Cancer Incidence in a Cohort of Swedish Chimney Sweeps, 1958–2006

Christer Hogstedt, MD, PhD, Catarina Jansson, PhD, Marcus Hugosson, MSc, Håkan Tinnerberg, PhD, and Per Gustavsson, MD, PhD

Objectives. We examined cancer incidence in an expanded cohort of Swedish chimney sweeps.

Methods. We added male chimney sweep trade union members (1981–2006) to an earlier cohort (employed 1918–1980) and linked them to nationwide registers of cancer, causes of deaths, and total population. The total cohort (n = 6320) was followed from 1958 through 2006. We estimated standardized incidence ratios (SIRs) using the male Swedish population as reference. We estimated exposure as years of employment and analyzed for exposure–response associations by Poisson regression.

Results. A total of 813 primary cancers were observed versus 626 expected (SIR = 1.30; 95% confidence interval = 1.21, 1.39). As in a previous follow-up, SIRs were significantly increased for cancer of the esophagus, liver, lung, bladder, and all hematopoietic cancer. New findings included significantly elevated SIRs for cancer of the colon, pleura, adenocarcinoma of the lung, and at unspecified sites. Total cancer and bladder cancer demonstrated positive exposure–response associations.

Conclusions. Exposure to soot and asbestos are likely causes of the observed cancer excesses, with contributions from adverse lifestyle factors. Preventive actions to control work exposures and promote healthier lifestyles are an important priority. (*Am J Public Health.* 2013;103:1708–1714. doi:10.2105/AJPH.2012.300860)

Scrotal cancer among young British chimney sweeps was first noted in 1775 in a classic report by Sir Percivall Pott, an English surgeon.[1] At that time, young boys were sent naked up the narrow chimneys, and Pott was inclined to blame the origin of the malignancy on the lodging of soot in the rugae of the scrotum. This observation is usually regarded as the first published description of an occupational cancer and, together with several later reports, resulted in preventive measures, mainly improved clothing and hygiene. However, it took more than 200 years before other aspects of the health of chimney sweeps were systematically studied and reported.

In 1982, the first findings from our initial epidemiological cohort study of 2071 Swedish chimney sweeps were published, showing increased mortality from several types of cancer, ischemic heart disease, diseases of the respiratory system, and accidents, injuries, or other external causes.[2] Follow-up studies (in 1987 and 1993) of an expanded cohort confirmed the initial findings on mortality.[3,4] The 1993 study also demonstrated increased risks of cancer incidence.[4] In the third, most recent update of mortality, we extended follow-up by 16 years, updated the employment histories, and added more than 1000 chimney sweeps first employed after 1980.[5] Results demonstrated significantly increased all-cause mortality, with 1841 observed deaths and a standardized mortality risk ratio of 1.29 (95% confidence interval [CI] = 1.24, 1.36). Moreover, cause-specific mortality was significantly increased for ischemic heart disease, nonmalignant respiratory diseases, alcoholism, liver cirrhosis, and external causes and suicides, as well as for all malignant tumors combined and for esophageal, bowel, liver, and lung cancer.

Increased risks of various cancer types,[6,7] ischemic heart disease,[8] and asthma[9] among chimney sweeps have also been observed in other studies. A recent large Nordic record-linkage study of occupation and cancer showed increased risks of cancer of the lung, esophagus, pharynx, bladder, pancreas, and colon among chimney sweeps.[10,11]

Traditional chimney sweeping ("black sweeping") includes removing soot from chimneys and connecting pipes, furnaces, and boilers with brushes and scrapers. Nowadays, some chimney sweeps also use handheld machines and collect soot during the sweeping with vacuum cleaners. The sweeps may also be exposed to soot while driving between 2 jobs through contamination of their clothes as well as contamination in the cars. After the sweeping, the soot is collected in closed containers at the back of the van, and at the end of the day the sweeps empty these containers in designated larger containers.

Since the middle of the 20th century, chimney sweeps in Sweden have gradually started performing additional work tasks; for example, the degreasing of kitchen ventilation equipment and fire safety inspections ("white sweeping"). Today, on average, Swedish chimney sweeps spend about half of their working hours doing black sweeping, another 25% performing fire safety inspections, and the remaining 25% degreasing kitchen and restaurant ventilation.[12]

There are no mandatory protective measures for chimney sweeps; they are supposed to use good hygiene practices (e.g., use of gloves, special soot vacuum cleaners for local exhaust ventilation, and protection masks). During normal sweeping, they usually wear normal working clothes with long legs and sleeves, but sometimes they wear shorts and short-sleeved shirts.

Coal and, to a smaller degree, wood and coke were the dominant fuels in Sweden until the introduction of oil, beginning around 1950.[13] More recently, there has been a decrease in the use of oil, with a return to wood supplemented by wood pellets. The time spent at different black sweeping tasks in 2006 was as follows: roughly 60% for wood boilers for residential heating, 25% for local wood stoves

and fireplaces, 10% for oil burners, and 5% for pellets stoves. It is estimated that this distribution has remained unchanged for several decades.[12]

Chimney soot contains several toxic agents, including carbon particles with adsorbed polycyclic aromatic hydrocarbons (PAHs), metals (e.g., arsenic, chromium, cadmium, nickel, and lead), combustion gases (e.g., sulfur dioxide and carbon monoxide), and asbestos from pipe and furnace insulation.[13] In addition, chimney sweeps are exposed to organic solvents or alkaline mixtures, especially during the cleaning of restaurants. Degreasing is performed by applying the degreasing agent as a foam and then cleaning the ducts with lots of water, which results in a high risk of dermal exposure despite use of protective clothing.

