

Cohort mortality study of 57 000 painters and other union members: a 15 year update

Kyle Steenland, Sue Palu

Abstract

Objectives—To study mortality patterns in the largest existing cohort of painters.

Methods—15 years of follow up were added to a study of 42 170 painters and 14 316 non-painters based on union records. There were 23 458 deaths, compared with 5313 in the earlier follow up.

Results—Comparisons with the United States population showed significantly increased rates in painters for lung cancer (standardised mortality ratio (SMR) 1.23, 95% confidence interval (95% CI) 1.17 to 1.29), bladder cancer (SMR 1.23, 95% CI 1.05 to 1.43), liver cancer (SMR 1.25, 95% CI 1.03 to 1.50), and stomach cancer (SMR 1.39, 95% CI 1.20 to 1.59). However, in direct comparisons with non-painters only the excesses for lung cancer (SRR 1.23, 95% CI 1.11 to 1.35, increasing to 1.32, 95% CI 1.16 to 1.49 with 20 years latency) and bladder cancer (SRR 1.77, 95% CI 1.13 to 2.77) were confirmed. Some confounding by smoking may affect these two outcomes, particularly with external referents. Cirrhosis of the liver was increased for both painters and non-painters (SMRs 1.21, 95% CI 1.07 to 1.35, and 1.26, 95% CI 1.03 to 1.51, respectively), possibly indicating high alcohol consumption. Suicide (SMR 1.21, 95% CI 1.05 to 1.38) and homicide (SMR 1.36, 95% CI 1.04 to 1.75) were increased for painters but not for non-painters; neuropsychiatric diseases have been associated with painters in earlier studies.

Conclusions—The results suggest modest occupational risks for lung and bladder cancer; these results are consistent with existing publications. The International Agency for Research on Cancer has classified painting as an occupation definitely associated with cancer.

(*Occup Environ Med* 1999;56:315-321)

Keywords: lung cancer; bladder cancer; painters

There are about 700 000 painters in the United States (Rick Hackney, International Brotherhood of Painters and Allied Trades, personal communication). In a 1989 monograph,¹ the International Agency for Research on Cancer (IARC) determined that painting as an occupation was a definite (class I) cause of cancer. Lung cancer was most strongly and consistently associated with painting, but excesses were also noted for oesophageal, stomach, and bladder cancer. The primary basis for the judgement of the IARC was

three large cohort studies of painters, from the United States,² Denmark,³ and Sweden,⁴ although several case-control studies provided supporting evidence. The excess of lung cancer was judged to be about 40% and to be beyond what might be expected due to possible confounding by smoking.

Since the IARC review in 1989, there have been a few small cohort studies of painters and many case-control studies of various sites in which positive findings were reported for painters. These studies have tended to support the original findings of excess lung cancer and stomach cancer, to provide additional evidence of excess bladder cancer, excess of haematopoietic cancers (especially myeloma⁵⁻⁶ and non-Hodgkin's lymphoma⁷⁻⁸), and excess upper respiratory cancer.⁹⁻¹¹ Of note among the recent publications on bladder cancer is a large case-control study of bladder cancer among white men (2100 cases, 116 of whom were painters) in the United States¹² in which an odds ratio (95% confidence interval (95% CI)) of 1.5 (1.2 to 2.0) was found for painters after adjustment for smoking, and a meta-analysis of 27 case-control studies on bladder cancer¹³ which found an overall relative risk for bladder cancer of 1.48.

There are several known or suspected carcinogens in paint, but known carcinogens have been measured at only a low concentrations or have been present only in certain specialised kinds of painting. Most exposures are airborne, although in some cases dermal exposure may be important. Early in the century, arsenic, a lung carcinogen, was common in paint pigments, but was removed from most paints due to incidents of arsenic poisoning.¹⁴ Turpentine was used before the second world war, and was associated in some reports with kidney dysfunction and contact allergy.¹⁴ Turpentine was later replaced by organic solvents, some of which can cause haematopoietic and perhaps liver cancer, and are common in paints. The main solvents have been petroleum solvents, toluene, xylene, ketones, alcohols, esters, and glycol ethers. Benzene, a known leukaemogen, was used in the past,¹ but has been less common since the 1950s, and often has been present only at low concentrations. Metals in paint pigments include titanium oxide, chromium compounds, and iron compounds; lead was used in the past. Hexavalent chromium is a confirmed lung carcinogen, but airborne concentrations of hexavalent chromium are and have been low for most painters. There is some evidence that lead may also be associated with lung cancer. However, high exposure to airborne lead has not been a

National Institute for Occupational Safety and Health (NIOSH), Cincinnati, OH 45208, USA

K Steenland
S Palu

Correspondence to:
Mr Kyle Steenland, National Institute for Occupational Safety and Health (NIOSH), Cincinnati, OH 45208, USA.
Fax 001 513 841 486.

