Original article

Scand J Work Environ Health 2006;32(4):270–275

Occupational exposures and head and neck cancers among Swedish construction workers

by Mark P Purdue, PhD,[1] Bengt Järvholm, MD,[2] Ingvar A Bergdahl, PhD,[2] Richard B Hayes, PhD,[1] Dalsu Baris, PhD[1]

Purdue MP, Järvholm B, Bergdahl IA, Hayes RB, Baris D. Occupational exposures and head and neck cancers among Swedish construction workers. *Scand J Work Environ Health* 2006;32(4):270–275.

Objectives Occupational exposures in the construction industry may increase the risk of head and neck cancers, although the epidemiologic evidence is limited by problems of low study power and inadequate adjustment for tobacco use. In an attempt to address this issue, the relationship between selected occupational exposures and head and neck cancer risk was investigated using data from a large cohort of Swedish construction workers.

Methods Altogether 510 squamous cell carcinomas of the head and neck (171 in the oral cavity, 112 in the pharynx, 227 in the larynx) were identified during 1971–2001 among 307 799 male workers in the Swedish construction industry. Exposure to diesel exhaust, asbestos, organic solvents, metal dust, asphalt, wood dust, stone dust, mineral wool, and cement dust was assessed using a semi-quantitative job-exposure matrix. Rate ratios (RR) and 95% confidence intervals (95% CI) were calculated for head and neck cancers in relation to occupational exposure, using Poisson regression with adjustment for age and smoking status.

Results Asbestos exposure was related to an increased laryngeal cancer incidence (RR 1.9, 95% CI 1.2–3.1). Excesses of pharyngeal cancer were observed among workers exposed to cement dust (RR 1.9, 95% CI 1.2–3.1). No occupational exposures were associated with oral cavity cancer. These findings did not materially change upon additional adjustment for cigarette pack-years.

Conclusions These findings offer further evidence that asbestos increases the risk of laryngeal cancer. The observation of a positive association between cement dust exposure and pharyngeal cancer warrants further investigation.

Key terms asbestos; cement dust; cohort study; laryngeal cancer; oral cancer; pharyngeal cancer.

There is considerable international variation in the incidence of squamous cell carcinomas of the oral cavity, the pharynx, and the larynx ("head and neck cancers") (1). In Sweden, the incidence of these cancers is relatively low. Incidence rates for cancer of the oral cavity and pharynx from 1993 to 1997 (age-standardized to the world population) were 6.78 and 3.19 per 100 000 for Swedish men and women, respectively, while the rates for laryngeal cancer were lower (2.20 per 100 000 for men, 0.29 per 100 000 for women) (1).

Risk factors for head and neck cancers include tobacco and alcohol use (2) and probably low dietary intake of fruits and vegetables (2). Some studies have pointed to occupational risks. Occupational exposure to mists of strong inorganic acids is an established cause of laryngeal cancer (3). Other suspected risk factors for laryngeal cancer include work in the rubber industry (4, 5) and exposure to cement dust (6, 7) and asbestos (8–11). There is little evidence of a relationship between occupational exposures and cancers of the oral cavity and pharynx; some chemicals that have been linked to these cancers include polycyclic aromatic hydrocarbons (12) and formaldehyde (13).

Construction workers may be exposed to known or suspected airborne carcinogens, including silica, asbestos, cement dust, solvents and diesel particulates, with potential exposure to the upper aerodigestive tract. Workers in the construction industry have been reported to have elevated rates of head and neck cancers in several studies (13–19), although not in all investigations (20,

1 Occupational and Environmental Epidemiology Branch, Division of Cancer Epidemiology and Genetics, National Cancer Institute, National Institutes of Health, Rockville, Maryland, United States.