Chimney sweeps' exposure to PAHs is different from that of other occupational groups. Chimney sweeps are mainly exposed to nonvolatile and high-molecular-weight PAHs in soot, whereas workers in many other occupations (e.g., coke and gas workers) are exposed to fresh PAHs (newly released PAHs as opposed to PAHs in particles from months- or years-old soot) , which has a larger content of volatile and semivolatile PAHs.[14] Furthermore, the different fuels used for heating also influence the PAH content in soot. The exposure to 5 PAHs demonstrated to be carcinogenic in animals was about twice as high in soot from solid fuels as in soot from oil-fueled burners.[15]

An extensive occupational hygiene survey performed in Sweden in 1985 and 1986 showed high dust levels during all work operations involving contact with soot.[13] Average levels of total dust during sweeping were 3, 9, 11, and 19 milligrams per cubic meter for the 4 most common sweeping operations, exceeding the Swedish threshold limit value of 3 milligrams per cubic meter for most work operations. Exposure to both PAH and benzo (a)pyrene (BaP) was highly variable; BaP exposure varied from less than detectable (about 0.2 $\mu g/m^3$) to 9.1 micrograms per cubic meter in samples from 15 chimney sweeps. Exposure to metals and asbestos was mostly below the respective Swedish threshold limit values.[13]

When we investigated chimney sweeps' exposure levels in 2010, we found that during chimney sweeping in private homes, the median 8-hour average was 3.8 milligrams per cubic meter for inhalable dust. We recorded different measures of exposure, such as hydroxypyrene in urine as a biomarker for PAH exposure, full-shift inhalation exposure, dermal exposure, and peak exposures. During sweeping in industrial settings, exposure could be so high that it was measured in grams rather than milligrams per cubic meters, but workers wore personal protective equipment (H. Tinnerberg, unpublished data, 2010). There was also considerable dermal exposure to PAH.[16]

In the present study, we have updated follow-up of cancer incidence by 19 years, carried out in parallel with the most recent mortality report.

METHODS

The design of the original 1982 study[2] and the recently updated mortality study[5] has been described in detail previously and is summarized briefly here. We identified male Swedish chimney sweeps who were members of the national trade union during the period 1981 to 2006 (n = 1082) and added them to a cohort including all chimney sweeps who were trade union members during the years 1918 to 1980 and alive in 1958, when the Swedish Cancer Registry was established (n = 5238). From 1981 through 1997, occupational titles were not registered in the databases by the Swedish Municipal Workers' Union; however, most members could be identified through local registers.[5] The total cohort (n = 6320; 198 132 person-years) was linked to nationwide registers of cancer, causes of death, and the total population and followed from 1958 through 2006. We studied incident primary cancers coded in the Cancer Register according to the *International Classification of Diseases, 7th Revision* (*ICD-7*).[17] Although *ICD-9* and *ICD-10* are also available, the National Board of Health and Welfare transforms all cancer diagnoses to *ICD-7* codes to enable study of long trends as recommended by the International Agency for Research on Cancer.[18]

We estimated standardized incidence ratios with 95% confidence intervals,[19] using the entire Swedish male population as reference in strata defined by calendar year and 5-year age period. With the absence of data on exposure levels, the analysis had to rely on risk in relation to time since first exposure (latency) and duration of exposure.[20]

We used number of years as an active chimney sweep as a proxy for cumulative exposure in analyses stratified by duration in 4 categories (0–9, 10–19, 20–29, ≥ 30 years). We considered chimney sweeps and chimney sweep masters to be occupationally exposed, and we combined employment as either in analyses stratified by duration. Ten percent of chimney sweeps become masters. The masters were always sweeps in early working life and usually continued to do black sweeping. We allocated person-years dynamically over duration categories, treating duration of employment as a time-dependent covariate.[20]

We calculated exposure–response associations by Poisson regression for sites that showed indications of a positive trend in standardized incidence ratio with duration of employment.[19] The log of the expected number of cases in each cell of the person-years matrix, stratified as for the standardized incidence ratio analysis, was used as an offset. We investigated the statistical significance of employment duration by assigning scores (1–4) to each 10-year stratum of employment duration. We included this score as a continuous variable in the regression model.

Finally, because of the shifts in fuels and work tasks already described, we separately analyzed those starting their employment before (1671 men; 53 241 person-years) and after (4649 men; 144 890 person-years) December 31, 1950, to tentatively examine risks associated with primary exposure to wood versus oil combustion.

We performed all statistical analyses with SAS version 9.2 (SAS Institute Inc, Cary, NC).

RESULTS

The total number of incident cancers had more than doubled since the 1993 study; we observed 813 primary cancers versus the 626 expected (standardized incidence ratio [SIR] = 1.30; 95% CI = 1.21, 1.39; Table 1). We observed significantly elevated risks for cancer of the esophagus (SIR = 2.08; 95% CI = 1.19, 3.38), colon (SIR = 1.36; 95% CI = 1.02, 1.76), liver (SIR = 2.48; 95% CI = 1.47, 3.91), lung (SIR = 2.14; 95% CI = 1.77, 2.56), pleura (SIR = 3.50; 95% CI = 1.60, 6.65), and