Accepted 23 October 1998

common exposure for most painters, occurring only in construction work in which lead paint was being removed by blasting. Benzidine and other aromatic amines, which can cause bladder cancer, have also been present in some paints,¹² but these are not common or high level exposures. Paint stripping involves exposure to methylene chloride,¹⁵ an animal carcinogen, but many painters do not perform this operation. Polyurethane paint, again not a common exposure for most painters, involves exposure to diisocyanates. There has been an increasing use of water based paints since the 1960s; these paints contain only small amounts of solvents and are thought to be less toxic.^{14 16}

In summary, although there are several known carcinogens which have been used by painters, most have been used in specialised settings or have resulted in only low levels of exposure. The organic solvents which have been commonly used, resulting in higher exposures, are not known human carcinogens, although some have been shown to cause cancer in animals. Additional research is needed to identify the exposures responsible for the increased cancer risk experienced by painters.

The United States cohort study cited by IARC is the largest existing cohort of painters, and is the subject of this paper. The study population is composed of 56 486 male union members of the International Brotherhood of Painters and Allied Trades (IBPAT). This union is composed primarily of painters, but the members from the allied trades are not painters. The cohort was originally followed up to the end of 1979, and included 5313 deaths.

The findings from the original follow up showed significant excesses among the painters compared with the United States population, for lung cancer (standardised mortality ratio (SMR) 1.18, 95% CI 1.06–1.32) and stomach cancer (1.36, 1.01 to 1.80), and nearly significant excesses for bladder cancer (1.26, 0.90 to 1.72), kidney cancer (1.41, 0.93 to 2.05), and liver cancer (1.56, 0.95 to 2.41).² Of the haematopoietic cancers, only leukaemias were reported (SMR 1.16, 95% CI 0.82 to 1.16). In a direct comparison of union locals composed of painters compared with other trades, lung cancer, bladder cancer, and leukaemia were significantly higher among the painters (SRRs 1.46, 2.30, and 2.41). However, in the direct comparison liver, stomach, and kidney cancers were no longer significantly increased (SRRs 1.26, 1.17, and 1.29).

We have extended follow up of this cohort by an additional 15 years, to the end of 1994, covering 23 458 deaths, with a goal of evaluating whether the observed cancer excesses have persisted over time.

Methods

The cohort is composed of members of the Painters Union with at least 1 year of union membership who had been born before 1940 and were alive at the end of 1975. Members could have been retired from 1975; retired members were kept on the union roster. No new union members after 1979 were included in the cohort.

Most union members were from local unions (each local union is defined by union members in a small geographical area) composed primarily of painters, but the rest were from other local unions composed primarily of other types of workers. Cohort members were identified from local unions located in four states (New York, Missouri, California, and Texas). Locals were characterised as mixed local composed primarily of painters, and speciality locals composed mainly of other allied trades (note that mixed does not imply a mixture of painters and non-painters, but a mixture of different types of painters). The titles of specialty locals included automobile, industrial, civil, glass, sign, paint, carpet, scenic, wood, paper, dry-wall, warehouse, and metal workers. The original analysis of this cohort² analysed the data separately for the mixed and specialty locals. Further discussion with the Painters Union for the present study, however, led to a determination that the locals entitled industrial, automobile, sign, and scenic were in fact also primarily composed of painters. The industrial painters painted bridges, painted steel in production factories, or painted on construction sites. Automobile, sign, and scenic locals were composed of painters who painted automobiles, signs, or special painting jobs. Hence, members of these locals (n=8029) were added to the mixed locals (n=34 141) and these subjects were all considered to be painters. The remaining specialty locals were confirmed to have been primarily non-painters (n=14 316). Workers in civil locals were government workers often driving trucks, workers in glass locals worked manufacturing glass, workers in paint locals worked paint manufacturing plants (but in fact most of these workers were judged to have had little potential exposure to paint), workers in carpet locals were carpet and tile layers, workers in wood locals were skilled wood finishers, workers in paper locals were paper hangers, workers in drywall locals were drywall finishers, workers in warehouse locals worked in warehouses in paint manufacturing companies or convention halls, whereas workers in metal locals worked installing window frames and other finished metal.

It is clear, however, that this classification of painters and non-painters is not definitive; the speciality locals are likely to have included a few painters, whereas the mixed, industrial, automobile, sign, and scenic locals are likely to likewise have included a few non-painters.