2 Occupational Medicine, Department of Public Health and Clinical Medicine, Umeå University, Umeå, Sweden.

Reprint requests to: Dr MP Purdue, Occupational and Environmental Epidemiology Branch, Division of Cancer Epidemiology and Genetics, National Cancer Institute, 6120 Executive Blvd, EPS 8111, Rockville, MD, 20852, USA. [E-mail: purduem@mail.nih.gov]

21). However, this epidemiologic evidence is generally limited by the absence of information on possibly confounding exposures. We analyzed data from a large cohort of Swedish construction workers for which smoking data were available to investigate further the relationship between occupational exposures in the construction industry and head and neck cancers.

Study population and methods

The studied cohort has been described earlier (22, 23). Briefly, in 1968 the Swedish construction industry started the Organization for Working Environment, Occupational Safety and Health (in Sweden, *Bygghälsan*), a program to offer nationwide health services to all employees of the Swedish construction industry. As part of this program, regular health examinations were offered to all blue-collar and white-collar workers in the building industry through regular personal invitations (at intervals of 2–3 years) and through visits or advertisements at virtually all major building sites. Although the program was voluntary, approximately 80% of the eligible workers participated at least once. Examinations were conducted at stationary or mobile clinics, typically staffed by a few nurses and a physician. A computer register includes examination data from 389 132 workers participating in *Bygghälsan* between 1971 and 1993.

A job-exposure matrix was developed to assign exposures (diesel exhaust, asbestos, organic solvents, metal dust, asphalt, wood dust, stone dust, mineral wool, and cement dust) for over 300 job codes in the industry (24, 25), based on a survey of occupational exposures carried out by *Bygghälsan* from 1971 through 1976, where each occupation was studied at visits to approximately five different sites in different geographic regions of Sweden. These assessments were carried out by a team of occupational hygienists and physicians. Only the job title at each worker's first health examination was used, since the information available was insufficient for constructing lifetime occupational histories. Each exposure was graded on an ordinal scale (0–5), with level 3 corresponding to the threshold limit for the exposure in question. We categorized the exposure scales into no exposure (0), moderate exposure (0.5–1), and high exposure (2–5). Most of the exposed workers had an ordinal score of 1; most of the workers classified as having high exposure had ordinal scores of 2 or 3. Workers were also categorized by smoking status (never, former, current smokers), pack-years of cigarette use [pack-years = (number of years smoked) × (number of packs [ie, 20 cigarettes] smoked per day), calculated as a time-dependent variable] and snuff use (never used, used ≤10 times per day, used >10 times per day, use unknown), based on information collected from the first health examination. When information on smoking and snuff use was not available, information from a later visit was used.

For this analysis, we linked the *Bygghälsan* computerized register of male participants (96% of all participants) to the Swedish National Cancer Registry through 2001 to identify first primary cancers of the oral cavity [International Classification of Diseases, seventh revision (ICD-7) codes 141–144], pharynx (ICD-7 145, 147, 148), and larynx (ICD-7 161). Less than 0.15% of the cohort members were lost to follow-up. Only cases of histologically determined squamous cell carcinoma (approximately 95% of all head and neck cancers) were included for this analysis. The person–years were computed for each cohort member from the date of the first health examination to the date of the cancer diagnosis, death, emigration or 31 December 2001, whichever occurred first. We used Poisson regression modeling with the software package EPICURE (26) to calculate rate ratios (RR) and 95% confidence intervals (95% CI) in relation to each cancer type to ever–never exposure to each workplace agent, adjusted for attained age (<50, 50–59, 60–69, ≥70 years), and smoking status (nonsmoker, former smoker, current smoker). Additional analyses of occupational exposures by exposure level (no exposure, moderate exposure, high exposure) were also performed, and models adjusting for all occupational exposures simultaneously and for pack-years of smoking were also fit. We tested for multiplicative interaction between occupational exposures and smoking variables (current smoker versus never or former smoker, <10 versus ≥10 pack-years) using the likelihood ratio test, comparing models with and without parameters specifying each interaction of interest.

