Mortality and incidence of cancer in a cohort of Swedish chimney sweeps: an extended follow up study

Bradley A Evanoff, Per Gustavsson, Christer Hogstedt

Abstract

Despite 200 years of efforts to regulate safety in this occupation, chimney sweeps have increased mortality from cancer, ischaemic heart disease, and respiratory disease. Mortality and incidence of cancer were examined in a cohort of 5542 Swedish chimney sweeps employed through their national trade union at any time between 1918 and 1980. Previous studies of this cohort found increased risks of ischaemic heart disease, respiratory disease, accidental deaths, and various neoplasms. By increasing follow up, we sought to increase the power of the study and examine disease time trends. Mortality analysis was extended 7·5 years to cover the period 1951–90; cancer incidence analysis was extended six years to cover the period 1958–87. New findings include increased incidence and mortality of prostate cancer (SMR 169, 95% CI 106–256, 22 observed) and increased incidence of total haematolymphatic cancers (SIR 151, 95% CI 106–209, 36 observed). When only the most recent follow up period was analysed, previously observed risks persisted for total lung cancer (SIR 178, 95% CI 99–293), oat cell lung cancer (SIR 240, 95% CI 103–472), bladder cancer (SIR 247, 95% CI 131–422), and oesophageal cancer (Obs/Exp = 2/1·1). Mortality from ischaemic heart disease (SMR 98, 95% CI 76–123) and respiratory disease (SMR 111, 95% CI 56–199) declined during recent follow up, although significant excess mortality remained during analysis of the entire study period (ischaemic heart disease SMR 128, 95% CI 112–145; respiratory disease SMR 159, 95% CI 115–213). In analyses of the entire study period, risks of ischaemic heart disease and lung, bladder, and oesophageal cancer were adjusted for smoking; oesophageal cancer was also adjusted for use of alcohol. All risks remained significantly raised. Exposure-response analyses showed significant positive associations between duration of employment and risks for mortality from lung, oesophageal, and total cancer. Chimney sweeps remain at increased risk for cancers of the lung, oesophagus, and bladder. Our study supports a causal role for exposure to chimney soot, which contains carcinogens including polycyclic aromatic hydrocarbons. Extended follow up of this cohort now shows increased risks of prostate and haematolymphatic cancers.

(*British Journal of Industrial Medicine* 1993;50:450–459)

Sir Percivall Pott's classic description of scrotal cancer among London chimney sweeps, published in 1775, is generally regarded as the first description of an occupationally induced cancer.[1] As in Pott's day, the modern chimney sweeps' primary exposure is to soot, a complex mixture produced by the combustion of coal, coke, oil, or wood. Chimney soot is rich in polycyclic aromatic hydrocarbons (PAHs), a group of compounds with well documented carcinogenic action: it is likely that these compounds are responsible for part of the excess risk of cancer in sweeps and other occupational groups exposed to combustion byproducts.[2,3] Assessments of industrial hygiene have shown that chimney soot contains potentially carcinogenic metals including arsenic, chromium, cadmium, and nickel; chimney sweeps are also exposed to combustion gases such as sulphur dioxide and carbon monoxide, asbestos from pipe and furnace insulation, and organic solvents used as degreasing agents.[4–7] Engineering controls and personal protective equipment have not been effectively used to eliminate these exposures. Although steam and water cleaning are used in large industrial stacks, most chimneys are swept dry. Enclosed vacuum sweeping systems have not been successful in controlling exposures; respirators are not widely used because of the high physical demands of sweeping and the need to work in tight spaces.

Division of Occupational Medicine, National Institute of Occupational Helath, Solna, Sweden
B A Evanoff, C Hogstedt
Department of Occupational Health, Karolinska Hospital, Stockholm, Sweden
B A Evanoff, P Gustavsson, C Hogstedt
Clinical Scholars Program and Occupational and Environmental Health Program, University of Washington, Seattle USA
B A Evanoff

Despite more than 200 years of efforts to regulate safety in this occupation (dating at least from the 1788 British *Act for the Better Regulation of Chimney Sweeps and their Apprentices*[8]) chimney sweeps remain at high risk for various cancers as well as cardiopulmonary diseases. Previous studies of this large cohort of Swedish chimney sweeps found excess incidence of oesophageal cancer, lung cancer, and bladder cancer as well as excess mortality from cardiovascular diseases, respiratory diseases, and violent deaths.[9–11] Excesses in mortality from lung cancer, stomach cancer, and ischaemic heart disease were noted among Danish chimney sweeps[12 13] and a Yugoslavian study found excesses of lung and laryngeal cancer.[14]

The present study extends the follow up of the Swedish chimney sweep cohort by 7·5 years for mortality and six years for incidence of cancer; it is thus possible to better define recent trends in mortality and incidence of cancer among the sweeps that may be due to changes in their working environment. As well as longer follow up, this study more fully evaluated the effects of potential confounding by alcohol and tobacco use.

Materials and methods

STUDY POPULATION

The study population included all registered members of the Swedish Chimney Sweeps Trade Union, from its inception in 1918 until 1980. This union represented about 95% of all chimney sweeps in Sweden, giving this study the character of a national survey. Every member's name, birthdate, dates of union membership, and years of active employment were obtained from the union's records. Civil registration numbers, a unique number assigned to each Swedish citizen, were obtained from the union records or by writing to the subjects' birth parishes. A total of 5542 men were members of the union between 1918 and 1980. For the mortality study, the cohort was followed up until 30 June 1990. By this time, 60 original cohort members had emigrated, nine were registered as officially missing, and 33 had been lost to follow up. Follow up of vital status was thus obtained for 98·2% of the cohort. Excluded from analysis were 127 members of the cohort who died before 1951, as reliable cause of death and comparison mortality figures were not available. The mortality analysis was thus performed on the remaining 5313 subjects who contributed person-years between 1 January 1951 and 30 June 1990. Mortality analysis was restricted to persons dying before the age of 80.

VITAL STATUS AND CAUSE OF DEATH

Follow up for vital status was accomplished by linking the cohort to the computerised National Cause of Death Registry and to the computerised register of the living population in Sweden; those not found in either register were traced through individual parish offices. Correspondence of both name and civil registration number was checked for every subject during the linkage process. Death certificates were obtained for all deaths occurring from 1951–88 inclusive; date and official cause of death were obtained from individual parish offices for deaths occurring in 1989 and 1990. All death certificates were reviewed; in each case the officially stated underlying cause of death was coded by the ICD–8 classification.