Vital status follow up of this cohort to the end of 1994 was conducted through the Internal Revenue Service, the Health Care Finance Administration, the Post Office, the Social Security Administration Death Tapes, and the National Death Index. Causes of death were obtained directly from death certificates or from computerised death certificate data for four states (New York, Missouri, California, and Texas) and one city (New York City). Cause of death information was coded with standard coding conventions in the same way for both the observed deaths and the deaths in the United States comparison population. Cause of death information extended to the

Table 1 Descriptive data for painters and non-painters

	Painters (n=42170)	Non-painters (n=14316)
Mean year of birth (range)	1919 (1865–1941)	1925 (1869–1939)
Mean year of union entry (range)	1957 (1900–1979)	1962 (1900–1979)
Mean years of follow up after 1975 (range)	14 (0–21)	15 (0–21)
Vital status as of 12/31/94 (%):		
Alive	21117 (50)	9011 (63)
Unknown*	2794 (7)	1058 (7)
Dead†	18259 (43)	4247 (30)
Mean year of death (range)	1985 (1975–1995)	1986 (1975–1995)

*Person-time ended at end of last known date alive.

†952 Study subjects were known to have died after the study end date of 12/31/94.

end of 1994, the study end date. Race was not known for all cohort members. Death certificate data indicated that the overwhelming majority of union members were white (98%),² and in the analyses all subjects were considered to be white. About 99% of union members were men,² and we restricted our analyses to them.

Mortality analyses consisted of standard life table analyses comparing the mortality of the painters with that of the population of the United States, after stratification of deaths and person-years at risk by potential confounders (age, calendar time).¹⁷ Rates were available for 99 different categories of death. The measure of effect was the SMR. Also, standardised rate ratios (SRRs) were calculated for several cancer outcomes in a direct comparison between painters and non-painters through Poisson regression. Analyses were conducted for several cancer outcomes by time since first in the union (potential latency). No data were available on duration of union membership.

Death certificates include information on other causes of death besides underlying cause. We used this additional information in a multiple cause proportionate mortality analysis of

this cohort, in which the observed proportion of deaths with a specific cause mentioned anywhere on the death certificate is compared with the expected proportion based on all United States deaths.¹⁸ Multiple cause of death data were available for 13 928 deaths (62% of all deaths); some computerised data bases—for example, Missouri, New York City for certain years—provided only underlying cause of death, and deaths from these data bases were therefore excluded from multiple cause analyses. The lack of multiple cause death on all deaths in the cohort prohibited a cohort analysis with person-time by multiple cause data; instead we conducted a proportional mortality rate (PMR) analysis on those deaths with multiple cause data, assuming the deaths for which we did have multiple cause data were a representative sample of all deaths. Multiple cause analysis is particularly useful in detecting excesses for causes that are not likely to be listed as the underlying causes, such as chronic but not usually fatal conditions. We conducted this analysis to investigate any possible excess of renal disease on the death certificate, because renal disease has been associated with solvents,¹⁹ although in the one study when data were presented by type of solvent exposure, exposure to solvents in paints was not associated with risk of renal disease.²⁰ We were also interested in mental disorders, which have sometimes been associated with painters and which are much more common as contributory rather than underlying causes of death.¹⁸

No direct information was available on the smoking habits of study subjects. Indirect information on the smoking habits of painters was available from the 1970s and 1980s from

Table 2 SMR results for selected causes

Cause	Painters SMR (95% CI) (observed)	Non-painters SMR (95% CI) (observed)
Pharyngeal cancer	1.15 (0.85 to 1.52)(49)	0.45 (0.17 to 0.98)(6)
Oesophageal cancer	1.12 (0.92 to 1.35)(110)	0.83 (0.54 to 1.22)(25)
Stomach cancer	1.39 (1.20 to 1.59)(197)	1.51 (1.14 to 1.97)(56)
Lung cancer	1.23 (1.17 to 1.29)(1746)	1.02 (0.92 to 1.12)(422)
Laryngeal cancer	0.97 (0.71 to 1.29)(48)	0.97 (0.53 to 1.62)(14)
Liver cancer (specified+unspecified)*	1.25 (1.03 to 1.50)(119)	0.93 (0.60 to 1.38)(24)
Bladder cancer	1.23 (1.05 to 1.43)(166)	0.74 (0.46 to 1.11)(22)
Kidney	1.06 (0.86 to 1.29)(100)	0.77 (0.48 to 1.18)(21)
Hodgkin's lymphoma	1.30 (0.74 to 2.11)(16)	0.54 (0.06 to 1.93)(4)
Non-Hodgkin's lymphoma†	1.06 (0.89 to 1.25)(137)	0.95 (0.66 to 1.32)(34)
Leukaemia	0.92 (0.78 to 1.11)(138)	0.63 (0.40 to 0.94)(24)
Myeloma	0.97 (0.75 to 1.24)(64)	0.90 (0.52 to 1.46)(16)
Ischaemic heart disease	0.97 (0.95 to 1.00)(5871)	0.92 (0.87 to 0.97)(1297)
Cardiomyopathy‡	1.17 (0.99 to 1.37)(146)	1.81 (1.41 to 2.28)(71)
Hypertension with heart disease	1.19 (1.01 to 1.39)(160)	1.40 (1.03 to 1.85)(49)
Pneumonia	1.14 (1.06 to 1.24)(641)	1.04 (0.86 to 1.25)(113)
Emphysema	1.13 (0.99 to 1.28)(232)	0.81 (0.57 to 1.12)(38)
Bronchitis	1.11 (0.82 to 1.47)(49)	0.89 (0.37 to 1.68)(8)
Asbestosis	4.70 (2.57 to 7.89)(14)	0.00 (0.0 to 4.55)(0)
Other non-malignant respiratory disease§	1.07 (0.99 to 1.14)(799)	0.90 (0.77 to 1.05)(157)
Liver cirrhosis	1.21 (1.07 to 1.35)(303)	1.26 (1.03 to 1.51)(109)
Alcoholism	1.11 (0.80 to 1.51)(42)	0.56 (0.24 to 1.09)(8)
Suicide	1.21 (1.05 to 1.38)(219)	1.06 (0.82 to 1.36)(63)
Homicide	1.36 (1.04 to 1.75)(62)	0.90 (0.52 to 1.44)(17)
HIV related¶	1.79 (1.08 to 2.79)(19)	1.84 (0.84 to 3.49)(9)
Falls	1.18 (0.98 to 1.41)(122)	1.20 (0.79 to 1.74)(27)
All cancers	1.12 (1.09 to 1.15)(4674)	0.96 (0.90 to 1.01)(1086)
All causes	1.04 (1.03 to 1.06)(18 259)	0.99 (0.96 to 1.02)(4247)