We repeated all of the analyses with office workers excluded from the cohort to assess whether our findings differed for a more socioeconomically homogeneous study population.

The study was approved by the local committee of ethics at the Umeå University and by the steering committee of the register.

Results

Occupational exposure data were available for 329 665 male workers. After exclusions due to missing information on smoking habits, 307 799 workers (93%) remained eligible for the analysis. The six most common occupations were wood worker (21% of all workers), concrete worker (12% of all workers), electrician (11% of all workers), foreman (9% of all workers), plumber

(8% of all workers), and painter (8% of all workers); other occupations were present in the cohort at a frequency of 5% or less. The study population contributed 6 471 211 person–years of observation and 510 incident cases of squamous-cell head and neck cancers to this study. Of these cancers, 171 occurred in the oral cavity, 112 in the pharynx and 227 in the larynx.

After adjustment for age and smoking status (table 1), asbestos exposure was associated with a 50% increase in head and neck cancer, the strongest associations being for laryngeal cancer. Mineral wool exposure was also associated with laryngeal cancer, and cement dust exposure was associated with pharyngeal cancer. No occupational exposures were significantly associated with cancer of the oral cavity. The findings for asbestos, mineral wool, and cement dust did not show an exposure–response relationship although the numbers of cases among the hightly exposed workers were small. These findings did not materially change upon adjustment for age only or upon additional adjustment for pack-years of cigarette use (data not shown).

Generally speaking, the aforementioned associations did not change upon adjustment for all other occupational exposures (data not shown). An exception was found for asbestos and mineral wool exposures, which were highly correlated (Pearson correlation coefficient 0.80). After adjusting one for the other, we found that the laryngeal cancer risk associated with asbestos exposure was only slightly attenuated (RR 1.8, 95% CI 0.8–4.3), while the mineral wool association was no longer present (RR 1.1, 95% CI 0.5–2.3).

When we repeated our analyses excluding office workers from the cohort, our findings did not change (data not shown).

Table 1. Relative incidence of head and neck cancers in relation to occupational exposure (ever versus never) to workplace dusts.[a] (RR = rate ratio, 95% CI = 95% confidence interval)