INCIDENCE OF CANCER

Incidence of cancer for the years 1958–87 was available through linkage with the National Cancer Registry, which lists cancer site and histological diagnosis along with other information. Four-digit codes according to the ICD–7 classification were used in this analysis. In cases where the listed diagnosis was unclear, individual case records, including pathological reports, were obtained from the National Cancer Registry. Persons dying before 1958 could not be included in this analysis, which was thus performed for 5242 subjects.

DATA ANALYSIS

The observed numbers of deaths or incident cases of cancer were compared with the expected numbers calculated by the person-years method. National rates for mortality and cancer incidence, stratified by five year calendar and age class, were obtained from Statistics Sweden and the National Cancer Registry. Analysis was performed with the Occupational Mortality Analysis Program (OCMAP/PC).[15] Tests for trends were performed according to the χ^2 method.[16]

EXPOSURE DATA

Years of active work as a sweep were used as a measure of cumulative exposure, as more detailed exposure data were unavailable. The mean employment time in this cohort was 12·0 years (SD 12·2) with a median employment of 9·0 years. Many of the long term employed sweeps became master sweeps, which in most cases involves primarily administrative work. Years spent as a master sweep were considered unexposed in the cumulative exposure analysis.

Employment data were available only until 1980 due to a change in the union's record keeping. Years worked after 1980 are thus not counted in the cumulative exposure analyses, causing underestimation of duration of exposure for sweeps active in the past decade.

TREATMENT OF ALCOHOL AND TOBACCO USE

Due to different smoking habits among the chimney

Table 1 Smoking habits, 1972 chimney sweeps (national male population in parentheses)

Age class	% Current smokers	% Former smokers	% Never smokers
30–39	63 (49)	15 (16)	23 (35)
40–49	70 (51)	15 (17)	15 (32)
50–59	57 (43)	21 (19)	21 (38)
60–69	56 (41)	26 (22)	19 (37)

sweeps, smoking adjusted risk ratios (RRs) were calculated for lung cancer mortality and incidence of ischaemic heart disease and bladder cancer to allow comparison with population norms. A method similar to that of Axelson[17] was used. Percentages of current smokers, former smokers, and those who had never smoked were available from a 1972 health survey of 1040 active sweeps.[18] Comparable national smoking data were available for the years 1963 and 1977, and were used to estimate national smoking habits in 1972.[19 20] Table 1 presents these data. Risk ratios for smoking were estimated from published data. For mortality from lung cancer, a RR of 10 was assumed for current smokers, and 2 for former smokers when compared with never smokers.[21] For ischaemic heart disease mortality, the assumed RRs were 1·7 and 1·15 respectively.[22] For incidence of bladder cancer smokers were assumed to have an RR of 3·0 and former smokers 2·0.[23] Using national mortality and cancer incidence data, calculated by 10 year age class, expected numbers of cases were adjusted proportional to the percentage of smokers and former smokers in each age class. The resulting age specific RRs were then weighted by the expected numbers of cases in each 10 year age class to obtain smoking adjusted RRs for the sweep cohort. These RRs were later used to adjust expected numbers of cases to account for excess risks attributable to smoking alone.

An analysis of severe alcohol abuse was performed by matching the chimney sweep cohort to the National Psychiatric Discharge Registry for the years 1973–83. This registry records all stays in hospital for psychiatric reasons in Sweden, including those related to alcohol use. For the cohort of sweeps and for the entire nation, all stays in hospital given the ICD discharge diagnosis 291 (alcoholic psychosis) or 303 (alcoholism) were counted by five year age class for each calendar year 1973–83, the only years for which these data were available. An expected number of stays due to alcohol abuse was then calculated in a manner analogous to the person-years method, with background rates derived from the national population. Observed stays in hospital among the sweeps were divided by the age adjusted expected values derived from the general population to obtain an observed:expected ratio for the period 1973–83. This ratio represents the RR of stays in hospital due to alcohol abuse among the sweeps compared with the general population.

Results

NON-CANCER MORTALITY

Tables 2 and 3 present mortality data for the entire study period 1951–90 and for the extended follow up

Table 2 All cause mortality, age <80, 1951–90 (140 402 person-years)

Cause of death	ICD-8	Obs	Exp	SMR (95% CI)
All causes	1–999	919	679·6	135 (127–144)
All malignant tumours	140–209	216	147·9	146 (127–167)
Oesophageal cancer	150	12	3·1	386 (200–675)
Stomach cancer	151	20	16·6	121 (74–187)
Bowel cancer	152–153	8	10·5	76 (33–150)
Rectal cancer	154	6	6·3	95 (35–207)
Liver cancer	155	11	3·3	331 (165–593)
Pancreatic cancer	157	10	10·0	100 (48–185)
Lung cancer	162	53	25·8	206 (154–269)
Prostatic cancer	185	22	13·0	169 (106–256)
Bladder cancer	188	4	3·9	103 (28–264)
Kidney cancer	189	6	7·2	84 (31–182)
Malignant cerebral tumours	191	5	5·5	91 (30–213)
All haematopoietic cancer	200–209	22	17·3	127 (80–192)
Malignant lymphoma	200–202	6	6·8	88 (32–191)
Myeloma	203	6	2·5	239 (88–521)
Leukaemia	204–207	10	7·2	139 (67–256)
Nervous diseases	320–389	6	10·2	59 (22–128)
Circulatory diseases	390–458	354	276·0	128 (115–142)
Ischaemic heart disease	410–414	249	194·9	128 (112–145)
Cerebrovascular diseases	430–438	33	44·8	74 (51–103)
Respiratory diseases	460–519	44	27·8	159 (115–213)
Asthma, bronchitis, emphysema	490–493	16	11·0	145 (83–245)
Digestive diseases	520–577	39	30·1	130 (92–177)
Liver cirrhosis	571	22	13·0	169 (106–256)
Genitourinary diseases	580–629	10	10·4	97 (46–177)
Nephritis, nephrosis	580–584	6	4·9	122 (45–265)
External causes	E800–999	189	123·7	153 (132–176)
Suicide	E950–959	60	43·8	137 (104–176)