*ICD-9 codes 155.0, 155.1, 155.2, 156 (this category includes cancers of bile duct and gall bladder; about 25% of these cancers involved the extrahepatic biliary tract; none were gall bladder cancers (ICD-9 15600)).

†ICD-9 codes 200, 202.

‡ICD-9 code 425.

§ICD-9 code 470–479, 494–499, 504, 506–519. Respiratory infection, emphysema, asthma, bronchitis, and pneumoconioses have their own categories. This category is primarily chronic obstructive pulmonary disease.

¶ICD-9 42–44.

Table 3 SMR results for cancers, 20 years since first union membership

Cause	Painters SMR (95% CI) (observed)	Non-painters SMR (95% CI) (observed)
Pharyngeal cancer*	0.99 (0.67 to 1.40)(31)	0.37 (0.08 to 1.08)(3)
Oesophageal cancer	1.15 (0.92 to 1.42)(86)	1.02 (0.62 to 1.58)(20)
Stomach cancer	1.46 (1.25 to 1.70)(167)	1.75 (1.27 to 2.35)(44)
Lung cancer	1.24 (1.18 to 1.31)(1360)	0.96 (0.85 to 1.08)(264)
Laryngeal cancer	0.92 (0.64 to 1.28)(35)	1.28 (0.60 to 2.24)(12)
Liver cancer (specified+unspecified)	1.17 (0.95 to 1.44)(90)	1.01 (0.60 to 1.60)(18)
Bladder cancer	1.25 (1.06 to 1.47)(146)	0.84 (0.51 to 1.31)(19)
Kidney	0.97 (0.76 to 1.22)(71)	0.67 (0.34 to 1.17)(12)
Hodgkin's lymphoma	1.17 (0.56 to 2.15)(10)	0.48 (0.01 to 2.67)(1)
Non-Hodgkin's lymphoma	1.10 (0.91 to 1.32)(110)	1.07 (0.70 to 1.57)(26)
Leukaemia	1.11 (0.74 to 1.08)(111)	0.56 (0.31 to 0.92)(15)
Myeloma	1.01 (0.76 to 1.32)(54)	0.97 (0.50 to 1.69)(12)
All cancers	1.12 (1.08 to 1.16)(3748)	0.99 (0.92 to 1.06)(762)
All causes	1.05 (1.03 to 1.07)(15163)	1.02 (0.98 to 1.06)(3025)

*For ICD codes see footnote to table 2.

two large surveys.^{21 22} Confounding by smoking for lung cancer and bladder cancer was assessed indirectly by a method described by Axelson and Steenland.²³ This method relies on estimates of smoking prevalence among the exposed cohort and the non-exposed population (actual smoking prevalences being unknown), and assumes known relative risks for specific cancers by smoking habit—for example, current, former, never. Expected rate ratios (RRs) due to differences in smoking habits can then be calculated and compared with observed RRs. In this adjustment we assumed relative risks for lung cancer of 10, 15, and 20 for smoking <19, 20–39, and ≥40 cigarettes a day, and a relative risk of 5 for former smokers.

