comparable durations of PCE exposure, PCE cannot be ruled out as the cause of the excess mortality in the PCE-plus subcohort. The overall cancer mortality rate has been increasing in the PCE-only subcohort over the past 45 years. It should also be remembered that the statistical significance of a given SMR depends on the number of observed deaths, and the PCE-only subcohort is about half the size of the PCE-plus subcohort. For example, both subcohorts had elevated rates of esophageal cancer (Table IV). Excess pancreas and bladder cancer mortality is apparent only in the PCE plus group but is associated with longer duration of exposure to PCE. These cancers may be related to occupational PCE exposures or exposure to other dry cleaning solvents, primarily Stoddard solvent.

Heart and circulatory system disease

The literature on chlorinated solvent exposure and cardiovascular disease is not very large. Katz and Jowett (1981) observed among female laundry and dry cleaning workers a statistically significant excess of ischemic heart disease deaths when comparing them to workers in all occupations, but not when comparing them to workers in lower wage occupations. Nakamura (1985) found excess deaths from ischemic heart disease and "other forms of heart disease" among Japanese dry cleaners. A cohort of aircraft maintenance workers exposed to trichloroethylene had a statistically significant excess of ischemic heart disease mortality (Blair et al., 1998) [both TCE and PCE are metabolized to trichloroacetic acid (Ikeda et al., 1972)]. There have been reports of transient arrhythmias following PCE exposure in lab animals (Kobayashi et al., 1982; Carlson, 1983), but no biologically plausible link between chronic exposure and cardiovascular pathology has been established. Kotseva and Popov (1998) suggested several mechanisms by which solvents might cause heart disease: direct toxicity on the heart muscle, sensitizing the myocardium to catecholamines, disturbing lipid metabolism, or increasing blood pressure. In our cohort, the mean age at death from IHD was 70.4 years and none of the age-specific SMRs for IHD was in statistically significant excess.

Respiratory system disease

There is little evidence for an association of respiratory disease with exposure to dry-cleaning solvents, although Blair et al. (1990) reported a nonstatistically significant excess of pneumonia deaths. Pulmonary edema has been reported for acute PCE exposure (Patel et al., 1977; Meyer, 1973) and Stoddard solvent, the most common second exposure for this cohort, is an irritant which has been associated with impaired respiratory function (IARC, 1989). However, the excess pneumonia deaths occurred among individuals age 70 or more, presumably no longer actively employed.. No biologically plausible mechanism for chronic pulmonary effects related to exposure to PCE or Stoddard solvent has been postulated. The observed excess mortality may be due to socioeconomic factors and/or access to medical care (Morris and Munasinghe, 1994).

Digestive system disease

The statistically significant SMR for stomach and duodenal disease may be due to lifestyle or socioeconomic factors; peptic ulcer mortality is higher in lower socioeconomic classes (Fleming, 1994). A rare intestinal condition,

pneumatosis cystoides intestinalis, has been associated with TCE exposure (Fleming, 1994) and increased stomach disease mortality has been reported for solvent-exposed cohorts (Dubrow and Gute, 1987; Duh and Asal, 1984). However in our cohort there is no association between length of employment and risk of stomach disease mortality, arguing against an occupational association.

Genitourinary disease

Both the animal literature and human studies provide support for the association of urinary tract disease with dry-cleaning solvent exposure seen in Table II. The occurrence of two deaths from urinary calculi among the 625 workers exposed only to PCE (Table IV) suggests, combined with previous studies and animal studies, that further studies of urinary tract disease in dry-cleaning workers might be warranted. However, it should be noted that both urinary calculi deaths occurred before 1970 and if work-related may reflect the higher exposures of the 1940's through 1960's.

Other causes of death

Brown and Kaplan (1987), in reporting the statistically significant deficit in deaths due to accidents which continues in this update, speculated that (presumably) lower-income city residents might not have automobiles, and would therefore be at lower risk for automobile accidents.

The limitations of this cohort study have been described in detail previously (Ruder et al., 1994). To summarize these limitations: (1) we updated vital status but not work histories for the cohort, so duration of exposure will be underestimated for some cohort members; (2) we cannot quantify exposure as we do not have job titles for two-thirds of the cohort, or personal exposure measurements for anyone; (3) we do not know to exactly which solvents, in addition to PCE, members of the PCE-plus subcohort were exposed (although for most of them the probable exposure was Stoddard solvent); and (4) we do not know about possibly confounding factors such as smoking, drinking, and diet. For the present analysis, the lack of complete work histories (beginning with first exposure to PCE in a unionized plant but not necessarily first exposure to PCE, and truncated at 1977) means we cannot evaluate precisely which deaths might have occurred during the period of active employment. Some deaths, such as respiratory disease deaths, could be associated with contemporaneous exposures. Underestimated durations of