Table 3 All cause mortality, age <80, 1983–90 (33 940 person-years)

Cause of death	*ICD–8*	*Obs*	*Exp*	*SMR (95% CI)*
All causes	1–999	251	208·7	120 (106–136)
All malignant tumours	140–209	61	48·5	126 (96–162)
Oesophageal cancer	150	2	1·2	169 (20–610)
Stomach cancer	151	3	4·1	73 (15–213)
Lung cancer	162	11	9·8	112 (56–201)
Prostatic cancer	185	9	4·6	197 (90–375)
Bladder cancer	188	1	1·3	77 (2–431)
All haematopoietic cancer	200–209	6	5·1	117 (43–255)
Malignant lymphoma	200–202	3	2·0	150 (31–439)
Myeloma	203	1	0·8	132 (3–738)
Leukaemia	204–207	2	2·0	100 (12–361)
Circulatory diseases	390–458	97	97·6	99 (80–121)
Ischaemic heart disease	410–414	69	70·8	98 (76–123)
Cerebrovascular diseases	430–438	7	12·4	56 (23–116)
Respiratory diseases	460–519	11	9·9	111 (56–199)
Liver cirrhosis	571	8	4·3	186 (81–367)
External causes	E800–999	44	29·3	151 (109–202)

period 1983–90. The cohort shows statistically significant excesses of all cause mortality, circulatory diseases, ischaemic heart disease, respiratory diseases, and external causes (primarily accidental deaths and suicide). The non-significant trend toward decreased mortality from cerebrovascular disease has continued. New findings include significant excess mortality from liver cirrhosis and suicide. Of the 239 excess deaths in the total study period, 68 were due to cancer, 78 to circulatory disease (including 54 excess deaths due to ischaemic heart disease), and 65 to external causes (including 16 excess suicides).

Comparison of mortality between the study periods 1951–82 and 1983–90 shows a decline in the excess risks for circulatory disease, ischaemic heart disease, and respiratory disease, although the mortality trends among the two periods are otherwise similar.

Mortality by cumulative exposure (see table 6) showed a trend toward increased mortality from respiratory disease ($\chi^2 = 1{\cdot}92$, 1 df) with increased employment time, which was of borderline significance. No such trend was evident for circulatory disease or ischaemic heart disease.

Smoking adjusted RRs were calculated as described. If the sweeps' smoking habits in 1972 were representative, they could be expected to have a risk of death from ischaemic heart disease approximately 1·08 times that of the general public due to smoking alone. Recalculation of the expected values based on this risk gives 211·2 expected deaths from ischaemic heart disease (249 observed; smoking adjusted SMR 117, 95% CI 102–132).

Table 4 Alcohol consumption of chimney sweeps drinking the equivalent of 6 litres/yr of EtOH in 1972 and percentage of the national population consuming more than 5 litres/yr of EtOH in 1968

Age	*% Of sweeps drinking >6 l/y (1972)*	*% Of nation drinking >5 l/y (1968)*
10–19	17	13
20–29	19	26·5
30–39	23	23
40–49	24	19·5
50–59	34	17
60–69	56	16·5

Table 4 presents data from the 1972 health survey of the sweeps as well as data derived from a national questionnaire from 1968–9.[24] The number of heavy drinkers is higher among the sweeps than the general population in almost every age category. Data from the Psychiatric Discharge Register, which lists all discharge diagnoses from psychiatric stays in hospital in Sweden, also indicate an excess of alcohol use among the sweeps. There were 2·85 times more alcohol related stays in hospital among the sweeps than would be expected; stays in hospital among sweeps employed for 10 years or more were 2·09 times expected. In these data, a single worker can contribute many stays in hospital within a single year. National comparison figures were not available to allow comparison of the percentage of sweeps ever included in this registry. As would be expected, the cohort shows an increased mortality from cirrhosis of the liver and from external causes.

CANCER INCIDENCE AND MORTALITY

Table 5 shows data for cancer incidence for the periods 1958–87 and 1982–87. New findings include significant excesses in incidence and mortality from prostate cancer and in the incidence of haematopoietic and lymphatic malignancies. Leukaemia and non-Hodgkin's lymphoma contributed most of the cases, although observed cases were also in excess of those expected for multiple myeloma/plasmacytoma, Hodgkin's disease, and chronic myelogenous leukaemia. The previously noted significant excess risks for cancers of the oesophagus, lung, and bladder, as well as total malignancies, persisted.

For the extended follow up period 1982–87, statistically significant excess risks were seen for total