Results

Table 1 provides some descriptive statistics for painters and non-painters. The non-painters were generally younger and joined the union later. Both painters and non-painters joined the union rather late in age (mean age 38 and 37, respectively), implying that earlier work in their trade may have been common.

We were unable to determine the current vital status of 7% of the cohort. Person-time for these cohort members ended when they were last known alive, which was generally at the end of the previous follow up in 1979. Cause of death data could not be obtained for 5.1% of the known deaths in the cohort. Assuming that these people are indeed dead, and assuming that their causes of death would be distributed between the specific causes in the same fashion as the deaths with known causes, then our observed SMRs are underestimated by about 5%. The missing causes would not be expected to appreciably affect internal comparisons of painters with non-painters.

Table 2 provides selected SMR results for painters and non-painters. Results are presented for causes of death which were of initial interest or of later interest because they showed a significant increase among either painters or non-painters. Among causes of initial interest are not only the suspected cancer sites but other causes of death possibly related to non-occupational risk factors—for example, non-malignant respiratory disease and smoking.

The results in table 2 indicate that painters have significant excesses of lung, stomach, bladder, and liver cancer compared with the

United States population. Non-painters also had a significant excess of stomach cancer, but did not show excesses of other cancers.

Results for non-cancer outcomes indicated a small excess of non-malignant respiratory diseases among painters, but not among non-painters. Asbestosis was significantly increased in painters, but not in non-painters, based on 14 deaths. This excess is not surprising given the inclusion in the cohort of construction painters who were likely to have had exposure to asbestos in the past. However, painters did not show excesses of cancer of the peritoneum (SMR 0.60, 95% CI 0.30 to 1.07, 11 deaths) or cancer of other parts of the respiratory system (SMR 1.09, 95% CI 0.60 to 1.77, 15 deaths), sites where mesotheliomas might be expected.

Both painters and non-painters had significant excesses of death from cirrhosis of the liver; the excess was more marked among non-painters. No corresponding excesses were found for death from alcoholism. Painters, but not non-painters, showed significant excess deaths from suicide and homicide.

Multiple cause analyses indicated a deficit of deaths from both acute and chronic renal disease for painters (PMR 0.77, 0.62 to 0.93, 96 deaths, and PMR 0.93, 0.84 to 1.01, 469 deaths, respectively). The corresponding PMRs for non-painters were also not remarkable (PMRs 1.13, 95% CI 0.78 to 1.60, 32 deaths, and PMR 0.93, 95% CI 0.76 to 1.13, 105 deaths for acute and chronic renal disease, respectively). The multiple cause result for painters for other mental disorders (9th revision of the international classification of diseases (ICD-9) 290–302, 204–319, including dementia, psychoses, delirium, and schizophrenia, was not increased (PMR 0.93, 95% CI 0.84 to 1.02, 397 deaths), as was the case for other diseases of the nervous system and sense organs (ICD-9 320–337, 341–389, including Parkinson's disease, multiple sclerosis, epilepsy, peripheral neuropathies, and disorders of eyes and ears, PMR 0.88, 95% CI 0.81 to 0.96, 516 deaths).

Table 3 shows analyses of cancer mortality based on data restricted to ≥20 years since entering the union (potential latency). These results differed little from the overall results, as 70% of cancer deaths occurred ≥20 years after entering the union.

Table 4 Poisson regression results comparing painters with non-painters*

Cause	Rate ratio (95% CI)	Rate ratio ≥ 20 years since joining union (95% CI)
Stomach cancer†	0.92 (0.68 to 1.25)	0.87 (0.62 to 1.22)
Lung cancer	1.23 (1.11 to 1.35)	1.32 (1.16 to 1.51)
Liver cancer (specified+unspecified)	1.36 (0.87 to 2.11)	1.16 (0.69 to 1.93)
Bladder cancer	1.77 (1.13 to 2.77)	1.55 (0.96 to 2.51)

*Age was categorised in 5 year intervals (<50, 50–54, ..., 75–80, ≥ 80), calendar time in four intervals (1975–1979, 1980–84, 1985–1989, 1990–1994).

†For ICD-9 codes see footnote to table 2.

Internal comparisons between painters and non-painters were conducted through Poisson regression for cancers which were increased in the external analyses (stomach, lung, liver, and bladder). The results are shown in table 4. Lung and bladder cancer were significantly increased, whereas liver cancer was non-significantly increased. The increase for lung cancer was slight when the analysis was restricted to those with ≥ 20 years since first joining the union (latency).

With regard to possible confounding of cancer SMRs by smoking, two national surveys indicate that painters have smoked more than the general population. Brackbill *et al*²¹ reported on a national probability sample of painters in 1978–90. Among painters in construction and maintenance (total estimated at 477 000), 48.9% were current smokers, 22.9% were former smokers, and 28.2% were never smokers. The corresponding figures for white men in the entire United States population were 36.3%, 26.1%, and 37.6%. Among painters who currently smoke, 32.4% smoked <20 cigarettes a day, 51.5% smoked 20–39 cigarettes a day, and 16.1% smoked >39 cigarettes a day. The corresponding figures for the entire population of white men were 25.5%, 53.6%, and 20.9%.