Exposure	All sites			Oral cavity			Pharynx			Larynx		
	N	RR[b]	95% CI	N	RR	95% CI	N	RR	95% CI	N	RR	95% CI
Asbestos												
Never	477	1.0	·	161	1.0	·	107	1.0	·	209	1.0	·
Ever	33	**1.5**	**1.1–2.2**	10	1.3	0.7–2.6	6	1.0	0.4–2.5	18	**1.9**	**1.2–3.1**
Moderate	**29**	**1.8**	**1.3–2.6**	9	1.7	0.9–3.3	5	1.1	0.4–3.0	16	**2.3**	**1.4–3.8**
High	4	0.7	0.3–1.8	1	0.5	0.1–3.4	1	0.7	0.1–5.2	2	0.8	0.2–3.3
Mineral wool												
Never	464	1.0	·	156	1.0	·	105	1.0	·	203	1.0	·
Ever	46	1.3	1.0–1.8	15	1.3	0.7–2.1	7	0.9	0.4–1.9	24	**1.6**	**1.03–2.4**
Moderate	32	1.4	0.9–1.9	10	1.3	0.7–2.4	5	0.9	0.4–2.3	17	**1.7**	**1.01–2.7**
High	14	1.2	0.7–2.0	5	1.2	0.5–3.0	2	0.7	0.2–3.0	7	1.4	0.7–3.0
Cement dust												
Never	445	1.0	·	151	1.0	·	92	1.0	·	202	1.0	·
Ever	65	1.2	0.9–1.6	20	1.1	0.7–1.8	20	**1.9**	**1.2–3.1**	25	1.0	0.6–1.5
Moderate	52	1.2	0.9–1.6	18	1.3	0.8–2.1	16	**1.9**	**1.1–3.2**	18	0.9	0.5–1.4
High	13	1.2	0.7–2.0	2	1.2	0.5–3.0	4	1.9	0.7–5.0	7	1.3	0.6–2.7
Asphalt												
Never	498	1.0	·	167	1.0	·	108	1.0	·	223	1.0	·
Ever	12	1.2	0.7–2.1	4	1.2	0.4–3.2	4	1.8	0.7–4.9	4	0.9	0.3–2.4
Stone dust												
Never	377	1.0	·	126	1.0	·	87	1.0	·	164	1.0	·
Ever	133	1.1	0.9–1.4	45	1.2	0.8–1.6	25	0.9	0.6–1.5	63	1.2	0.9–1.6
Diesel fumes												
Never	419	1.0	·	142	1.0	·	95	1.0	·	182	1.0	·
Ever	91	1.1	0.9–1.4	29	1.1	0.7–1.6	17	0.9	0.6–1.6	45	1.2	0.9–1.7
Metal dust												
Never	458	1.0	·	151	1.0	·	105	1.0	·	202	1.0	·
Ever	52	1.1	0.8–1.5	20	1.3	0.8–2.1	7	0.6	0.3–1.4	25	1.2	0.8–1.8
Solvents												
Never	462	1.0	·	150	1.0	·	105	1.0	·	207	1.0	·
Ever	48	1.0	0.7–1.3	21	1.3	0.8–2.0	7	0.6	0.3–1.3	20	0.9	0.6–1.4
Wood dust												
Never	490	1.0	·	166	1.0	·	108	1.0	·	216	1.0	·
Ever	20	0.7	0.4–1.0	5	0.5	0.2–1.2	4	0.6	0.2–1.6	11	0.8	0.5–1.5

[a] Results in boldface are statistically significant (P<0.05).
[b] Adjusted for age, smoking status (never smoker, former smoker, current smoker), snuff use (never user, user ≤10 times/day, user >10 times/day, unknown).

Discussion

In this large cohort of male Swedish construction workers, we found an increased incidence of laryngeal cancer among asbestos-exposed workers and elevated pharyngeal cancer risks among workers exposed to cement dust.

Asbestos

Asbestos is the commercial name for naturally occurring silicate mineral fibers that were earlier added to many products used in the construction industry, including insulation and fire-proofing materials, cement, and wallboard. Asbestos inhalation causes mesothelioma and lung cancer; however, its role in laryngeal cancers is uncertain (8–11). Animal models are lacking, and epidemiologic evidence is inconsistent. Altogether 14 case–control studies (6 hospital-based, 8 population-based) have investigated the relationship between asbestos and laryngeal cancer. While nearly all of these studies found odds ratios greater than one (6, 12, 14, 27–37), the association was statistically significant in only three studies (12, 36, 37). The case–control design is generally well-suited to the study of rare diseases such as laryngeal cancer; however, the low prevalence of occupational asbestos exposure in the general population limits the statistical power of such studies, particularly when the sample size is small. Most of these studies were too small to detect, with reasonable power, a statistically significant association with asbestos of moderate magnitude; of the five studies that included at least 250 cases (6, 12, 28, 36, 37), three reported a statistically significant association with asbestos.

The findings from cohort studies of asbestos-exposed workers have also given inconsistent results. A pooled analysis of 27 cohort studies yielded a 30% excess risk of laryngeal cancer relative to the general population (meta-standardized mortality ratio 133, 95% CI 114–155) (38). When the authors used the mesothelioma mortality rate of each cohort as a surrogate for the severity of asbestos exposure, no dose–response relationship between exposure severity and laryngeal cancer was observed. However, misdiagnosis of mesothelioma may have attenuated a dose–response effect.

Our findings offer additional evidence supporting an association between asbestos and laryngeal cancer. The strength of our results as evidence of causation is limited by the fact that we did not observe a dose–response relationship with increasing asbestos exposure, although there were too few laryngeal cancers in the high-exposure group (N=2) to address this question adequately.