TABLE 1—Incidence of Cancer Among Chimney Sweeps: Sweden, 1958–2006

Tumor Site	*ICD-7* Codes	Total Cohort			First Employed Before 1951			First Employed in 1951 or Later		
		No. Observed	No. Expected	SIR (95% CI)	No. Observed	No. Expected	SIR (95% CI)	No. Observed	No. Expected	SIR (95% CI)
All sites	140–209	813	625.7	1.30 (1.21, 1.39)	523	368.1	1.42 (1.30, 1.55)	290	257.6	1.13 (1.00, 1.26)
Lip	140	4	4.1	0.99 (0.27, 2.52)	3	3.0	0.99 (0.20, 2.88)	1	1.0	0.98 (0.02, 5.47)
Mouth	141–144	9	7.9	1.13 (0.52, 2.15)	6	3.9	1.53 (0.56, 3.32)	3	4.0	0.75 (0.15, 2.19)
Pharynx	145–148	9	5.7	1.57 (0.72, 2.98)	5	2.7	1.88 (0.61, 4.39)	4	3.1	1.30 (0.36, 3.34)
Esophagus (not divided by histological type)	150	16	7.7	2.08 (1.19, 3.38)	13	4.7	2.79 (1.49, 4.77)	3	3.0	0.99 (0.20, 2.89)
Esophageal adenocarcinoma (PAD 096)		2	2.5	0.80 (0.10, 2.89)	2	1.2	1.68 (0.20, 6.06)	0	1.3	
Esophageal squamous-cell carcinoma (PAD 146)		13	4.4	2.94 (1.57, 5.03)	10	2.9	3.43 (1.65, 6.31)	3	1.5	1.99 (0.41, 5.81)
Stomach	151	36	26.8	1.34 (0.94, 1.86)	28	20.1	1.39 (0.92, 2.01)	8	6.6	1.20 (0.52, 2.37)
Colon	153	55	40.6	1.36 (1.02, 1.76)	42	26.1	1.61 (1.16, 2.18)	13	14.5	0.90 (0.48, 1.54)
Rectum and anus	154	29	29.3	0.99 (0.66, 1.42)	19	18.3	1.04 (0.63, 1.62)	10	11.0	0.91 (0.44, 1.67)
Biliary passages and liver, primary	155	20	12.3	1.62 (0.99, 2.50)	13	8.2	1.58 (0.84, 2.70)	7	4.1	1.71 (0.69, 3.52)
Liver, primary	155.0	18	7.3	2.48 (1.47, 3.91)	11	4.7	2.32 (1.16, 4.16)	7	2.5	2.76 (1.11, 5.69)
Extrahepatic bile ducts	155.2	2	1.3	1.60 (0.19, 5.78)	2	0.8	2.35 (0.29, 8.50)	0	0.4	
Pancreas	157	18	16.4	1.10 (0.65, 1.74)	12	11.0	1.09 (0.56, 1.90)	6	5.4	1.12 (0.41, 2.44)
Nose and nasal sinuses	160	1	1.4	0.71 (0.02, 3.96)	0	0.8		1	0.6	1.76 (0.04, 9.81)
Larynx	161	10	6.0	1.65 (0.79, 3.04)	6	3.8	1.59 (0.58, 3.45)	4	2.3	1.77 (0.48, 4.54)
Bronchus and lung, primary	162.1	119	55.6	2.14 (1.77, 2.56)	84	36.5	2.30 (1.84, 2.85)	35	19.2	1.82 (1.27, 2.54)
Adenocarcinoma (PAD 096)		24	12.8	1.88 (1.20, 2.80)	12	6.8	1.76 (0.91, 3.08)	12	6.0	2.01 (1.04, 3.51)
Squamous-cell carcinoma (PAD 146)		36	18.0	2.00 (1.40, 2.77)	27	13.3	2.03 (1.34, 2.96)	9	4.8	1.89 (0.87, 3.60)
Undifferentiated and small cell carcinoma (PAD 186+196)		52	21.5	2.42 (1.81, 3.17)	40	14.3	2.79 (1.99, 3.80)	12	7.2	1.68 (0.87, 2.93)
Pleura	162.2	9	2.6	3.50 (1.60, 6.65)	8	1.3	6.19 (2.67, 12.19)	1	1.3	0.78 (0.02, 4.37)
Prostate	177	173	154.7	1.12 (0.96, 1.30)	112	96.6	1.16 (0.95, 1.40)	61	58.1	1.05 (0.80, 1.35)
Testis	178	14	10.9	1.28 (0.70, 2.16)	2	1.7	1.20 (0.15, 4.35)	12	9.2	1.30 (0.67, 2.27)
Kidney	180	27	21.8	1.24 (0.82, 1.80)	16	13.4	1.20 (0.69, 1.95)	11	8.5	1.30 (0.65, 2.32)
Urinary bladder	181.0	70	38.9	1.80 (1.40, 2.27)	54	24.6	2.19 (1.65, 2.86)	16	14.3	1.12 (0.64, 1.82)
Malignant melanoma of skin	190	20	26.8	0.75 (0.46, 1.15)	7	10.0	0.70 (0.28, 1.44)	13	16.8	0.77 (0.41, 1.32)
Skin (melanoma excluded)	191	16	21.4	0.75 (0.43, 1.22)	12	14.1	0.85 (0.44, 1.48)	4	7.2	0.55 (0.15, 1.42)
Nervous system	193	21	24.4	0.86 (0.53, 1.32)	5	9.8	0.51 (0.17, 1.19)	16	14.6	1.09 (0.63, 1.78)
Connective tissue, muscle	197	3	5.2	0.58 (0.12, 1.70)	2	2.4	0.82 (0.10, 2.95)	1	2.7	0.37 (0.01, 2.06)
Unspecified sites	199	33	18.3	1.80 (1.24, 2.53)	18	10.9	1.65 (0.98, 2.60)	15	7.4	2.03 (1.13, 3.34)
All hematopoietic cancers	200–209	75	58.1	1.29 (1.02, 1.62)	38	29.9	1.27 (0.90, 1.74)	37	28.2	1.31 (0.92, 1.81)
Malignant non-Hodgkin lymphoma	200	28	21.5	1.30 (0.87, 1.88)	13	10.6	1.23 (0.65, 2.10)	15	10.9	1.37 (0.77, 2.26)
Hodgkin disease	201	8	5.7	1.41 (0.61, 2.77)	2	2.0	0.98 (0.12, 3.53)	6	3.6	1.65 (0.60, 3.58)
Reticulosis and related forms	202	3	1.5	1.95 (0.40, 5.70)	1	0.6	1.75 (0.04, 9.78)	2	1.0	2.07 (0.25, 7.47)
Multiple myeloma plasmocytoma	203	9	8.8	1.02 (0.47, 1.93)	6	5.6	1.07 (0.39, 2.34)	3	3.2	0.92 (0.19, 2.70)
Leukemia	204–207	21	17.2	1.22 (0.76, 1.87)	13	9.3	1.40 (0.75, 2.40)	8	7.9	1.01 (0.44, 1.99)
Lymphatic leukemia	204	13	8.4	1.55 (0.83, 2.65)	6	4.9	1.23 (0.45, 2.68)	7	3.5	1.99 (0.80, 4.11)
Myeloid leukemia	205	6	7.2	0.83 (0.30, 1.80)	5	3.5	1.41 (0.46, 3.30)	1	3.7	0.27 (0.01, 1.51)