Stellman *et al*²² reported on 1263 male painters aged 45–70 in the American Cancer Society CPS-II population, surveyed in 1982. The percentage of current, former, and never cigarette smokers are 35.7%, 39.8%, and 24.5%. The corresponding figures for the entire male survey population were 23.2%, 43.6%, and 33.2% (estimated from table VI²³). Amount smoked among current smokers was comparable for painters and the entire population.

With an indirect adjustment for smoking,²³ the data of Brackbill *et al* suggest that a lung cancer RR of 1.14 for painters versus the national population would be expected based on smoking differences alone. With the data from Stellman *et al*, the estimated RR for lung cancer due to smoking differences alone would be 1.16.

Similar adjustments for bladder cancer, much more weakly related to smoking, would lead to estimated RRs due to confounding by smoking in the order of 1.05.

The adjustments apply only to comparisons with the national population. Confounding by smoking would be expected to be less important in internal comparisons between painters and non-painters (both groups being blue collar workers), who would be expected to share similar smoking habits.

Discussion

Our results show a significant but modest increase in lung (SMR 1.23, 95% CI 1.17 to 1.29) and bladder cancer (1.23, 95% CI 1.05 to 1.50), with an external (United States) comparison population with follow up to the end of 1994. The corresponding SRRs were 1.23 (95% CI 1.11 to 1.35) and 1.77 (95% CI 1.13 to 2.77), respectively, with an internal (non-painter) comparison. These increases, although relatively small, are consistent with the publications on painters.

Analyses of lung cancer SMRs among painters by 5-year period showed an increasing trend (SMRs of 1.10 (385 deaths), 1.23 (458 deaths), 1.23 (476 deaths), and 1.38 (427 deaths), for the periods 1975–9, 1980–4, 1985–9, and 1990–4, respectively). The corresponding SMRs for bladder cancer were 1.34 (50 deaths), 1.18 (41 deaths), 1.15 (38 deaths), and 1.24 (37 deaths), and for liver cancer they were 1.51 (35 deaths), 1.02 (24 deaths), 1.12 (28 deaths), and 1.38 (32 dead); neither cancer showed a consistent trend.

By comparison, the earlier findings of Matanowski *et al*,² based on the period 1975–9 but with fewer union members classified as painters than in the present analysis (33 098 *v* 42 170), showed SMRs of 1.18 (326 deaths) for lung cancer, 1.26 (40 deaths) for bladder cancer, and 1.56 (20 deaths) for liver cancer, all slightly higher than our own findings for 1975–9.

In our own data, direct comparisons (by Poisson regression) of painters to non-painters by period showed SRRs for lung cancer of 1.25 (95% CI 0.97 to 1.62) for 1975–9, and 1.22 (95% CI 1.08 to 1.38) for 1980–94. The SRRs for liver cancer were 1.30 (95% CI 0.50 to 3.35) for 1975–9 and 1.35 (95% CI 0.82 to 2.23) for 1980–94. The SRRs for bladder cancer were 9.11 (95% CI 1.26 to 66.7) for 1975–9, and 1.39 (95% CI 0.87 to 2.22) for 1980–94, with unstable numbers for the 1975–9 period due to too few deaths from bladder cancer among non-painters. These results indicate that our SRR findings for the 1975–9 period (corresponding roughly to the results of Matanowski *et al*²) do not differ from the period 1980–94 for lung cancer or liver cancer, but numbers are unstable for bladder cancer.

For lung cancer, smoking differences between painters and the United States population may explain part of the lung cancer excess we found for painters, and to a lesser degree may explain part of the excess SMRs for bladder cancer, when the external United States comparison group was used. Indirect adjustments for smoking suggest that differences in smoking between the painters in large United States surveys, and the overall United States comparison population, would account for about a 15% excess for lung cancer, and a 5% excess for bladder cancer. Hence, neither the lung nor bladder cancer excess found in this study is likely to be fully explained by smoking, in the light of this adjustment. However, we do not have direct smoking data on our cohort,

limiting the usefulness of this kind of indirect adjustment.

Such confounding by smoking is less likely to have an effect on internal comparisons (SRRs) between painters and non-painters, as both are blue collar workers likely to share similar smoking habits. For example, the lung cancer RR of 1.323 comparing painters with non-painters (both with 20 years potential latency) is unlikely to be explained by confounding by smoking.