Cement dust

Cement is a powder composed mainly of lime (CaO, 60–67%), silica (SiO_2, 17–25%), alumina (Al_2O_3, 2–8%), and iron oxide (Fe_2O_3, 0–6%) that is combined with water and sand to create concrete and mortar. Cement dust may be carcinogenic due to the presence of hexavalent chromium, an established carcinogen (39), in some cements or due to the lime content, which, upon contact with oral mucosa, induces alkaline conditions potentially leading to the formation of reactive oxygen species (6, 40). Increased risks of lung, stomach, bladder, and colon cancer have been reported in some cohort studies of workers involved in the manufacture or handling of cement (41–43), and an increase in chromosomal aberrations among cement production workers has also been reported (44). In addition, in our present cohort, an increased risk for esophageal adenocarcinoma has been linked to cement exposure (45). Cement dust has also been associated with laryngeal cancer (6, 7, 28, 46) and pharyngeal cancer (47) in case–control studies.

In the past, asbestos was included in the manufacture of some types of cement to increase durability. Given the absence of an association between asbestos and pharyngeal cancer in our data, exposure to asbestos cement is an unlikely explanation for the observed association with cement dust.

Other exposures

We observed an association between mineral wool exposure and the incidence of laryngeal cancer. However, this finding disappeared upon adjustment for asbestos. Others have reported excess risks of laryngeal cancer accompanying exposure to wood dust (34, 48), diesel fumes (34), and metal dust (49). In our study no such relationships were identified. Regarding wood dust, it should be mentioned that exposure to dust in our cohort mainly came from pine, and dust from hardwood such as cedar, teak, and the like was very uncommon. Thus a negative result was expected. In addition, in our study, there were too few cases exposed to wood to detect moderate associations with reasonable statistical power.

Strengths and limitations

This is the largest cohort study investigating exposure to asbestos and cement dust and cancers of the head and neck. Furthermore, we were able to control directly for the potentially confounding effects of tobacco use (both snuff use and smoking), the major risk factor for these cancers. Use of a job-exposure matrix developed for this cohort to assess occupational exposure is another advantage, compared with previous studies in which exposure was self-reported or based on job titles alone. Nevertheless, exposure misclassification probably biased our results toward the null. Furthermore, the small numbers of workers with high-level exposure and the lack of

information on duration of exposure prevented us from conducting more-detailed analyses. It should be noted, however, that most of the workers remained in the same job; among the workers who participated in repeated examinations, 74% reported the same occupation. We were not able to adjust for alcohol consumption, another established risk factor for head and neck cancer. However, it is unlikely that alcohol use would vary in relation to asbestos and cement dust exposure within this socioeconomically homogeneous cohort.

In conclusion, we identified an increased incidence of laryngeal cancer among workers likely to have been exposed to asbestos; this result represents an important contribution to the ongoing debate as to whether asbestos increases the risk of laryngeal cancer. We also found an increased incidence of pharyngeal cancer accompanying exposure to cement dust. Our finding adds to the accumulating evidence suggesting increased risks of cancers at various sites, including the pharynx, among exposed workers. The relationships between asbestos, cement dust, and head and neck cancer risk warrant further investigation.

Acknowledgments

This research was supported by the Intramural Research Program of the National Institutes of Health, National Cancer Institute.