employment and the inability to quantify exposure (even as high or low) would tend to bias results toward the null. Many of the diseases with elevated mortality rates in this cohort have been associated with cigarette smoking. One would expect smoking-related disease risk to be associated with factors related to smoking (number of pack-years, type of cigarettes) but not to occupational factors such as length of employment or time since first exposure. However, in this cohort both a pattern of increasing risk with increasing length of employment and one of low risk during the 15-20 years after first exposure and high risk thereafter are found for diseases associated with cigarette smoking in the literature (Table III), although, lacking smoking histories, we cannot with certainty attribute these diseases to occupational exposures. Lack of other lifestyle data could affect results for diseases known to be associated with social class, since this is a relatively low income group. It should be noted, however, that for these unionized workers, statistics on medical insurance and access to medical care would be more similar to those of other, higher paid unionized workers than to those of nonunionized lower income workers.

This cohort is experiencing statistically significant excess cancer mortality which appears to be related to occupational exposure, as well as excesses for IHD, pneumonia and diseases of the stomach and duodenum, for which the evidence supporting an occupational etiology is not as strong. We plan to continue our investigation of cancer in this cohort with incidence studies to evaluate cancers which may not have resulted in death, such as bladder and cervical cancer, which have high survival rates if diagnosed early. To determine if the excess cancer risk in dry-cleaning workers can be attributed to a specific solvent, it would be necessary to study a large cohort with complete exposure information. However, a feasibility study by NCI and NIOSH to characterize a nonunionized large cohort suitable for a retrospective study was unsuccessful, due to the predominance in this industry of small to medium workplaces which do not maintain complete personnel records, due to high employee turnover rates (ITEP, 1992; Table I). Other approaches, such as cross-sectional studies examining intermediate endpoints, may be necessary to better understand the potential carcinogenicity of PCE.

The bladder, cervical, and intestinal (colon) cancer excesses argue for cancer screening efforts among current and former dry-cleaning workers. Modalities for screening for cervical and colon cancer are available and recommended for the general population (Miller, 1996; Read and Kodner, 1999); screening for bladder cancer is not recommended

for the general population but has been used for high risk groups. These findings also support NIOSH's recommendation to reduce occupational exposure to perchloroethylene.

Summary

This cohort of 1,708 dry-cleaning workers, identified from union records and followed through 1996, is experiencing a statistically significant excess of cancer deaths (271 deaths, standardized mortality ratio [SMR] 1.25, 95% confidence interval [CI] 1.11-1.41). For all cancer deaths, SMRs were higher among those with 20 years or more since first exposure and among those who worked more than five years. Elevated SMRs for cancer of the tongue, bladder, esophagus, intestine, lung, and cervix were statistically significant, as were those for pneumonia and diseases of the stomach and duodenum. Those exposed only to PCE (n=625) experienced statistically significant excesses for cancer of the tongue, ischemic heart disease, and urinary calculi, and a deficit of circulatory system disease deaths. A statistically significant excess of esophageal cancer was seen among those who worked only with PCE, for over five years, and whose first exposure was at least 20 years before death.

Conclusion

The current study confirms findings of prior updates and other studies that dry cleaning workers have excess cancer mortality at several sites. Our ability to associate these excesses directly with PCE exposure is limited because relatively few workers were exposed only to PCE, and the remainder of the cohort also had exposure to other solvents. Although important lifestyle and socioeconomic risk factors exist for both cervical and esophageal cancer mortality, the excesses in the PCE only subcohort and among workers with longer duration of PCE exposure suggest an association with PCE exposure. Excess pancreatic and bladder cancer mortality is apparent only in the PCE plus group but associated with longer duration of PCE exposure. These cancers may be related to exposure to PCE or other dry cleaning solvents, primarily Stoddard solvent. Epidemiologic evidence concerning the association of other causes of mortality in excess is even more limited. The results of this study add to the weight of the evidence that solvents used in the dry cleaning industry are carcinogenic. PCE and Stoddard solvent, extensively used in the U.S. dry cleaning industry for 50 years, remain in common use in dry cleaning and other industries, potentially posing ongoing hazards to current workers. In view of widespread PCE exposure and the evidence for PCE's carcinogenicity in animals, definitive epidemiologic studies of PCE's carcinogenicity are needed. Data concerning

Stoddard solvent carcinogenicity in animals and epidemiologic studies of occupational groups exposed only to Stoddard solvent are needed to evaluate whether the observed elevations for pancreatic and bladder cancer may be associated with Stoddard solvent exposure.