Table 5 Incidence of cancer 1958–87 and 1982–87

Tumour type	ICD–7	Cancer incidence 1958–87 (114 980 person-years) Obs	Exp	SMR (95% CI)	Cancer incidence 1982–87 (27 160 person-years) Obs	Exp	SMR (95% CI)
All malignant tumours	140–209	335	235·0	143 (128– 159)	113	78·6	144 (118–173)
Oropharnyx	140–148	9	7·1	127 (58– 242)	3	2·4	126 (25–368)
Oesophagus	150	11	2·8	387 (193– 693)	2	1·0	198 (22–715)
Stomach	151	23	16·6	138 (88– 208)	5	4·2	105 (34–246)
Colon	153	18	16·1	112 (66– 199)	4	5·3	76 (20–195)
Rectum and anus	154	12	11·0	109 (57– 191)	7	3·7	191 (77–394)
Biliary and liver primary	155	7	5·5	127 (51– 261)	2	2·0	99 (11–357)
Pancreas	157	10	8·1	123 (59– 226)	3	2·7	113 (23–330)
Larynx	161	4	2·8	141 (38– 362)	1	0·9	108 (1–598)
Bronchus and lung primary	162·1	50	23·9	209 (155– 276)	15	8·5	178 (99–293)
Oat cell and undifferentiated*		28	10·2	274 (182– 396)	8	3·3	240 (103–472)
Squamous cell		17	9·4	181 (106– 290)	5	3·3	153 (49–357)
Adenocarcinoma		3	3·3	92 (18– 268)	1	1·4	74 (1–409)
Pleura	162·2	3	0·6	469 (94–1370)	1	0·3	333 (4–185)
Prostate	177	45	35·4	127 (93– 170)	22	12·9	171 (107–258)
Kidney	180	7	10·8	65 (26– 133)	2	3·7	54 (6–196)
Bladder	181	37	14·6	253 (178– 349)	13	5·3	247 (131–422)
Malignant melanoma	190	10	8·9	113 (54– 207)	4	3·6	113 (30–288)
Skin (excluding melanoma)	191	4	6·5	61 (17– 157)	3	2·4	124 (25–364)
Nervous system	193	7	11·3	62 (25– 127)	2	3·4	58 (7–210)
Connective tissue, muscle	197	2	2·4	84 (9– 305)	0	0·7	—
Unspecified and sites unknown	199	11	6·5	170 (85– 304)	4	2·2	183 (49–470)
All haematopoietic cancer	200–209	36	23·8	151 (106– 209)	11	7·4	149 (75–267)
Leukaemia	204	11	7·9	140 (70– 250)	3	2·4	128 (26–375)
Chronic lymphatic leukaemia		2	2·6	76 (9– 275)	2	0·9	227 (26–821)
Chronic myelogenous leukaemia		2	1·6	127 (14– 457)	0	0·4	—
Malignant non-Hodgkin's lymphoma	200	11	7·2	154 (77– 275)	7	2·6	268 (107–553)
Hodgkin's disease	201	5	3·9	127 (41– 297)	1	0·8	125 (2–695)
Multiple myeloma, plasmacytoma	203	6	3·4	179 (65– 389)	0	1·2	—

*Includes one case of alveolar cell carcinoma and one case of unknown histological type.

malignancies, prostate cancer, bladder cancer, and non-Hodgkin's lymphoma. A large excess risk for lung carcinoma continued in the period 1982–87; this risk was especially pronounced for small cell and undifferentiated cancer.

One new case of pleural mesothelioma was found during the extended follow up; three histologically confirmed pleural mesotheliomas have thus been found in this cohort to date.

The sweeps continue to show an appreciable increase in incidence of bladder cancer, without any increase in mortality. Of the 37 incident cases, 33 had the pathological diagnosis of transitional cell carcinoma, two were undifferentiated carcinoma, and two had other histological diagnoses.

Cancer mortality by duration of employment (table 6) showed statistically significant trends for increased mortality from lung cancer, oesophageal cancer, and total malignancies with continued exposure.

Incidence of cancer by cumulative exposure showed increasing incidence of total lung cancer, small cell and undifferentiated cancer of the lung, and total malignancies with continued exposure. A negative correlation was seen for incidence of bladder cancer and duration of employment.

Cancer mortality by latency since first employment (table 7) showed increased mortality from total malignancies, oesophageal cancer, lung cancer, prostate cancer, circulatory diseases, and ischaemic heart disease only after long latencies.

Investigation of the 11 deaths coded as liver cancer (ICD–8 155) by comparison with the Cancer Registry data showed that only four of these deaths were due to histologically confirmed hepatocellular carcinoma. One was a primary tumour of the biliary tree, whereas the others represented secondary involvement of the liver from another primary cancer. The degree of such misclassification of death certificates on a national scale is not known. No excess of hepatic cancer was found in the analysis for incidence of cancer.

Smoking adjustment for mortality from lung cancer estimated an excess risk of 1·28 times that of the general population based on tobacco use alone. Recalculation of expected values based on this risk estimate gave 33·0 expected deaths (53 observed; smoking adjusted SMR 161, 95% CI 120–210). Adjustment for incidence of bladder cancer gave an estimated relative risk of 1·16 due to smoking alone. Recalculation of expected values based on this risk estimate gave 16·2 expected cases of bladder cancer (37 observed; smoking adjusted SIR 228, 95% CI 160–314).

Fuels used in residential furnaces have changed in Sweden. In the years around 1950, coal was largely replaced by oil; woodburning stoves have remained. To evaluate possible health effects of this switch from

Table 6 Mortality and incidence of cancer by cumulative exposure

	Years of work as an active sweep							
	1–9		*10–19*		*20–29*		*>30*	
	Obs/Exp	*(SMR)*	*Obs/Exp*	*(SMR)*	*Obs/Exp*	*(SMR)*	*Obs/Exp*	*(SMR)*
Mortality:								
All causes	263/179·9	(146)	251/198·2	(127)	182/133·0	(137)	223/168·6	(132)
All malignancies	32/32·6	(98)	64/44·2	(145)	50/31·3	(160)	70/39·7	(176)*
Oesophageal cancer	1/0·59	(169)	3/0·95	(316)	2/0·68	(294)	6/0·89	(674)*
Stomach cancer	3/2·5	(122)	3/4·47	(67)	4/3·7	(108)	10/5·9	(169)
Lung cancer	4/5·2	(77)	17/8·1	(211)	13/5·8	(225)	19/6·7	(282)*
Prostate cancer	1/1·5	(66)	8/3·5	(232)	4/3·1	(129)	9/5·0	(181)
Circulatory disease	71/49·2	(144)	109/81·4	(134)	75/62·3	(120)	99/83·4	(119)
Ischaemic heart disease	41/33·9	(121)	87/58·4	(149)	45/44·6	(101)	76/58·1	(131)
Respiratory disease	8/5·7	(142)	8/7·9	(101)	11/5·9	(186)	17/8·3	(205)
Liver cirrhosis	7/4·1	(170)	9/4·5	(201)	2/2·5	(81)	4/2·0	(205)
Cancer incidence:								
All tumours	72/54·1	(133)	88/69·3	(127)	81/50·3	(161)	94/61·4	(153)*
Oesophagus	2/0·49	(408)	3/0·84	(357)	3/0·66	(455)	3/0·85	(353)
Stomach	4/2·5	(158)	5/4·5	(112)	5/3·9	(129)	9/5·7	(158)
Lung	4/4·5	(88)	16/7·4	(217)	15/5·6	(270)	15/6·4	(234)*
Oat cell and undifferentiated	3/2·0	(150)	9/3·1	(287)	8/2·4	(340)	8/2·7	(292)*
Squamous cell	0/1·63	(—)	6/2·9	(211)	7/2·3	(311)	5/2·7	(189)
Adenocarcinoma	1/0·73	(137)	1/1·1	(94)	0/0·71	(—)	1/0·77	(130)
Prostate	6/3·9	(154)	11/9·1	(121)	11/8·9	(124)	17/13·6	(125)
Bladder	10/2·99	(334)	13/4·5	(291)	7/3·3	(212)	7/3·9	(182)*
All haematopoietic	14/7·62	(184)	12/7·1	(169)	3/4·4	(68)	7/4·7	(150)
Leukaemia	1/2·44	(41)	7/2·3	(306)	1/1·5	(68)	2/1·7	(119)