Note. CI = confidence interval; *ICD-7 = International Classification of Diseases, 7th Revision;* PAD = pathologic anatomic diagnosis; SIR = standardized incidence ratio. We estimated SIRs using the Swedish male population as reference, adjusting for effects of age and calendar year.

bladder (SIR = 1.80; 95% CI = 1.40, 2.27), in addition to all hematopoietic cancers (SIR = 1.29; 95% CI = 1.02, 1.62) and cancers of unspecified sites (SIR = 1.80; 95% CI = 1.24, 2.53; Table 1).

Compared with the prior update with follow-up (1958–1987), the earlier nonsignificant excesses of colon cancer, pleural malignancies, and cancer at unspecified sites were now statistically significant. The elevated incidence of adenocarcinoma of the lung (SIR = 1.88) was a new finding. There was no excess of melanoma nor nonmelanoma skin cancer (SIR = 0.75) and not a single observed case of scrotal cancer.

Total cancers of all sites combined demonstrated a significant positive exposure–response association in the analyses stratified by duration ($P < .03$), and there were borderline positive significant exposure–response tendencies for several sites (Table 2). The trend tests resulted in P values of .06 for bladder cancer, .07 for prostate cancer, .09 for kidney cancer, and .11 for colon cancer.

The latency analyses were not very informative, as chimney sweeps start their employment at young ages, and 718 out of the 813 observed cancers were diagnosed more than 30 years after first employment (data not shown).

There were differences in the risk estimates for those employed from 1951 onwards, when oil became the dominant fuel, compared with those first employed before 1951 (Table 1). The total excess was only of borderline significance for the younger subcohort, and there were no significant excesses of cancer of the esophagus, colon, pleura, or bladder in this subcohort. However, the excesses for lung and

TABLE 2—Incidence of Cancer Among Chimney Sweeps, by Duration of Employment: Sweden, 1958–2006

Tumor Site	*ICD-7* Codes	Duration of Employment: 0–9 Years, No. (SIR; 95% CI)	10–19 Years, No. (SIR; 95% CI)	20–29 Years, No. (SIR; 95% CI)	≥ 30 Years, No. (SIR; 95% CI)	*P* for Trend[a]
All sites	140–209	233 (1.19; 1.04, 1.35)	154 (1.20; 0.02, 1.41)	142 (1.42; 1.20, 1.68)	284 (1.41; 1.25, 1.58)	.028
Pharynx	145–148	3 (1.41; 0.29, 4.11)	1 (0.80; 0.02, 4.48)	0	5 (3.46; 1.12, 8.07)	.235
Esophagus (not divided by histological type)	150	3 (1.33; 0.27, 3.89)	5 (3.23; 1.05, 7.55)	3 (2.39; 0.49, 6.98)	5 (1.89; 0.62, 4.42)	.825
Stomach	151	8 (1.40; 0.61, 2.77)	6 (1.26; 0.46, 2.74)	6 (1.39; 0.51, 3.02)	16 (1.34; 0.76, 2.17)	
Colon	153	12 (1.04; 0.54, 1.82)	8 (0.99; 0.43, 1.95)	11 (1.66; 0.83, 2.98)	24 (1.67; 1.07, 2.49)	.11
Rectum and anus	154	9 (1.07; 0.49, 2.02)	4 (0.68; 0.19, 1.74)	10 (2.08; 1.00, 3.83)	6 (0.59; 0.22, 1.29)	
Biliary passages and liver, primary	155	6 (1.82; 0.67, 3.97)	2 (0.84; 0.10, 3.05)	1 (0.51; 0.01, 2.86)	11 (2.32; 1.16, 4.15)	
Liver, primary	155.0	6 (2.98; 1.09, 6.48)	2 (1.43; 0.17, 5.16)	1 (0.89; 0.02, 4.95)	9 (3.30; 1.51, 6.27)	.746
Extrahepatic bile ducts	155.2	0	0	0	2 (4.19; 0.51, 15.14)	
Pancreas	157	3 (0.71; 0.15, 2.08)	4 (1.26; 0.34, 3.23)	4 (1.48; 0.40, 3.80)	7 (1.11; 0.45, 2.29)	.588
Larynx	161	5 (2.87; 0.93, 6.70)	1 (0.80; 0.02, 4.45)	3 (2.88; 0.59, 8.42)	1 (0.50; 0.01, 2.77)	
Bronchus and lung, primary	162.1	41 (2.69; 1.93, 3.65)	19 (1.71; 1.03, 2.66)	18 (1.90; 1.12, 3.00)	41 (2.07; 1.49, 2.81)	.323
Adenocarcinoma (PAD 096)		9 (2.10; 0.96, 3.98)	5 (1.82; 0.59, 4.25)	3 (1.39; 0.29, 4.05)	7 (1.96; 0.79, 4.05)	
Squamous-cell carcinoma (PAD 146)		14 (3.32; 1.81, 5.57)	3 (0.87; 0.18, 2.55)	6 (1.95; 0.71, 4.24)	13 (1.78; 0.95, 3.05)	
Undifferentiated and small cell carcinoma (PAD 186+196)		16 (2.80; 1.60, 4.54)	9 (2.11; 0.96, 4.00)	7 (1.89; 0.76, 3.89)	20 (2.57; 1.57, 3.97)	
Pleura	162.2	2 (2.29; 0.28, 8.27)	1 (1.75; 0.04, 9.77)	0	6 (8.63; 3.17, 18.77)	.08
Prostate	177	36 (0.83; 0.58, 1.15)	32 (1.05; 0.72, 1.48)	41 (1.63; 1.17, 2.22)	64 (1.15; 0.89, 1.47)	.072
Kidney	180	5 (0.77; 0.25, 1.80)	4 (0.87; 0.24, 2.24)	6 (1.61; 0.59, 3.50)	12 (1.71; 0.88, 2.98)	.087
Urinary bladder	181.0	14 (1.24; 0.68, 2.09)	13 (1.64; 0.87, 2.80)	13 (2.02; 1.07, 3.45)	30 (2.26; 1.52, 3.22)	.055
Malignant melanoma of skin	190	15 (1.28; 0.72, 2.11)	0	2 (0.50; 0.06, 1.80)	3 (0.62; 0.13, 1.82)	
Skin (melanoma excluded)	191	4 (0.68; 0.19, 1.74)	1 (0.24; 0.01, 1.33)	5 (1.51; 0.49, 3.51)	6 (0.75; 0.28, 1.64)	
Unspecified sites	199	7 (1.24; 0.50, 2.56)	13 (3.43; 1.83, 5.87)	6 (2.02; 0.74, 4.39)	7 (1.18; 0.47, 2.43)	
All hematopoietic cancer	200–209	33 (1.55; 1.07, 2.18)	18 (1.46; 0.86, 2.30)	3 (0.34; 0.07, 0.99)	21 (1.34; 0.83, 2.05)	.243
Malignant non-Hodgkin lymphoma	200	15 (1.88; 1.05, 3.11)	4 (0.86; 0.23, 2.19)	2 (0.60; 0.07, 2.15)	7 (1.27; 0.51, 2.61)	
Hodgkin disease	201	5 (1.72; 0.56, 4.01)	2 (1.56; 0.19, 5.64)	0	1 (1.18; 0.03, 6.59)	
Multiple myeloma plasmocytoma	203	3 (1.18; 0.24, 3.46)	2 (1.12; 0.14, 4.05)	0	4 (1.30; 0.35, 3.33)	
Leukemia	204–207	4 (0.66; 0.18, 1.68)	8 (2.24; 0.97, 4.42)	1 (0.38; 0.01, 2.14)	8 (1.62; 0.70, 3.19)	