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Appendix I: Dry-cleaning operations and regulations

Dry cleaning is machine laundering using a nonaqueous solvent instead of water. The solvents have included carbon tetrachloride, widely used until just after World War II (Brown and Kaplan, 1987), petroleum solvents (Stoddard solvent), trichloroethylene (TCE) and tetrachloroethylene (perchloroethylene, PCE).

PCE is used now by over 90% of all dry-cleaning plants, by other industries as a degreaser, and as a solvent in the manufacture of rubber solutions, paint removers and printing inks. The National Occupational Exposure Survey (NOES) estimated that in the early 1980's over one-half million dry-cleaning plant employees and other industrial workers in over 40,000 plants potentially were exposed to PCE (NOES estimate, November 27, 1989). Occupational exposure to PCE is regulated by the Occupational Safety and Health Administration (OSHA) with the airborne concentration not to exceed an 8-hour time-weighted average (TWA) of 100 ppm (678 mg/m^3) and peak exposure not to exceed 200 ppm (1356 mg/m^3) for 5 minutes every 3 hours (OSHA, 1993). NIOSH classifies PCE as a potential occupational carcinogen and recommends that occupational exposures be reduced and the number of workers exposed be limited. NIOSH studies have shown that an exposure level of less than 5 ppm can be achieved with well-maintained modern dry-to-dry equipment (Burroughs et al, 1999; Earnest et al, 1997).

Exposure depends on equipment type (transfer or combination washer-dryer) and extent of solvent recovery (charcoal absorber and/or water-cooled or refrigerated condenser) from the drum contents before the drum is opened; on work practices (use of gloves and/or respirator, machine maintenance, number of loads per day, number of pounds per load); on shop layout; and on climate (open doors dissipate solvent but in the north in winter doors are not likely to be left open).

Dermal exposure to PCE has not been considered an important route of exposure in the dry cleaning industry since the development of dry-to-dry machines. However, based on comparisons of end-exhaled breath and time-weighted average (TWA) breathing zone air levels of PCE during a recent pilot intervention, dermal exposures may significantly contribute to the overall absorbed doses of workers. In addition, transfer operations–separate washer and dryer--still prevail in thousands of dry cleaning shops. During the transfer the operator removes the still-damp

items from the washer and puts them in the dryer. Because of this step, operator exposures in transfer shops average twice as high as in dry-to-dry shops (Stricoff, 1980). One of the limitations of the current study is that we have no information for the dry-cleaning cohort about work practices or machine types which would enable us to assign workers to relatively high or relatively low exposure categories.

Stoddard solvent, a petroleum-based mixture of alkane and aromatic hydrocarbons, is used by about 10% of all dry cleaning plants as a dry cleaning fluid and by a wide variety of other industries as a degreaser, metal cleaner, and paint remover. In the early 1980's, about 1.7 million workers in more than 130,000 plants potentially were exposed to Stoddard solvent (NOES estimate, November 27, 1989). OSHA mandates an 8-hour TWA occupational exposure to Stoddard solvent of no more than 100 ppm (525 mg/m^3), while NIOSH recommends a TWA of no more than 67 ppm (350 mg/m^3)(NIOSH, 1990).

To reduce the possibility of explosion, all Stoddard operations use transfer equipment, often with an extractor step between the washer and dryer. More recently developed petroleum solvents have higher flash points than Stoddard solvent. However, companies merchandising conversion of PCE to petroleum solvent propose converting a PCE dry-to-dry machine to a petroleum solvent washer and adding a dryer, so the transfer step would continue to exist.

In addition to the main cleaning solvent, workers–especially the spotter–are esposed to small amounts of other solvents used for stain removal. These include trichloroethylene, acetic acid, Ketone and acetone solvents, petroleum naphthas, and hydrogen fluoride and hydrofluoric acid. Other exposures include heat, noise, high humidity, and lint and other dusts (Earnest et al, 1997).

Table I Dry Cleaners, USA: Composition of the Entire Cohort and Subcohorts as of December 31, 1996