References

1. Parkin DM, Whelan SL, Ferlay J, Teppo L, Thomas DB. Cancer incidence in five continents, volume VIII. Lyon: IARC Press; 2002.
2. Mucci L, Adami HO. Oral and pharyngeal cancer. In: Adami HO, Hunter D, Trichopoulos D, editors. Textbook of cancer epidemiology. New York (NY): Oxford University Press; 2002. p 115–36.
3. International Agency for Research on Cancer (IARC). Occupational exposures to mists and vapours from strong inorganic acids, and other industrial chemicals. Lyon (France): IARC Press; 1992. IARC monographs on the evaluation of carcinogenic risk to humans, vol 54.
4. Weiland SK, Straif K, Chambless L, Werner B, Mundt KA, Bucher A, et al. Workplace risk factors for cancer in the German rubber industry: part 1. mortality from respiratory cancers. Occup Environ Med. 1998;55:317–24.
5. Kogevinas M, Sala M, Boffetta P, Kazerouni N, Kromhout H, Hoar-Zahm S. Cancer risk in the rubber industry: a review of the recent epidemiological evidence. Occup Environ Med. 1998;55:1–12.
6. Dietz A, Ramroth H, Urban T, Ahrens W, Becher H. Exposure to cement dust, related occupational groups and laryngeal cancer risk: results of a population based case-control study. Int J Cancer. 2004;108:907–11.
7. Maier H, Gewelke U, Dietz A, Heller WD. Risk factors of cancer of the larynx: results of the Heidelberg case-control study. Otolaryngol Head Neck Surg. 1992;107:577–82.
8. Edelman DA. Laryngeal cancer and occupational exposure to asbestos. Int Arch Occup Environ Health. 1989;61:223–227.
9. Kraus T, Drexler H, Weber A, Raithel HJ. The association of occupational asbestos dust exposure and laryngeal carcinoma. Isr J Med Sci. 1995;31:540–8.
10. Browne K, Gee JB. Asbestos exposure and laryngeal cancer. Ann Occup Hyg. 2000;44:239–250.
11. Griffiths H, Molony NC. Does asbestos cause laryngeal cancer? Clin Otolaryngol Allied Sci. 2003;28:177–82.
12. Gustavsson P, Jakobsson R, Johansson H, Lewin F, Norell S, Rutkvist LE. Occupational exposures and squamous cell carcinoma of the oral cavity, pharynx, larynx, and oesophagus: a case-control study in Sweden. Occup Environ Med. 1998;55:393–400.
13. Merletti F, Boffetta P, Ferro G, Pisani P, Terracini B. Occupation and cancer of the oral cavity or oropharynx in Turin, Italy. Scand J Work Environ Health. 1991;17:248–54.
14. Wortley P, Vaughan TL, Davis S, Morgan MS, Thomas DB. A case-control study of occupational risk factors for laryngeal cancer. Br J Ind Med. 1992;49:837–44.
15. Robinson C, Stern F, Halperin W, Venable H, Petersen M, Frazier T, et al. Assessment of mortality in the construction industry in the United States, 1984–1986. Am J Ind Med. 1995;28:49–70.
16. Wang E, Dement JM, Lipscomb H. Mortality among North Carolina construction workers, 1988–1994. Appl Occup Environ Hyg. 1999;14:45–58.
17. Maier H, Tisch M, Dietz A, Conradt C. Arbeiter in der Bauindustrie: eine Hochstrisikogruppe für Krebsterkrankungen des oberen Atmungs- und Verdauungstraktes? [Construction workers as an extreme risk group for head and neck cancer?]. HNO. 1999;47:730–6.
18. Elci OC, Dosemeci M, Blair A. Occupation and the risk of laryngeal cancer in Turkey. Scand J Work Environ Health. 2001;27:233–9.
19. Boffetta P, Richiardi L, Berrino F, Esteve J, Pisani P, Crosignani P, et al. Occupation and larynx and hypopharynx cancer: an international case-control study in France, Italy, Spain, and Switzerland. Cancer Causes Control. 2003;14:203–12.
20. Huebner WW, Schoenberg JB, Kelsey JL, Wilcox HB, McLaughlin JK, Greenberg RS, et al. Oral and pharyngeal cancer and occupation: a case-control study. Epidemiology. 1992;3:300–9.
21. Dong W, Vaughan P, Sullivan K, Fletcher T. Mortality study of construction workers in the UK. Int J Epidemiol. 1995; 24:750–7.
22. Nyrén O, Bergström R, Nyström L, Engholm G, Ekbom A, Adami HO, et al. Smoking and colorectal cancer: a 20-year follow-up study of Swedish construction workers. J Natl Cancer Inst. 1996;88:1302–7.
23. Hakansson N, Floderus B, Gustavsson P, Feychting M, Hallin N. Occupational sunlight exposure and cancer incidence among Swedish construction workers. Epidemiology. 2001;12:552–7.
24. Bergdahl IA, Toren K, Eriksson K, Hedlund U, Nilsson T, Flodin R, et al. Increased mortality in COPD among construction workers exposed to inorganic dust. Eur Respir J. 2004;23:402–6.
25. Anonymous. Miljöbeskrivning av sysselsättningar inom byggbranschen [Environmental descriptions of occupations with-