Note. CI = confidence interval; *ICD*-7 = International *Classification of Diseases, 7th Revision;* PAD = pathologic anatomic diagnosis; SIR = standardized incidence ratio.
[a]From Poisson regression.

liver cancers from the pre-1951 subcohort remained significantly increased in the younger subcohort.

DISCUSSION

Consistent with our earlier findings from 1993,[4] this cohort study of Swedish chimney sweeps showed a significantly increased risk of all malignant tumors combined and of esophageal, lung, bladder, and all hematopoietic cancers. New findings include a significantly increased incidence of pleural malignancies, liver and colon cancer, and tumors at unspecified sites as well as a new finding of excess of lung adenocarcinoma. The results are dramatic, with more site-specific cancer excesses than those described for any other occupational group, demonstrating significantly increased risks for twice as many cancer sites as the latest mortality study did (8 vs 4). Results in the earlier follow-up (1982–1993) could have resulted from historic work environment exposures, but this follow-up demonstrates that those working during more recent decades also had high risks of excess cancers.

Our study includes a large number of participants with long-term and complete follow-up. Because of the trade union's high coverage and the high quality of the nationwide registers used, the selection bias is minimized. The follow-up and the detection of cancer through the Swedish Cancer Register are regarded as virtually complete.[21]

The known carcinogens to consider for individual cancer sites are mainly PAH and asbestos but also metals and adverse lifestyle factors. The chimney sweeps were exposed to high levels of soot[12,13,22] rich in PAH. Experimental animal studies show that several PAHs, including BaP, cause cancer,[23] and increased risks of cancer, predominantly cancer of the lung and bladder, have been observed in a large number of epidemiological studies of workers exposed to PAHs.[22,23] A working group at the International Agency for Research on Cancer recently assessed the cancer risks associated with household use of biomass fuels and coal.[24] It found sufficient evidence that the indoor household combustion of coal is associated with cancer in humans (particularly lung cancer) and limited evidence that household combustion of biomass fuel (primarily wood) is associated with cancer.

Excesses in lung, bladder, and esophageal cancer have been associated with tobacco smoking, and excesses of esophageal and liver cancer with high alcohol consumption. Unfortunately, no individual data regarding adverse lifestyle factors are included in the union membership databases or in the nationwide registers used. However, we have some information from a health survey of 1040 chimney sweeps performed in 1972.The proportion of current smokers was approximately 25% higher among the chimney sweeps than in the same-aged general population. For those younger than 50 years, alcohol use among chimney sweeps was similar to that for the general population, whereas for those 50 to 69 years old, it was almost double.[25]

Although the observed risks may have been partially caused by smoking, it is not likely that this potential confounding is substantial. There is good evidence that occupational cancer studies are not often seriously confounded by smoking.[26] This is because modest differences in smoking habits between 2 groups (e.g., the 25% higher prevalence of smoking reported in 1972 for the chimney sweeps) will result in only small amounts of confounding. As Blair et al. noted,

> For confounders to have much of an impact, both associations (i.e., risk factor for the disease and correlation with the exposure of interest) must be strong. If this is not the case, the impact of confounding cannot be large. Situations fulfilling these requirements are not common.[26(p200)]

Axelson estimated that lung cancer relative risks of 1.6 or higher are very unlikely to be explained by confounding from smoking.[27] Similar estimates would be reasonable for other cancer sites where smoking is a risk factor. In the following sections, potential lifestyle confounding is further discussed in relation to specific cancer sites.