Characteristic	Entire Cohort (n=1708)	PCE Only (n=625)	PCE Plus (n=1083)
EXCLUDED FROM ANALYSIS*	5 (0.3%)	3 (0.5%)	2 (0.2%)
RACIAL DISTRIBUTION			
Females	1111 (65%)	415 (67%)	695 (64%)
White	520 (47%)	191 (46%)	331 (48%)
Nonwhite	591 (53%)	224 (54%)	364 (52%)
Males	592 (35%)	207 (33%)	385 (36%)
White	298 (51%)	108 (52%)	191 (50%)
Nonwhite	294 (49%)	99 (48%)	194 (50%)
VITAL STATUS			
Females alive†	538 (48%)	241 (58%)	297 (43%)
dead	573 (52%)	174 (42%)	399 (57%)
Males alive†	170 (29%)	79 (38%)	91 (24%)
dead	422 (71%)	128 (62%)	294 (76%)
Mean year of birth	1916 ± 11.6	1919 ± 11.9	1914 ± 11.0
Mean year first employed	1951 ± 5.7	1953 ± 4.6	1949 ± 5.7
Mean age at first employment	34.6 ± 10.2	34.2 ± 10.8	34.8 ± 9.8
Mean duration of employment‡	9.6 ± 6.8	6.4 ± 5.5	11.4 ± 6.7
using PCE	6.2 ± 5.0	6.4 ± 5.5	6.0 ± 4.7
using other solvents	3.4 ± 4.8	----------	5.3 ± 5.0
Mean number of shops worked in	2.3 ± 2.7	1.1 ± 0.4	3.0 ± 3.2**
Range	(1-62)	(1-4)	(2-62)
Person years at risk from PCE	53622	20677	33591

* Missing information (such as date of birth) essential for the analysis.

† "Alive" includes a total of 72 persons lost to followup before December 31, 1979, when the National Death Index was initiated.

‡ Through 1977.

** By definition everyone in this subcohort worked in at least two shops (one where PCE was the cleaning solvent and one where it was not)

Table II Dry Cleaners, USA: Mortality (through 12/31/96) for Selected Causes of Death

CAUSE OF DEATH	ICD Codes (9th Revision)	Obs	Exp	SMR	95% C.I.
Buccal and pharyngeal cancer	140-149	9	4.3	2.07	0.94- 3.93
Tongue	141	5	1.0	5.00**	1.62-11.68
Digestive organ cancer	150-159	81	60.9	1.33*	1.06- 1.65
Esophagus	150	14	5.7	2.47**	1.35- 4.14
Intestine (except rectum)	152-153	32	21.6	1.48*	1.01- 2.09
Liver and biliary	155, 156	1	6.2	0.16	0.00- 1.32
Pancreas	157	18	11.7	1.53	0.91- 2.42
Respiratory system cancer	160-165	68	50.4	1.35*	1.05- 1.71
Trachea, bronchus, lung	161	65	47.9	1.36*	1.05- 1.73
Breast cancer	174-175	20	22.1	0.91	0.55- 1.40
Female genital organ cancer	179-184	25	18.5	1.35	0.87- 1.99
Cervix uteri	180	12	6.2	1.95*	1.00- 3.40
Male genital organ cancer	185-187	11	12.0	0.92	0.46- 1.64
Urinary organ cancer	188-189	15	8.1	1.86*	1.04- 3.07
Kidney cancer	189.0-189.2	5	3.5	1.41	0.46- 3.30
Bladder & other urinary cancer	188,189.3-189.9	10	4.5	2.22*	1.06- 4.08
Other & unspecified sites	170-173,190-199	29	23.3	1.25	0.83- 1.79
Lymphatic & hematopoietic cancer	200-208	13	17.0	0.76	0.41- 1.30
All cancers	140-208	271	216.5	1.25**	1.11- 1.41
Tuberculosis	010-018	0	6.2	†	
Benign neoplasms		0	3.6	†	
Diabetes mellitis	250	21	26.0	0.81	0.50- 1.23
Blood & blood-forming diseases	281-289	4	3.5	1.15	0.31- 2.94
Alcoholism & mental disorders	290-319	5	7.9	0.63	0.20- 1.47
Nervous system diseases	320-337,349-389	5	11.2	0.45	0.14- 1.05
Diseases of the heart	390-398,402-404, 410-414,420-429	342	352.5	0.97	0.87- 1.08
Ischemic heart disease	410-414	256	252.7	1.01	0.89- 1.15
Diseases of the circulatory system	401,403,405,415-417,430-438,440-459	110	125.5	0.88	0.72- 1.06
Cerebrovascular disease	430-438	83	91.0	0.91	0.73- 1.13
Diseases of arteries, veins, & pulmonary circulation	415-417,440-459	19	30.9	0.70	0.42- 1.09