*Test for trend significant at $p < 0·05$.

exposure to soot from coal/wood to oil/wood, we examined mortality in sweeps exposed only after 1951, who would have had minimal exposure to coal soot (table 8). With the exception of ischaemic heart disease, this group exhibits the same general pattern of excess mortality as the whole cohort, although the number of cases is small.

Discussion

This long term study of chimney sweeps found excess mortality from ischaemic heart disease, respiratory disease, lung cancer, oesophageal cancer, prostate cancer, liver cirrhosis, and violent deaths. Incidence was increased for bladder cancer, haematopoietic and lymphatic cancer, lung cancer, oesophageal cancer, and mesothelioma. Dose-response relations were noted for mortality from lung cancer, oesophageal cancer, and total malignancies as well as incidence of total malignancies, total lung cancers, and small cell or undifferentiated cancer of the lung. Although there is a significantly increased mortality from ischaemic heart disease seen for the entire study period 1951–90, this risk has approached that of the general population during the extended follow up.

POSSIBLE CONFOUNDING FACTORS

When compared with the general population, a greater percentage of the sweeps reported use of tobacco. As already noted, significant excess mortality from lung cancer and ischaemic heart disease remain after adjustments for smoking habits. These

Table 7 Observed and expected mortality by latency (years since first exposure)

	Years since first exposure							
	1–9		*10–19*		*20–29*		*>30*	
Cause of death	*Obs/Exp*	*(SMR)*	*Obs/Exp*	*(SMR)*	*Obs/Exp*	*(SMR)*	*Obs/Exp*	*(SMR)*
All causes	52/44·9	(116)	78/58·7	(133)*	126/84·7	(149)*	663/491·6	(135)*
All malignancies	3/4·6	(65)	6/8·1	(74)	26/16·1	(162)*	181/119·2	(152)*
Oesophageal cancer	0/0·01	(—)	0/0·06	(—)	1/0·24	(417)	11/2·8	(393)*
Lung cancer	0/0·13	(—)	0/0·61	(—)	4/2·4	(167)	49/22·6	(217)*
Prostate cancer	0/0·01	(—)	0/0·07	(—)	1/0·28	(357)	21/12·7	(166)*
Haematolymphatic cancer	1/1·7	(60)	3/2·0	(152)	6/2·71	(221)	12/11·0	(110)
Circulatory disease	2/2·5	(81)	6/7·2	(83)	30/22·0	(136)	316/244·7	(129)*
Ischaemic heart disease	0/0·63	(—)	4/3·5	(115)	22/14·4	(152)	223/176·4	(126)*
Respiratory disease	2/1·0	(206)	2/1·35	(148)	3/2·5	(121)	37/23·0	(161)*
Liver cirrhosis	1/0·25	(400)	1/1·16	(86)	6/3·3	(185)	14/8·4	(167)

$p < 0·05$.

Table 8 Mortality of sweeps exposed only after 1951

Mortality	*Obs*	*Exp*	*SMR (95% CI)*
All causes	195	134·1	146 (126– 167)
All malignancies	23	20·3	113 (72– 170)
Oesophageal cancer	0	0·3	— (—)
Lung cancer	3	2·6	113 (23– 330)
Prostate cancer	1	0·3	309 (8–1721)
Haematolymphatic cancer	7	4·5	155 (62– 318)
Circulatory disease	25	24·2	103 (67–152)
Ischaemic heart disease	19	15·0	127 (76–198)
Respiratory disease	5	3·5	144 (47–335)
Liver cirrhosis	5	3·5	143 (46–334)
External causes	105	63·8	165 (135– 199)

calculations assume that the baseline mortalities among the sweeps are the same as those of the general male population. In this strenous job, however, a lower baseline mortality would be predicted by the healthy worker effect.[25] Thus the standardised mortality ratio (SMR) for ischemic heart disease may represent an underestimation of the true excess risk among chimney sweeps.

In this study, confounding from the sweeps' greater intake of alcohol is primarily of interest in evaluating the excess of oesophageal cancer. Risks of oesophageal cancer and cirrhosis among cohorts of chronic alcohol abusers provide a useful comparison. Members of a cohort of Swedish men treated for alcohol abuse were five times more likely to die of oesophageal cancer and seven times more likely to die from cirrhosis than the general population.[26] Alcoholic persons treated in Canada had an SMR of 520 for oesophageal cancer and 1149 for cirrhosis,[27] and a group of treated alcoholic patients in Oslo had SMRs of 426 for oesophageal cancer and 359 for cirrhosis.[28]

Thus the excess of mortality from oesophageal cancer among the sweeps approaches the risk seen in cohorts composed entirely of chronic alcohol abusers, whereas deaths from cirrhosis were much lower among the sweeps than among the alcoholic cohorts. Although precise data on smoking habits were not reported for these cohorts of chronic alcohol abusers, they were noted to have tobacco consumption in excess of the general population. Although SMRs cannot be directly compared between cohorts, the large discrepancy between RRs for oesophageal cancer and cirrhosis of the liver lead us to conclude that the high risk of oesophageal cancer seen in the chimney sweep cohort cannot be attributed entirely to alcohol and tobacco.