The less increased risks for those employed from 1951 onwards, compared with the subcohort employed earlier, may indicate that exposure to oil soot is somewhat less carcinogenic compared with soot from wood and coal burning. However, some types of cancer need longer latency periods to develop, and the number of cancers in the younger cohort is limited. Furthermore, there was neither a quick nor a complete change from wood to oil burning, and the reintroduction of wood burning prevents firmer conclusions.

Lower socioeconomic position has been demonstrated to affect cancer survival negatively in various ways.[28] With the exception of bladder cancer, in both the present and the mortality study,[5] the cancers showing excess risks were at sites with generally poor survival, regardless of early diagnosis. The standardized incidence risk ratio for bladder cancer (SIR = 1.80; 95% CI = 1.40, 2.27) was somewhat higher than the standardized mortality ratio (1.31; 95% CI = 0.72, 2.20). The fact that chimney sweeps belong to a lower socioeconomic group could have meant higher ratios for mortality than for incidence, but this was not the case.

We observed consistent exposure–response associations mainly between employment duration and the risk of total cancer and bladder cancer. However, employment duration is a nonspecific proxy for exposure, and the absence of association with duration does not imply absence of effect of the occupational exposure.

Pleural and Lung Cancer

All pleural malignancies coded according to the *ICD-7* were mesotheliomas (pathologic anatomic diagnosis [PAD] 776) and showed excess risks, mainly among the long-term employed. Mesotheliomas are strongly attributed to asbestos exposure but not to tobacco smoking.[29]

Our finding of an increased risk of lung cancer could be explained by exposure to chemical carcinogens such as PAHs, arsenic, nickel, chromium, and asbestos—all substances to which chimney sweeps are potentially exposed. The analyses of histological types of lung cancer showed increased risks of similar magnitude for all 3 types. Tobacco smoking has been reported to increase the risk of squamous-cell, oat-cell, and undifferentiated carcinoma more strongly than for adenocarcinoma,[30] whereas asbestos exposure has been associated with a relatively larger increase in adenocarcinoma compared with other histological lung cancer types.[31] The high proportion of adenocarcinoma might indicate that asbestos has influenced the lung cancer excess in the cohort.

The magnitude of the excess risk of lung cancer in this study appears too large to be explained simply by confounding from tobacco smoking, although interaction of smoking and work exposures could not be assessed.

Bladder Cancer

An interesting finding was that the standardized incidence ratio for bladder cancer increased monotonically with employment duration and that the trend approached statistical significance. Since a similar trend was not seen for lung cancer, the increased risks for the different cancers are possibly related to different exposure scenarios among the chimney sweeps. The increased risk of bladder cancer appeared stronger among chimney sweeps employed before 1951 (Table 1).

In a record linkage study from the Nordic countries, an excess of bladder cancer incidence was also noted among chimney sweeps in Denmark, and a small excess based on low numbers was found among Norwegian and Finnish chimney sweeps.[10] To our knowledge, it has not been investigated whether chimney sweeps are exposed to aromatic amines, which are known bladder carcinogens.[31] Tobacco smoking is a well-established risk factor for bladder cancer[32] and may have had some, but limited, influence on the risk estimate.[26]

Esophageal Cancer

The increased risk of esophageal squamous-cell carcinoma could be explained by exposures experienced by chimney sweeps. PAH exposure[33] and occupational exposure to combustion products[33,34] have been suggested as risk factors for esophageal squamous-cell carcinoma. PAHs or dust could be deposited in the airway region and then swallowed, thereby directly acting on the esophageal mucosa[33,35] and potentially explaining the finding of an increased risk of esophageal cancer. Alcohol and tobacco smoking are well-known risk factors for esophageal cancer,[36,37] but are not likely to have had a major influence on the risk estimates. Despite the finding in the 1972 survey of heavier alcohol consumption among chimney sweeps in the older age groups, there is little suggestion that these habits remain in our cohort. In our mortality study of chimney sweeps employed for more than 30 years, there was no excess of death from either liver cirrhosis or external causes and alcoholism.[5]

Liver Cancer

The risk of liver cancer was significantly increased for the older as well as the younger subcohorts. Alcohol is a well-known risk factor for liver cancer; in the Nordic countries, increased rates of liver cancer have been found in occupations with potentially easy workplace access to alcohol or with cultural traditions of high alcohol consumption, such as waiters, cooks, beverage workers, journalists, and seamen.[10] In a Finnish study, an elevated liver cancer incidence was observed for male printers, varnishers, and lacquerers, all with exposure to different kinds of chlorinated as well nonchlorinated solvents.[38] Chimney sweeps are exposed to organic solvents for cleaning fatty ventilation tubes, which might have contributed to the excess. The significant excess of liver cancer among chimney sweeps employed more than 30 years seems less likely to be caused by excessive alcohol habits, as this group did not show an excess of death from either alcoholism or liver cirrhosis in our mortality study.[5] However, we cannot exclude potential influence from solvent exposure and alcohol habits.

Colon Cancer

Colon cancer incidence demonstrated excess risk and a nonsignificant employment–duration association, suggesting the influence of occupational risk factors. The most established occupational risk factor for colon cancer is physical inactivity,[39] which certainly does not characterize chimney sweeping. Some studies suggest an association with asbestos exposure,[40] whereas the effect of diet, alcohol, and tobacco is weak and under debate.[41] Occupational asbestos exposure seems to be the most likely explanation if this cancer is work related, although the chimney sweeps' exposure to asbestos was only moderate.[13]

Skin Cancer

It is somewhat surprising that skin squamous cancer risk was less than unity, as BaP is a well-known skin carcinogen, and the original finding by Pott was of scrotal cancer.[1] Sunlight and ultraviolet radiation are well-known risk factors for squamous skin cancer and malignant melanoma.[42] In a large Nordic study, many white-collar occupations demonstrated increased risks of malignant melanoma as well as nonmelanoma skin cancer, whereas many blue-collar occupations had decreased risks.[10] A possible explanation of our finding is that, in previous decades, visiting southern countries for sun tourism was more common among Swedish white-collar workers than blue-collar workers.