Respiratory system diseases	460-466,470-478, 480-487,490-519	82	63.0	1.30*	1.04- 1.62
Pneumonia	480-486	43	28.1	1.53**	1.11- 2.06
Emphysema	492	10	6.6	1.51	0.72- 2.78
Digestive system diseases	520-537,540-543, 550-553,555-558, 560,562-579	49	40.3	1.22	0.90- 1.61
Diseases of stomach & duodenum	531-537	11	4.7	2.33*	1.16- 4.17
Cirrhosis of the liver	571	20	16.0	1.41	0.77- 1.94
Genitourinary system diseases	580-608,610, 611,614-629	28	23.0	1.22	0.81- 1.76
Acute glomerulonephritis, nephrotic syndrome, acute renal failure	580,581,584	4	1.7	2.33	0.63- 5.95
Chronic & unspecified nephritis, renal failure, other renal sclerosis	582,583, 585-587	8	10.4	0.77	0.33- 1.52
Calculi of urinary system	592	2	0.4	4.85	0.59-17.50
Other genitourinary system diseases	588,589,591, 593,595-599	7	6.2	1.13	0.45- 2.33
Skin & subcutaneous tissue diseases	680-686,690-709	2	2.2	0.92	0.11- 3.32
Musculoskeletal diseases	710-739	1	3.1	0.33	0.01- 1.81
Symptoms & ill-defined conditions	780-796,798,799	1	14.0	0.07**	0.00- 0.40
Accidents	E800-848, E850-888,E890-949	8	31.4	0.25**	0.11- 0.50
Suicide & homicide	E950-E978	15	14.6	1.02	0.57- 1.69
Other causes	Residual ICD Codes	17	20.8	0.82	0.48- 1.32
Certificates not obtained		34			
Total deaths		995	965.4	1.03	0.97- 1.10

* 95% CI excludes the null value (1.0). ** 99% CI excludes the null value.

† SMR cannot be calculated.

Table IV Dry Cleaners, USA: Mortality in the PCE-only and PCE+ Subcohorts* for Selected Causes

	PCE-only			PCE-plus		
CAUSE OF DEATH	n	SMR	95% CI	n	SMR	95% CI
Tongue cancer	3	9.03‡	1.86-26.39	2	3.04	0.37-10.99
Esophageal cancer	5	2.65	0.85- 6.20	9	2.40†	1.10- 4.56
Intestinal cancer	8	1.18	0.51- 2.33	24	1.63†	1.04- 2.42
Rectal cancer	0			7	2.16	0.86-4.45
Pancreatic cancer	3	0.80	0.17- 2.35	15	1.89†	1.06- 3.11
Trachea, bronchus, lung cancer	19	1.17	0.71- 1.83	46	1.46†	1.07- 1.95
Breast cancer	6	0.78	0.28- 1.69	14	0.98	0.54- 1.65
Female genital organ cancer	10	1.60	0.77- 2.94	15	1.24	0.69- 2.04
Cervical cancer	4	1.89	0.52- 4.84	8	1.98	0.85- 3.91
Male genital organ cancer	2	0.65	0.08- 2.35	9	1.02	0.46- 1.93
Kidney cancer	2	1.73	0.21- 6.25	3	1.27	0.26- 3.72
Bladder & other urinary cancer	0			10	3.15‡	1.51- 5.79
Other & unspecified sites	9	1.17	0.53- 2.22	20	1.30	0.79- 2.01
Lymphatic & hematopoietic cancer	6	1.08	0.39- 2.36	7	0.61	0.25- 1.26
All cancers	76	1.08	0.85- 1.36	195	1.35‡	1.16- 1.55
Nervous system	3	0.82	0.17- 2.41	2	0.27†	0.03- 0.97
Diseases of the heart	116	1.11	0.92- 1.33	226	0.92	0.81- 1.05
Ischemic heart disease	93	1.27†	1.02- 1.55	163	0.92	0.78- 1.07
Circulatory system	24	0.66†	0.42- 0.97	86	0.98	0.78- 1.21
Arteries,veins,pulmonary circulation	2	0.25†	0.03- 0.90	17	0.88	0.51- 1.41
Respiratory system	27	1.43	0.94- 2.07	55	1.26	0.95- 1.64
Pneumonia	12	1.48	0.76- 2.58	31	1.57	1.07- 2.23
Emphysema	4	2.08	0.57- 5.31	6	1.28	0.47- 2.80
Digestive system	15	1.14	0.64- 1.88	34	1.28	0.89- 1.79
Stomach & duodenum	1	0.72	0.02- 4.01	10	3.11‡	1.49- 5.72
Cirrhosis of liver	8	1.40	0.60- 2.77	12	1.19	0.61- 2.08
Genitourinary system	7	1.01	0.40- 2.08	21	1.33	0.82- 2.03
Acute glomerulonephritis,nephrotic syndrome,acute renal failure	2	3.80	0.46-13.71	2	1.71	0.21- 6.19
Urinary calculi	2	16.97†	2.05-61.25	0	0.00	
Total deaths	302	1.01	0.90- 1.14	693	1.05	0.98- 1.13

* 625 cohort members were exposed only to PCE; the rest were exposed to PCE and other dry-cleaning solvents.