The data for other diseases highly related to smoking do not show strong consistently high risks for painters compared with the United States population, also arguing against confounding by smoking. Oesophageal cancer, laryngeal cancer, emphysema, bronchitis, and other non-malignant respiratory disease (primarily chronic obstructive pulmonary disease (COPD)) are all known to have a relative risk of the order of 10 for smokers versus non-smokers, and would be expected to be increased in our SMR analyses if painters smoked much more heavily than the United States population; the RRs for these outcomes were 1.12, 0.97, 1.13, 1.11, and 1.07 respectively. These results suggest that if there is a confounding effect by smoking, it is not severe.

The lung cancer excess among painters could also be partly attributable to the use of spackling compounds which formerly contained asbestos, although the level of past exposure to respirable asbestos for painters is not known. The SMR for asbestosis was significantly increased (SMR 4.70). However, only 14 deaths were found due to asbestosis among the 18 989 deaths of painters, which does not indicate widespread exposure to high levels of asbestos. Furthermore, there were no excesses of cancer in sites likely to include mesothelioma (peritoneum, other parts of the respiratory system), another disease caused almost exclusively by asbestos.

A nested case-control study of New York painters and non-painters within our cohort was conducted by other investigators in the early 1980s,²⁴ based on the earlier follow up of this cohort. This study was based on interviews (primarily with next of kin for cases) collected by posted questionnaires, and was adjusted for smoking. Painters and non-painters had similar smoking habits. A threefold risk of lung cancer was found for being a painter, after adjustment for smoking, which increased for painters who reportedly did not wear respiratory protection. Limitations of these results include a low response rate and the reliability of detailed work history data collected by posted questionnaires from next of kin.

Our data also indicated a modest but significant increase in liver cancer in painters compared with the United States population (SMR 1.25); non-painters did not show such an increase. However, the importance of this finding is lessened because painters did not show an increase in liver cancer in a direct comparison with non-painters, especially in the comparison with ≥ 20 years latency where an effect would be most likely. Also, both painters and non-painters had excesses of cir-

rhosis, which is associated both with liver cancer and alcohol consumption. Although cirrhosis could conceivably be related to exposure to solvents among painters, the fact that non-painters had a significant increase in cirrhosis compared with the United States population lessens the probability that the excess of cirrhosis among painters is due to an occupational exposure.

Stomach cancer was in excess for both painters and non-painters, compared with the United States population. However, painters did not have an excess of stomach cancer when compared directly with non-painters. The excess of stomach cancer versus the United States population for both painters and non-painters may be due to non-occupational risk factors—such as diet.

Both painters and non-painters had excesses of death from falls compared with the United States population. It is likely that both these excesses are partly due to occupational factors, given that working in high places is common for both groups.

Painters had modest but significantly higher rates of suicide and homicide than the United States population, but non-painters did not. We have no explanation why this should be, but solvent exposure could possibly play a part. Such an interpretation is speculative. For suicide, there is some published evidence that painters have an increased rate of neuropsychiatric disorders.²⁵

In summary, the modest excesses of lung and bladder cancers that we have found among painters are consistent with an occupational aetiology and are consistent with the scientific literature. Confounding by smoking may play a part in these excesses, particularly for lung cancer which is highly related to smoking, but any such confounding is unlikely to completely explain these findings. The main weaknesses in our data are the lack of detailed work history and the lack of any specific exposure information; the agents responsible for the cancer excesses found here are unknown (see introduction on this point). Further investigation of these excesses, through nested case-control studies, may be warranted.

Reviewers Bob Roscoe (NIOSH) and Paolo Boffetta (IARC) kindly provided comments. Rick Hackney and the union (IBAT) provided important help.

- 1 International Agency for Research on Cancer. Some organic solvents, resin monomers, and related compounds, pigments, and occupational exposures in paint manufacturing and painting. Lyon, France: IARC, 1989. (Monograph 47.)
- 2 Matanowski G, Stockwell H, Diamond E, et al. A cohort mortality study of painters and allied tradesmen. *Scand J Work Environ Health* 1986;12:16–21.
- 3 Englund A. Cancer incidence among painters and some allied trades. *J Toxicol Environ Health* 1980;6:1267–1273.
- 4 Olsen J and Jensen O. Occupation and risk of cancer in Denmark, an analysis of 93,810 cancer cases. *Scand J Work Environ Health* 1987;13(suppl 1):1–91.
- 5 Bethwaite P, Pearce P, Fraser J. Cancer risks in painters: study based on the New Zealand Cancer Registry. *Br J Ind Med* 1990;47:742–6.
- 6 Demers P, Vaughan T, Koepsell T, et al. A case-control study of multiple myeloma and occupation. *Am J Ind Med* 1993;23:629–39.
- 7 Schumacher M, Delzell E. A death certificate case-control study of non-Hodgkin's lymphoma and occupation in men in North Carolina. *Am J Ind Med* 1988;13:317–30.