Conclusions

The present study provides strong evidence of increased cancer incidence risk among chimney sweeps, particularly for cancer of the esophagus, colon, liver, lung, pleura, bladder, and all hematopoietic cancers. Exposure to various occupational carcinogenic agents appears the most likely explanation for these increases, although contribution from and potential interaction with smoking and alcohol consumption cannot be dismissed. Thus, although protective measures seem to have prevented development of scrotal cancer among chimney sweeps, they suffer from excesses of cancers of many other sites as well as other diseases. Preventive actions to promote a healthier lifestyle should be provided to halt a continuation of these strong excess risks, and chemical exposures during different operations must be better measured and controlled. In addition, our findings may have relevance for the general population exposed to similar compounds in higher doses (e.g., during indoor cooking in developing countries) or in lower doses in the general environment. ■

About the Authors

Christer Hogstedt, Marcus Hugosson, and Per Gustavsson are with the Unit of Occupational Medicine, Institute of Environmental Medicine, Karolinska Institutet, Stockholm, Sweden. Catarina Jansson is with the Division of Insurance Medicine, Department of Clinical Neuroscience, Karolinska Institutet, Stockholm. Håkan Tinnerberg is with the Division of Occupational and Environmental Medicine, Department of Laboratory Medicine, Skåne University Hospital, Lund University, Lund, Sweden.

Correspondence should be sent to Christer Hogstedt, Tullgårdsgatan 28, 116 68 Stockholm, Sweden (e-mail: christer.hogstedt@gmail.com). Reprints can be ordered at http://www.ajph.org by clicking the "Reprints" link.

This article was accepted April 10, 2012.

Contributors

All authors contributed directly to the conceptualization of the study, the establishment of the final database, the design, the analysis of the data, and the writing of

the article. C. Hogstedt established the original cohort, participated in all elements of the study, and was responsible for the article. C. Jansson established the database and contributed to the analyses. M. Hugosson did the statistical analyses. H. Tinnerberg was responsible for the occupational hygiene descriptions. P. Gustavsson, the principal investigator for the current chimney sweep project, participated in all elements of the study.

Acknowledgments

Funding was received from the Swedish Council for Working Life and Social Research and from the Swedish Municipal Workers' Union.

We thank the Swedish Municipal Workers' Union and the Swedish Association of Chimney Sweep Masters for valuable assistance during this study. We are most grateful for editing by David Wegman, University of Massachusetts at Lowell; statistical input from Matteo Bottai, Unit of Biostatistics, Institute of Environmental Medicine, Karolinska Institutet; and for data of exposure measurements among chimney sweeps from Ronny Kammer at Lund University. A work visit by C. Hogstedt to the San Michele Foundation at Capri, Italy, was very helpful for finalizing the report.

Human Participant Protection

This study satisfied all criteria for the ethical treatment of human participants and was approved by the Regional Ethics Committee in Stockholm (Dnr 2007-306-31).

References

1. Waldron HA. A brief history of scrotal cancer. *Br J Ind Med.* 1983;40(4):390–401.

2. 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(suppl 1):72–78.

3. Gustavsson P, Gustavsson A, Hogstedt C. Excess mortality among Swedish chimney sweeps. *Br J Ind Med.* 1987;44(11):738–743.

4. Evanoff BA, Gustavsson P, Hogstedt C. Mortality and incidence of cancer in a cohort of Swedish chimney sweeps: an extended follow-up study. *Br J Ind Med.* 1993;50(5):450–459.

5. Jansson C, Alderling M, Hogstedt C, Gustavsson P. Mortality among Swedish chimney sweeps (1952–2006): an extended cohort study. *Occ Env Med.* 2011; 69(1):41–47.

6. Hansen ES, Olsen JH, Tilt B. Cancer and non-cancer mortality of chimney sweeps in Copenhagen. *Int J Epidemiol.* 1982;11(4):356–361.

7. Perez-Gomez B, Pollan M, Gustavsson P, et al. Cutaneous melanoma: hints from occupational risks by anatomic site in Swedish men. *Occup Environ Med.* 2004;61(2):117–126.

8. Hansen ES. Mortality from cancer and ischemic heart disease in Danish chimney sweeps: a five-year follow-up. *Am J Epidemiol.* 1983;117(2):160–164.

9. Li X, Sundquist J, Sundquist K. Socioeconomic and occupational groups and risk of asthma in Sweden. *Occup Med (Lond).* 2008;58(3):161–168.

10. Pukkala E, Martinsen JI, Lynge E, et al. Occupation and cancer follow-up of 15 million people in five Nordic countries. *Acta Oncol.* 2009;48(5):646–790.

11. Kjaerheim K, Martinsen JI, Lynge E, et al. Effects of occupation on risks of avoidable cancers in the Nordic countries. *Eur J Cancer.* 2010;46(14):2545–2554.

12. AG-Framtid. *The Future Regulation of Chimney Sweeping Activities* [in Swedish]. Stockholm, Sweden: Swedish Chimney Sweep Master Union; December 2008. Final report.

13. Andersson K. *Chimney Sweeps' Exposure to Dust, PAH, Metals, and Asbestos* [in Swedish]. Gothenburg, Sweden: Swedish Environmental Research Institute; 1987. Report no. B 850, 3.

14. Kim D, Kumfer BM, Anastasio C, Kennedy AM, Young TM. Environmental aging of polycyclic aromatic hydrocarbons on soot and its effect on source identification. *Chemosphere.* 2009;76(8):1075–1081.