† 95% CI excludes the null value (1.0). ‡ 99% CI excludes the null value (1.0).

Table III Dry Cleaners, USA: SMRs* for Selected Cancers by Time Since First Employment in PCE Shops and Duration of Employment

SITE	Time<20 yrs. Duration<5 yrs.	Time<20 yrs. Duration 5+ yrs.	Time 20+ yrs. Duration <5 yrs.	Time 20+ yrs. Duration 5+ yrs.	Statistically Significant Difference by Time?	Statistically Significant Difference by Duration?
All	26 deaths	30 deaths	95 deaths	120 deaths	***	***
	0.67**	1.11	1.36***	1.48***		
	(0.44- 0.99)	(0.75- 1.59)	(1.10- 1.66)	(1.23- 1.77)		
Tongue	0	0	4	1	***	*** [1]
	0.00	0.00	13.64***	3.09		
			(3.71-38.27)	(0.04-25.49)		
Esophagus	0	0	4	10	***	***
	0.00	0.00	2.20	5.03***		
			(0.59- 6.13)	(2.41- 9.47)		
Intestine	2	4	13	13	**	No
	0.60	1.68	1.83	1.48		
	(0.07- 2.61)	(0.45- 4.66)	(0.97- 3.18)	(0.79- 2.58)		
Lung	2	5	32	26	***	No
	0.32	1.05	1.80***	1.36		
	(0.04- 1.39)	(0.34- 2.60)	(1.23- 2.55)	(0.89- 2.01)		
Cervix	3	4	2	3	No	**
	1.28	2.78	1.77	2.40		
	(0.26- 4.20)	(0.75- 7.71)	(0.20- 7.68)	(0.48- 7.86)		
Bladder	0	1	1	8	***	***
	0.00	1.90	0.72	4.31***		
		(0.02-15.39)	(0.01- 5.87)	(1.85- 8.76)		

* Approximate 90% confidence intervals calculated and heterogenity and trend tested with formulas of Breslow and Day (1987).

** 95% CI excludes the null value (1.0). *** 99% CI excludes the null value (1.0). [1] Short-term workers at significantly higher risk.

Supplementary Table: Mortality (through 12/31/96) for 1703 Dry-Cleaning Workers

Selected SMRs (and 95% Confidence Intervals) by Geographic Location Using Local[1] Rates for 1960-96

	California	Michigan	Illinois	New York	------entire cohort------	
SITE	(127 in subcohort)	(625 in subcohort)	(307 in subcohort)	(649 in subcohort)	combined state rates	U.S. rates
All	28 deaths	110 deaths	46 deaths	82 deaths	266 deaths	266 deaths
cancer	1.41 (0.94- 2.04)	1.37 (1.12- 1.65)**	1.21 (0.88- 1.61)	1.25 (1.00- 1.56)	1.31 (1.15- 1.47)**	1.40 (1.24- 1.58)**
tongue	no deaths	2 deaths	no deaths	3 deaths	5 deaths	5 deaths
		5.00 (0.61-18.06)		7.17 (1.48-20.96)*	4.45 (1.43-11.03)*	5.67 (1.83-13.25)**
esophagus	2 deaths	5 deaths	4 deaths	3 deaths	14 deaths	14 deaths
	4.46 (0.54-16.11)	2.19 (0.71- 5.12)	2.85 (0.78- 7.28)	1.39 (0.29- 4.08)	2.23 (1.22- 3.80)*	2.75 (1.50- 4.61)**
intestine	5 deaths	11 deaths	4 deaths	12 deaths	32 deaths	32 deaths
	2.42 (0.78- 5.65)	1.30 (0.65- 2.32)	0.98 (0.27- 2.49)	1.75 (0.90- 3.05)	1.49 (1.02- 2.11)*	1.67 (1.14- 2.36)**
pancreas	2 deaths	7 deaths	2 deaths	7 deaths	18 deaths	18 deaths
	1.73 (0.21- 6.24)	1.54 (0.62- 3.18)	0.94 (0.11- 3.38)	2.05 (0.82- 4.23)	1.60 (0.95- 2.56)	1.73 (1.02- 2.73)*
lung[2]	8 deaths	24 deaths	12 deaths	19 deaths	63 deaths	63 deaths
	1.71 (0.74- 3.38)	1.26 (0.81- 1.88)	1.38 (0.71- 2.42)	1.42 (0.85- 2.15)	1.38 (1.06- 1.77)*	1.46 (1.12- 1.87)**
cervix	no deaths	2 deaths	6 deaths	3 deaths	11 deaths	11 deaths
		1.00 (0.12- 3.61)	5.60 (2.04-12.19)**	1.78 (0.37- 5.21)	2.20 (1.10- 4.02)*	2.16 (1.08- 3.87)*
bladder	2 deaths	7 deaths	1 death	no deaths	10 deaths	10 deaths
	4.64 (0.56-16.74)	3.72 (1.49- 7.67)**	1.23 (0.03- 6.83)		2.28 (1.09- 4.30)*	2.54 (1.21- 4.66)*