The three cases of pleural malignant mesothelioma confirm the industrial hygiene assessment that chimney sweeps are exposed to asbestos. We attempted to estimate the extent to which the excess of lung cancer in this cohort may be due to exposure to asbestos and to what extent it is due to other carcinogens. Each fibre/ml-year of chrysotile asbestos exposure results in an increase in mortality from lung cancer of about 1%.[29] Industrial hygiene surveys of chimney sweeping[7] have shown that sweeps have short intermittent exposures to low levels of asbestos; their overall time weighted exposure is certainly less than 0·1 fibres/ml. Using these figures, sweeps employed for 30 years would be expected to have less than a 3% increase in mortality from lung cancer due to exposure to asbestos. This estimate does not account for synergy with smoking and other exposures, and exposure to asbestos may have been heavier in previous years. It is unlikely, however, that exposure to asbestos could account for much of the excess of lung cancer seen in this cohort.

Although exposure to asbestos and tobacco may explain part of the excess risk for lung cancer seen in the sweeps, a substantial unexplained excess risk remains. This is most likely due to the sweeps' other chemical exposures.

IMPLICATIONS FOR OCCUPATIONAL CARCINOGENESIS

The major new results of this study are the increase in the incidence and mortality from prostate cancer, and the significant increase in the incidence of lymphatic and haematopoietic malignancies. Previously noted excesses of lung, oesophageal, and bladder cancer persisted in the extended follow up; the excess of small cell and undifferentiated cancer of the lung was striking. With extended follow up, stronger dose-response relations were seen for mortality from lung and oesophageal cancer.

Genitourinary cancer

The aetiology of prostate cancer remains obscure and no firm links have been established between occupational exposures and this malignancy. Chimney sweeps are exposed to low concentrations of cadmium, which has been related to prostate cancer in some studies,[30 31] although other studies have provided variable results.[32 33] Excess mortality from prostate cancer has also been noted in several cohorts of rubber workers,[34] although no common exposure has been identified. Given the sweeps' exposure to combustion products, it is of interest that excess mortality from prostate cancer has also been associated with exposure to automobile exhaust in a case-control study[35] and in a large registry based study.[36] Mortality from prostate cancer has also been linked to cigarette smoking in a large cohort study.[37] No conclusions can be drawn at this time regarding a possible aetiological link between combusion products and prostate cancer, but this finding merits further study.

The excess incidence of bladder cancer has continued to rise in this cohort during the extended follow up, although there has been no significant increase in mortality. This discrepancy may be due to chance, given the low mortality of bladder cancer. Alternatively, it is possible that sweeps may receive more

intensive screening for cancer than the general population, thus increasing the detection of common tumours such as those of the prostate and bladder. This seems unlikely, however. A third possibility is that the bladder cancer seen in this cohort may have a different natural history than that of the general population. The reason for the negative correlation between incidence of bladder cancer and duration of employment is unknown.

Lymphatic and haematopoietic cancer

A significant excess was seen in the incidence of lymphatic and haematopoietic cancer, as well as an increased mortality that was of borderline statistical significance. The role of PAHs in inducing lymphoma and leukaemia is unclear, although exposure to PAHs may induce leukaemia in animal models[38] and cigarette smoking has been weakly linked to leukaemia.[39] The sweeps' exposure to organic solvents is recent and probably does not account for their excess risk of leukaemia.

Gastrointestinal cancer

As discussed, the large excess risk of oesophageal cancer seen in this cohort is more than would be expected from alcohol and tobacco alone, even taking into account the well known multiplicative interaction of these exposures.[40] It seems highly likely that exposure to PAHs plays a causal part in the sweeps' excess of oesophageal cancer. Direct oesophageal mucosal contact with PAHs must occur often among the sweeps, due to the swallowing of soot after mucociliary clearance and expectoration of inhaled particles.

Alcohol may have a role in potentiating this occupational exposure. Chronic intake of alcohol may directly enhance metabolic activation of carcinogens by the epithelial cells of target tissue; synergy between alcohol and tobacco may also be due to increased transport of carcinogen across the mucosa into the basal layer where the target tissue is assumed to lie. In animal studies, solutions of benzo-[a]-pyrene applied to the oesophageal mucosa were better absorbed when alcohol rather than oil was used as the solute; increased absorption was obtained with stronger solutions of alcohol.[41]

Other cohorts of Swedish workers exposed to combustion products have also shown increased rates of oesophageal cancer, although the numbers of cases have been small.[42–44] Statistically significant excesses of oesophageal cancer have been seen in other cohorts exposed to petroleum or combustion byproducts, such as mastic asphalt workers[45] and firefighters.[46] High rates of oesophageal cancer in some areas of the world have been attributed to the consumption of pyrolysis products such as pipe scrapings.[47]

Lung cancer

Although excesses in incidence of squamous cell and adenocarcinoma were seen, the largest excess was for small cell and undifferentiated lung cancer (which are coded together in the Swedish Cancer Registry). This may in part explain why a larger excess risk was seen in incidence of cancer than mortality, as small cell cancer generally has a lower mortality than other histological types. A strong dose-response relation was seen for total lung cancer as well as for small cell and undifferentiated cancer.

Skin cancer

As in previous analyses, no excess of skin cancer was seen in this cohort. This may be attributable to the fact that Swedish sweeps have long had the right to bathe, on paid time, at the end of their work day.

IMPLICATIONS FOR OTHER DISEASES

The increased mortality from circulatory diseases and ischaemic heart disease is particularly interesting. Several other occupational chemical exposures have been associated with increased mortality or morbidity from heart disease,[48] although the causal mechanisms through which these exposures are linked to heart disease are obscure. In the case of exposure to combustion byproducts, animal data on PAHs and atherogenesis may offer some clues. There is evidence from several species that PAHs, including benzo-[a]-pyrene and dimethyl-benz-anthracene, can act as promoters of atherosclerosis.[49 50] In these studies, PAHs seem to act as promoters of vascular smooth muscle proliferation within plaques. One model of atherogenesis is based on the hypothesis that atherosclerotic plaques arise as monoclonal proliferations of smooth muscle cells, subject to processes of initiation and promotion analogous to those found in malignant tumours.[51 52] This suggests one mechanism by which exposure to combustion byproducts could cause increased mortality from ischaemic heart disease.