- 8 Scherr P, Hutchison G, Neiman R. Non-Hodgkin's lymphoma and occupational exposure. *Can Res* 1992;52:4409s-5503s.
- 9 Wortley P, Vaughan T, Cavis S, *et al.* A case-control study of occupational risk factors for laryngeal cancer. *Br J Ind Med* 1992;49:837-44.
- 10 Huebner W, Schoenberg J, Kelsey J, *et al.* Oral and pharyngeal cancer and occupation: a case-control study. *Epidemiology* 1992;3:300-9.
- 11 Skov T, Weinger J, Pukkala E, *et al.* Risk for cancer of the pharynx and oral cavity among male painters in the Nordic countries. *Arch Environ Health* 1993;48:176-80.
- 12 Silverman D, Levin L, Hoover R, *et al.* Occupational risks of bladder cancer in the United States: I. White men. *J Natl Cancer Inst* 1989;81:1472-80.
- 13 Yagamuchi N, Watanabe S, Okubo T, *et al.* Work-related bladder cancer risks in male Japanese workers. *J Cancer Res* 1991;82:624-31.
- 14 Wieslander G, Norback D, Edling C. Occupational exposure to water based paint and symptoms from the skin and eyes. *Occup Environ Med* 1994;51:181-6.
- 15 Vincent R, Poirot P, Subra I, *et al.* Occupational exposure to organic solvents during paint stripping and painting operations in the aeronautical industry. *Arch Occup Environ Health* 1994;65:377-80.
- 16 Van Faasen A, Borm P. Composition and health hazards of water-based construction paints: results from a survey in the Netherlands. *Environ Health Perspect* 1991;92:147-54.
- 17 Steenland K, Beaumont J, Spaeth S, *et al.* New developments in the NIOSH life table analysis system. *J Occup Med* 1990;32:1091-8.
- 18 Steenland K, Nowlin S, Ryan B, *et al.* Use of multiple-cause mortality data in epidemiologic analyses. *Am J Epidemiol* 1992;136:855-62.
- 19 Stengel B, Cenee Y, Lmiasset J, *et al.* Organic solvent exposure may increase the risk of glomerular nephropathies with chronic renal failure. *Int J Epidemiol* 1995;24:427-34.
- 20 Steenland K, Thun M, Ferguson B, *et al.* Occupational and other exposures associated with end-stage renal disease: a case-control study. *Am J Public Health* 1990;80:153-7.
- 21 Brackbill R, Frazier T, Shiling S. Smoking characteristics of US workers, 1978-80. *Am J Ind Med* 1988;13:5-41.
- 22 Stellman S, Boffetta P, Garfinkel L. Smoking habits of 800 000 American men and women in relation to their occupations. *Am J Ind Med* 1988;13:43-58.
- 23 Axelson O, Steenland K. Indirect methods of assessing tobacco use in occupational studies. *Am J Ind Med* 1988;13:105-18.
- 24 Stockwell J, Matanowski G. A case-control study of lung cancer in painters. *J Occup Med* 1985;27:1125-6.
- 25 Brackbill R, Maizlish N, Fischbach T. Risk of neuropsychiatric disability among painters in the US. *Scand J Work Environ Health* 1990;16:182-8.

Vancouver style

All manuscripts submitted to *Occup Environ Med* should conform to the uniform requirements for manuscripts submitted to biomedical journals (known as the Vancouver style.)

Occup Environ Med, together with many other international biomedical journals, has agreed to accept articles prepared in accordance with the Vancouver style. The style (described in full in the *JAMA*[1]) is intended to standardise requirements for authors, and is the same as in this issue.

References should be numbered consecutively in the order in which they are first mentioned in the text by Arabic numerals on the line in square brackets on each occasion the reference is cited (Manson[1] confirmed other reports[2][3][4][5]). In future references to papers submitted to *Occup Environ Med* should include: the names of all

authors if there are three or less or, if there are more, the first three followed by *et al*; the title of journal articles or book chapters; the titles of journals abbreviated according to the style of *Index Medicus*; and the first and final page numbers of the article or chapter. Titles not in *Index Medicus* should be given in full.

Examples of common forms of references are:

- 1 International Committee of Medical Journal Editors. Uniform requirements for manuscripts submitted to biomed journals. *JAMA* 1993;269:2282-6.
- 2 Soter NA, Wasserman SI, Austen KF. Cold urticaria: release into the circulation of histamine and eosinophil chemotactic factor of anaphylaxis during cold challenge. *N Engl J Med* 1976;294:687-90.
- 3 Weinstein L, Swartz MN. Pathogenic properties of invading micro-organisms. In: Sodeman WA Jr, Sodeman WA, eds. *Pathologic physiology, mechanisms of disease*. Philadelphia: W B Saunders, 1974:457-72.