other & unspec.	3 deaths 2.19 (0.45- 6.40)	11 deaths 2.25 (1.12- 4.02)*	3 deaths 1.30 (0.27- 3.80)	5 deaths 1.19 (0.38- 2.78)	22 deaths 1.72 (1.08- 2.63)*	22 deaths 1.61 (1.01- 2.43)*
heart disease	19 deaths 0.62 (0.37- 0.96)*	131 deaths 0.89 (0.74- 1.05)	65 deaths 0.92 (0.71- 1.17)	120 deaths 1.14 (0.94- 1.36)	335 deaths 0.94 (0.85- 1.05)	335 deaths 1.10 (0.98- 1.22)
pneumonia	2 deaths 0.71 (0.09- 2.55)	13 deaths 1.30 (0.69- 2.23)	10 deaths 1.68 (0.81- 3.09)	19 deaths 1.90 (1.14-2.97)*	44 deaths 1.53 (1.11- 2.06)**	44 deaths 1.86 (1.35- 2.50)**
stomach & duodenum	1 death 2.35 (0.06-13.05)	5 deaths 2.88 (0.93- 6.74)	3 deaths 3.74 (0.77-10.93)	2 deaths 1.33 (0.16- 4.81)	11 deaths 2.46 (1.23- 4.50)*	11 deaths 2.88 (1.43- 5.15)**
hernia & intes. obstruc.	no deaths	5 deaths 4.16 (1.35- 9.73)*	no deaths	no deaths	5 deaths 1.87 (0.60- 4.64)	5 deaths 1.74 (0.56- 4.08)
cirrhosis	3 deaths 1.67 (0.35- 4.90)	2 deaths 0.25 (0.03- 0.91)*	2 deaths 0.67 (0.08- 2.41)	13 deaths 1.32 (0.70- 2.25)	20 deaths 0.89 (0.54- 1.38)	20 deaths 1.42 (0.87- 2.19)
nephritis[3]	no deaths	2 deaths 0.63 (0.08- 2.29)	1 death 0.54 (0.01- 3.00)	6 deaths 2.84 (1.04- 6.18)*	9 deaths 1.18 (0.54- 2.29)	9 deaths 1.08 (0.49- 2.04)
accidents	1 death 0.44 (0.01- 2.47)	4 deaths 0.52 (0.14- 1.33)	no deaths	3 deaths 0.50 (0.10- 1.45)	8 deaths 0.41 (0.18- 0.84)*	8 deaths 0.32 (0.14- 0.63)**
All deaths	81 deaths 0.98 (0.78- 1.21)	386 deaths 1.08 (0.97- 1.19)	189 deaths 1.12 (0.97- 1.30)	322 deaths 1.21 (1.08- 1.35)**	978 deaths 1.12 (1.05- 1.19)**	978 deaths 1.18 (1.11- 1.26)**

* $p < 0.05$ ** $p < 0.01$

1 Alameda county rates for the California subcohort, Cook county rates for the Illinois subcohort, Wayne county rates for the Michigan subcohort, and New York City rates (Bronx, Kings, New York, and Queens counties) for the New York subcohort. For the entire cohort, the sums of observed and expected deaths for the four subcohorts were used to calculate a state-rate-based SMR. The U.S.-based SMR is also shown. Causes selected are those with statistically significant SMRs for 1960-96, in the entire cohort or in one or more subcohorts. Note that individuals may no longer be living in the same geographic area as when they were enrolled in the study.