Exposure to carbon monoxide may also be important in causing the increased mortality from ischaemic heart disease seen in this cohort. Other studies have found increased mortality in cohorts with occupational exposure to carbon monoxide and other exhaust products.[53] It is not clear whether carbon monoxide has atherogenic effects or whether exposure acts through other mechanisms such as hypoxia, platelet aggregation, or the induction of arrhythmias.[54 55] With continued follow up, the excess mortality from ischaemic heart disease among the sweeps has declined. It is interesting to note that their exposure to carbon monoxide has also declined since fuel oil started to replace coal in the 1950s. Industrial hygiene measurements[6] indicated that carbon monoxide concentrations during sweeping of coal fired furnaces ranged from 5–400 ppm, whereas

concentrations near fuel oil fired furnaces were less than 1 ppm.

This study found an increased mortality from respiratory diseases, with a non-significant trend toward increased risk with longer exposures. The chimney sweeps' heavy occupational exposure to dust and irritant gases probably has a causal role in this excess of mortality. When Danish chimney sweeps were compared to postmen in a matched pair design that controlled for smoking habits, a significant excess of sweeps reported respiratory symptoms; 9% of the sweeps reported coughing up an expectorate of black phlegm.[56]

Conclusion

Despite changes in the work environment, chimney sweeping remains a hazardous occupation. The alcohol and tobacco consumption habits of this cohort do not explain their excess risks of ischaemic heart disease, lung cancer, or oesophageal cancer. These excess risks and the striking increase in incidence of bladder cancer are most likely due to occupational exposure to PAHs and other substances contained in chimney soot. The excess of prostate cancer and haematolymphatic cancers seen in this cohort deserves study in other groups.

Neither these diseases nor exposure to pyrolysis compounds are unique to chimney sweeps, but are shared by other occupations and the general public although at lower levels. The health state of chimney sweeps is thus relevant to other populations exposed to combustion byproducts.

We thank Annika Gustavsson for computer assistance and for her work in assembling the cohort and maintaining follow up, and Gunilla Ringbäck for preparing the input rate files. This study was supported by the Swedish Work Environment Fund contract No 90-1596. BE was partially supported by a Fogarty Institute Post Doctoral Research Fellowship and by a grant from the American-Scandinavian Foundation.

Requests for reprints to: Dr Bradley Evanoff, Clinical Scholars Program HQ-18, University of Washington, Seattle, WA 98195, USA.

1 Pott P. *Chirurgical observations relative to the cataract, the polypus of the nose, the cancer of the scrotum, the different kinds of ruptures, and the mortification of the toes and feet.* London: Hawkes, Clarke, and Collins, 1775:7–13.
2 International Agency for Research on Cancer. *IARC Monographs on the evaluation of the carcinogenic risks of chemicals to man.* Vol 32. *Polynuclear aromatic compounds.* Lyon: IARC, 1983.
3 Knecht U, Bolm-Audorff U, Woitowitz H-J. Atmospheric concentrations of polycyclic aromatic hydrocarbons during chimney sweeping. *Br J Ind Med* 1989;**46**:479–82.
4 Bagchi NJ, Zimmerman RE. An industrial hygiene evaluation of chimney sweeping. *Am Ind Hyg Assn J* 1980;**41**:297–9.
5 Chang G. *Chemical health hazards in chimney sweeping.* Stockholm: Research Department, National Board of Occupational Safety and Health, Report No 2603/79, 1980. (In Swedish.)
6 Fehrmann R. *Characterization of chimney sweep's occupational environment.* Copenhagen: Danish Work Environment Fund, 1982. (In Danish.)
7 Andersson K. *Chimney sweeps' exposure to dust, PAH, metals, and asbestos.* Stockholm: Swedish Environmental Research Institute, report No B 850, 1987. (In Swedish.)
8 Kipling MD, Waldron HA. Percivall Pott and cancer scroti. *Br J Ind Med* 1975;**32**:244–50.
9 Hogstedt C, Andersson K, Frenning B, Gustavsson A. A cohort study on mortality among long-time employed Swedish chimney sweeps. *Scand J Work Environ Health* 1982;**8**:72–8.
10 Gustavsson P, Gustavsson A, Hogstedt C. Excess mortality among Swedish chimney sweeps. *Br J Ind Med* 1987;**44**: 738–43.
11 Gustavsson P, Gustavsson A, Hogstedt C. Excess of cancer in Swedish chimney sweeps. *Br J Ind Med* 1988;**45**:777–81.
12 Hansen ES. Mortality from cancer and ischemic heart disease in Danish chimney sweeps: A five-year follow up. *Am J Epidemiol* 1983;**117**:160–4.
13 Hansen ES. *Cancer and ischemic heart disease in a chimney sweep cohort.* Odense: Institute for Social Medicine, 1983. (Doctoral dissertation, in Danish.)
14 Cadez E. Ergebnisse langjähriger Untersuchungen über den Einfluss von Schadstoffen auf die Gusundheit von Schornsteinfegern. *Staub Reinhalt Luft* 1983;**43**:116–7.
15 Marsh GM, Ehland J, Paik M, Preininger M, Caplan R. OCMAP/PC: A user-oriented occupational cohort mortality analysis program for the IBM PC. *American Statistician* 1986;**40**:308–9.
16 Breslow N, Lubin JH, Marek P, Langholz B. Multiplicative models and cohort analysis. *Journal of the American Statistical Association* 1983;**78**:1–12.
17 Axelson O. Aspects on confounding in occupational health epidemiology. *Scand J Work Environ Health* 1978;**4**:98–102.
18 Swensson Å, Swensson A. *Chimney sweeps trade union health survey, 1972 Stockholm:* Research Department, National Board of Occupational Safety and Health, Report AMMM 101/04, 1974. (In Swedish.)
19 Statistics Sweden. *Smoking habits in Sweden.* Stockholm: Statistics Sweden, 1965. (In Swedish.)
20 Swedish National Board of Health and Welfare. *Tobacco habits in Sweden: an overview and analysis.* Stockholm: Swedish National Board of Health and Welfare, 1986. (In Swedish.)
21 Pike MC, Henderson B. *Epidemiology of polycyclic hydrocarbons: quantifying the cancer risk from cigarette smoking and air pollution. Polycyclic hydrocarbons and cancer.* Vol 13. London: Academic Press, 1981:317–34.
22 US Surgeon General. *Smoking and health: a report of the Surgeon General.* United States Department of Health, Education, and Welfare DHEW Publ No (PHS) 79-50066, 1979.
23 International Agency for Research on Cancer. *IARC Monographs on the evaluation of the carcinogenic risk of chemicals to man.* Vol 38. *Tobacco smoking.* Lyon: IARC, 1985.
24 Swedish Government Official Report. *Alcohol habits in Sweden: 1971–77.* Stockholm: Department of Financial Affairs, 1977. (In Swedish.)
25 McMichael AJ. Standardized mortality ratios and the "healthy worker effect:" Scratching beneath the surface. *J Occup Med* 1976;**18**:165–8.
26 Lindberg S, Ågren G. Mortality among male and female hospitalized alcoholics in Stockholm 1962–1983. *Br J Addict* 1988;**83**:1193–200.
27 Schmidt W, de Lint J. Causes of death of alcoholics. *Quarterly Journal of Studies of Alcoholism* 1972;**33**:171–85.
28 Sundby P. *Alcoholism and Mortality.* Oslo: The National Institute for Alcohol Research, Publ No 6, 1967.
29 Doll R, Peto J. *Effects on health of exposure to asbestos.* London: Health and Safety Commission, Her Majesty's Stationery Office, 1985.
30 Kipling MD, Waterhouse JA. Cadmium and prostatic carcinoma. *Lancet* 1967;730–1.
31 Elghany NA, Schumacher MC, Slattery ML, West DW, Lee JS. Occupation, cadmium exposure, and prostrate cancer. *Epidemiology* 1990;**1**:107–15.
32 Elinder CG, Kjellström T, Hogstedt C, Andersson K, Spång G. Cancer mortality of cadmium workers. *Br J Ind Med* 1985;**42**:651–5.
33 Kazantzis G, Lam TH, Sullivan KR. Mortality of cadmium-exposed workers: A five-year update. *Scand J Work Environ Health* 1988;**14**:220–3.
34 Monson R, Fine LJ. Cancer mortality and morbidity among