15. Knecht U, Bolm-Audorff U, Woitowitz H-J. Atmospheric concentrations of polycyclic aromatic hydrocarbons during chimney sweeping. *Br J Ind Med.* 1989;46 (7):479–482.

16. Kammer R, Eriksson K, Tinnerberg H. Development and evaluation of a tape-stripping technique for measuring dermal exposure to pyrene and benzo(a)pyrene. *J Environ Monit.* 2011;13(8):2165–2171.

17. *Manual of The International Statistical Classification of Diseases, Injuries and Causes of Death, Seventh Revision.* Geneva, Switzerland: World Health Organization; 1967.

18. Muir CS, Percy C. Cancer registration: principles and methods. Classification and coding of neoplasms. *IARC Sci Publ.* 1991;(95):64–81.

19. Ulm K. A simple method to calculate the confidence interval of a standardized mortality ratio (SMR). *Am J Epidemiol.* 1990;131(2):373–375.

20. Breslow NE, Day NE. Statistical methods in cancer research. Volume II—the design and analysis of cohort studies. *IARC Sci Publ.* 1987;(82):1–406.

21. Barlow L, Westergren K, Holmberg L, Talbäck M. The completeness of the Swedish Cancer Register: a sample survey for year 1998. *Acta Oncol.* 2009;48 (1):27–33.

22. International Agency for Research on Cancer. Some non-heterocyclic polycyclic aromatic hydrocarbons and some related exposures. In: *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans.* Vol. 92. Lyon, France: International Agency for Research on Cancer; 2010:89–93.

23. Boffetta P, Jourenkova N, Gustavsson P. Cancer risk from occupational and environmental exposure to polycyclic aromatic hydrocarbons. *Cancer Causes Control.* 1997;8(3):444–472.

24. *Household Use of Solid Fuels and High-temperature Frying.* Lyon, France: World Health Organization, International Agency for Research on Cancer; 2010.

25. Swensson Å. *Chimney Sweeps Trade Union Health Survey 1972* [in Swedish]. Stockholm, Sweden: National Board of Occupational Safety and Health; 1974. Report AMMM 101/04.

26. Blair A, Stewart P, Lubin JH, Forastiere F. Methodological issues regarding confounding and exposure misclassification in epidemiological studies of occupational exposures. *Am J Ind Med.* 2007;50(3): 199–207.

27. Axelson O. Aspects on confounding in occupational health epidemiology. *Scand J Work Environ Health.* 1978;4:98–102.

28. Kogevinas M, Porta M. Socioeconomic differences in cancer survival: a review of the evidence. *IARC Sci Publ.* 1997;(138):177–206.

29. Straif K, Benbrahim-Tallaa L, Baan R, et al. A review of human carcinogens—part C: metals, arsenic, dusts, and fibres. *Lancet Oncol.* 2009;10(5):453–454.

30. Pesch B, Kendzia B, Gustavsson P, et al. Cigarette smoking and lung cancer—relative risk estimates for the major histological types from a pooled analysis of case–control studies. *Int J Cancer.* 2012;131(5):1210–1219.

31. Henderson DW, Rödelsperger K, Worrowitz H-J, Leigh J. After Helsinki: a multidisciplinary review of the relationship between asbestos exposure and lung cancer, with emphasis on studies published during 1997–2004. *Pathology.* 2004;36(6):517–550.

32. Silverman DT, Devesa SS, Moore LE, et al. Bladder cancer. In: Schottenfeld D, Fraumeni JF, eds. *Cancer Epidemiology and Prevention.* 3rd ed. New York, NY: Oxford University Press; 2006:1101–1127.

33. Roth MJ, Wei WQ, Baer J, et al. Aryl hydrocarbon receptor expression is associated with a family history of upper gastrointestinal tract cancer in a high-risk population exposed to aromatic hydrocarbons. *Cancer Epidemiol Biomarkers Prev.* 2009;18(9):2391–2396.

34. Gustavsson P, Evanoff B, Hogstedt C. Increased risk of esophageal cancer among workers exposed to combustion products. *Arch Environ Health.* 1993;48(4):243–245.

35. Gustavsson P, Jakobsson R, Johansson H, et al. Occupational exposures and squamous cell carcinoma of the oral cavity, pharynx, larynx, and oesophagus: a case–control study in Sweden. *Occup Environ Med.* 1998;55 (6):393–400.

36. Jansson C, Johansson AL, Bergdahl IA, et al. Occupational exposures and risk of esophageal and gastric cardia cancers among male Swedish construction workers. *Cancer Causes Control.* 2005;16(6):755–764.

37. Castellsagué X, Muñoz N, De Stefani E, et al. Independent and joint effects of smoking and alcohol drinking on the risk of esophageal cancer in men and women. *Int J Cancer.* 1999;82(5):657–664.

38. Lindbohm ML, Sallmén M, Kyyrönen P, Kauppinen T, Pukkala E. Risk of liver cancer and exposure to organic solvents and gasoline vapors among Finnish workers. *Int J Cancer.* 2009;124(12):2954–2959.

39. Friedenreich CM, Neilson HK, Lynch BM. State of the epidemiological evidence on physical activity and cancer prevention. *Eur J Cancer.* 2010;46(14):2593–2604.

40. Clin B, Morlais F, Launoy G, et al. Cancer incidence within a cohort occupationally exposed to asbestos: a study of dose–response relationships. *Occup Environ Med.* 2011;68(11):832–836.

41. Schottenfeld D, Winawer SJ. Cancers of the large intestine. In: Schottenfeld D, Fraumeni JF, eds. *Cancer Epidemiology and Prevention.* 2nd ed. Oxford, UK: Oxford University Press; 1996:813–840.

42. Leiter U, Garbe C. Epidemiology of melanoma and nonmelanoma skin cancer—the role of sunlight. *Adv Exp Med Biol.* 2008;624:89–103.