2 Deaths from cancer of the trachea, bronchus, and lung

3 Deaths due to chronic and unspecified nephritis, renal failure, and other renal sclerosis

Supplementary Table: Mortality (through 12/31/96) for 1703 Dry-Cleaning Workers

SMRs for Selected Causes* in the Total Dry-Cleaning Cohort by Race and Gender

CAUSE	Nonwhite females	Nonwhite males	White females	White males
Tongue	2 deaths	3 deaths	no deaths	no deaths
cancer	10.2 (1.2- 36.9)†	8.81 (1.8- 25.8)‡		
Esophageal	4 deaths	5 deaths	2 deaths	3 deaths
cancer	2.82 (0.8- 7.2)	1.92 (0.6- 4.5)	3.42 (0.4-12.4)	2.68 (0.6- 7.8)
Intestinal	7 deaths	8 deaths	15 deaths	2 deaths
cancer	1.11 (0.4- 2.3)	2.28 (1.0- 4.5)†	2.03 (1.1- 3.4)	0.42 (0.1- 1.5)
Pancreatic	7 deaths	5 deaths	4 deaths	2 deaths
cancer	1.96 (0.8- 4.0)	2.01 (0.6- 4.7)	1.22 (0.3- 3.1)	0.79 (0.1- 2.9)
Lung	16 deaths	25 deaths	10 deaths	14 deaths
cancer	1.87 (1.1- 3.0)†	1.61 (1.0- 2.4)†	1.10 (0.5- 2.0)	0.93 (0.5- 1.6)
Cervical	7 deaths		5 deaths	
cancer	1.53 (0.6- 3.2)		2.62 (0.8- 6.1)	
Bladder	no deaths	4 deaths	3 deaths	3 deaths
cancer		4.15 (1.1-10.6)†	3.20 (0.7- 9.4)	1.80 (0.4- 5.3)
Other & unsp.	5 deaths	7 deaths	5 deaths	5 deaths
neoplasms	1.00 (0.3- 2.3)	2.03 (0.8- 4.2)	1.16 (0.4- 2.7)	1.67 (0.5- 3.9)
Heart	95 deaths	59 deaths	106 deaths	82 deaths
diseases	1.03 (0.8- 1.3)	0.81 (0.6- 1.0)	1.10 (0.9- 1.3)	0.84 (0.7- 1.0)
Circulatory	29 deaths	29 deaths	32 deaths	20 deaths
diseases	0.70 (0.5- 1.0)	1.09 (0.7- 1.6)	0.92 (0.6- 1.3)	0.79 (0.5- 1.2)
Respiratory	20 deaths	17 deaths	33 deaths	12 deaths
diseases	1.52 (0.9- 2.3)	1.16 (0.7- 1.9)	1.84 (1.3- 2.6)‡	0.66 (0.3- 1.2)
Pneumonia	10 deaths	14 deaths	13 deaths	6 deaths
	1.47 (0.7- 2.7)	1.96 (1.1- 3.3)†	1.61 (0.9- 2.7)	0.89 (0.3- 1.9)

Emphysema	2 deaths 3.76 (0.5-13.6)	1 death 0.74 (0.0- 4.1)	6 deaths 3.81 (1.4- 8.3)‡	1 death 0.31 (0.0- 1.7)
Digestive diseases	15 deaths 1.24 (0.7- 2.0)	7 deaths 0.74 (0.3- 1.5)	12 deaths 1.14 (0.6- 2.0)	15 deaths 1.62 (0.9- 2.7)
Stomach & duodenum	3 deaths 3.21 (0.7- 9.4)	1 death 0.83 (0.0- 4.6)	2 deaths 1.74 (0.2- 6.3)	5 deaths 3.11 (1.0- 7.3)†
Cirrhosis of liver	4 deaths 0.80 (0.2- 2.0)	4 deaths 0.93 (0.3- 2.4)	8 deaths 2.44 (1.1- 4.8)†	4 deaths 1.07 (0.3- 2.7)
Genitourinary diseases	11 deaths 1.16 (0.6- 2.1)	3 deaths 0.51 (0.1- 1.5)	7 deaths 1.45 (0.6- 3.0)	7 deaths 1.88 (0.8- 3.9)
Urinary calculi	no deaths	no deaths	no deaths	2 deaths 18.6 (2.3-67.2)‡
Accidents	1 death 0.14 (0.0- 0.8)†	4 deaths 0.35 (0.1- 0.9)†	3 deaths 0.49 (0.1- 1.4)	no deaths ‡
All cancers	80 deaths 1.31 (1.0- 1.6)†	73 deaths 1.49 (1.2- 1.9)‡	72 deaths 1.16 (0.9- 1.5)	46 deaths 0.95 (0.7- 1.3)
All causes	281 deaths 1.01 (0.9- 1.1)	215 deaths 0.98 (0.9- 1.1)	292 deaths 1.13 (1.0- 1.3)†	207 deaths 0.90 (0.8- 1.0)

* 95% CI given in parentheses. Selection includes all causes with a statistically significant SMR for a race-gender group or for the entire cohort, or causes of death of special interest in this cohort.

† 95% CI excludes the null value (1.0).

‡ 99% CI excludes the null value.