rubber workers. *J Natl Cancer Inst* 1978;**61**:1047–53.
35 Rotkin ID. Studies in the epidemiology of prostatic cancer: Expanded sampling. *Cancer Treat Res* 1977;**61**:173–80.
36 Ernster VL, Selvin S, Brown S, Sacks S, Winkelstein W, Austin DF. Occupation and prostate cancer: A review and retrospective analysis based on death certificate in two California counties. *J Occup Med* 1979;**21**:175–83.
37 Hsing AW, McLaughlin K, Hrubec Z, Blot W, Fraumeni J. Tobacco use and prostate cancer: 26-year follow-up of US veterans. *Am J Epidemiol* 1991;**133**:437–41.
38 Sugiyama T, Venaka H, Veda N, Fukuhara S, Maeda S. Reproducible chromosomal changes of polycyclic hydrocarbon-induced rat leukemia: incidence and chromosome banding pattern. *J Natl Cancer Inst* 1978;**69**:153–60.
39 Austin H, Cole P. Cigarette smoking and leukemia. *Journal of Chronic Diseases* 1986;**39**:417–21.
40 Tuyns AJ, Péquignot G, Jensen OM. Le cancer de l'oesophage en Ille-et-Vilaine en fonction des niveaux de consommation d'alcool et de tabac: Des risques qui se multiplient. *Bull Cancer* 1977;**64**:45–60.
41 Kuratsune M, Kohchi S, Horie A. Carcinogenesis in the esophagus. I. Penetration of benzo[a]pyrene and other hydrocarbons into the esophageal mucosa. *Gann* 1965;**56**: 177–87.
42 Gustavsson P. Mortality among workers at a municipal waste incinerator. *Am J Ind Med* 1989;**15**:245–53.
43 Gustavsson P, Reuterwall C. Mortality and incidence of cancer among Swedish gas workers. *Br J Ind Med* 1990;**47**:169–74.
44 Gustavsson P, Plato N, Lidström EB, Hogstedt C. Lung cancer and exposure to diesel exhaust among bus garage workers. *Scand J Work Environ Health* 1990;**16**:348–54.
45 Hansen ES. Cancer incidence in an occupational cohort exposed to bitumen fumes. *Scand J Work Environ Health* 1989;**15**: 101–5.
46 Beaumont JJ, Chu GST, Jones JR *et al.* An epidemiologic study of cancer and other causes of mortality in San Francisco firefighters. *Am J Ind Med* 1991;**19**:357–72.
47 Day NE, Munoz N. "Esophagus." In: Schottenfeld D, Fraumeni JF. *Cancer epidemiology and prevention*. Philadelphia: Saunders, 1982;596–623.
48 Kristensen TS. Cardiovascular diseases and the work environment: A critical review of the epidemiologic literature on chemical factors. *Scand J Work Environ Health* 1989;**15**: 245–64.
49 Bond JA, Gown AN, Yang HL, Benditt EP, Juchau MB. Further investigations of the capacity of polynuclear aromatic hydrocarbons to elicit atherosclerotic lesions. *J Toxicol Environ Health* 1981;7:327–35.
50 Penn A, Snyder C. Arteriosclerotic plaque development is "promoted" by polynuclear aromatic hydrocarbons. *Carcinogenesis* 1988;**12**:2185–9.
51 Benditt EP. Origins of human atherosclerotic plaques. *Arch Pathol Lab Med* 1988;**112**:997–1001.
52 Murray CD, Lee KT, Kroms M, Janakidevi K. Clonal nature of atherosclerotic plaques. *Experimental and Molecular Pathology* 1988;**48**:391–402.
53 Stern FB, Halperin WE, Hornung RW, Ringenburg VL, McCammon CS. Heart disease mortality among bridge and tunnel officers exposed to carbon monoxide. *Am J Epidemiol* 1988;**128**:1276–88.
54 Sheps DS, Herbst MC, Hinderliter AL, *et al.* Production of arrythmias by elevated carboxyhemoglobin in patients with coronary artery disease. *Ann Intern Med* 1990;**113**:343–51.
55 Walden SM, Gottlieb SO. Urban angina, urban arrythmias: carbon monoxide and the heart (editorial). *Ann Intern Med* 1990;**113**:337–8.
56 Hansen ES. Chest symptoms in chimney sweeps and postmen—A comparative survey. *Int J Epidemiol* 1990;**19**:339–42.

Accepted 20 July 1992