in the construction industry]. Stockholm: Bygghälsan; 1977.
26. Preston DL, Lubin JH, Pierce DA. EPICURE user's guide. Seattle (WA): Hirosoft; 1992.
27. Ahrens W, Jockel KH, Patzak W, Elsner G. Alcohol, smoking, and occupational factors in cancer of the larynx: a case-control study. Am J Ind Med. 1991;20:477–93.
28. Olsen J, Sabroe S. Occupational causes of laryngeal cancer. J Epidemiol Community Health. 1984;38:117–21.
29. De Stefani E, Boffetta P, Oreggia F, Ronco A, Kogevinas M, Mendilaharsu M. Occupation and the risk of laryngeal cancer in Uruguay. Am J Ind Med. 1998;33:537–42.
30. Zheng W, Blot WJ, Shu XO, Gao YT, Ji BT, Ziegler RG, et al. Diet and other risk factors for laryngeal cancer in Shanghai, China. Am J Epidemiol. 1992;136:178–91.
31. Maier H, Tisch M. Epidemiology of laryngeal cancer: results of the Heidelberg case-control study. Acta Otolaryngol Suppl. 1997;527:160–4.
32. Brown LM, Mason TJ, Pickle LW, Stewart PA, Buffler PA, Burau K, et al. Occupational risk factors for laryngeal cancer on the Texas Gulf Coast. Cancer Res. 1988;48:1960–4.
33. Zagraniski RT, Kelsey JL, Walter SD. Occupational risk factors for laryngeal carcinoma: Connecticut, 1975–1980. Am J Epidemiol. 1986;124:67–76.
34. Muscat JE, Wynder EL. Tobacco, alcohol, asbestos, and occupational risk factors for laryngeal cancer. Cancer. 1992;69:2244–51.
35. Imbernon E, Goldberg M, Bonenfant S, Chevalier A, Guenel P, Vatre R, et al. Occupational respiratory cancer and exposure to asbestos: a case-control study in a cohort of workers in the electricity and gas industry. Am J Ind Med. 1995;28:339–52.
36. Marchand JL, Luce D, Leclerc A, Goldberg P, Orlowski E, Bugel I, et al. Laryngeal and hypopharyngeal cancer and occupational exposure to asbestos and man-made vitreous fibers: results of a case-control study. Am J Ind Med. 2000;37:581–9.
37. Berrino F, Richiardi L, Boffetta P, Esteve J, Belletti I, Raymond L, et al. Occupation and larynx and hypopharynx cancer: a job-exposure matrix approach in an international case-control study in France, Italy, Spain and Switzerland. Cancer Causes Control. 2003;14:213–23.
38. Goodman M, Morgan RW, Ray R, Malloy CD, Zhao K. Cancer in asbestos-exposed occupational cohorts: a meta-analysis. Cancer Causes Control. 1999;10:453–65.
39. International Agency for Research on Cancer. Chromium, nickel and welding. Lyon (France): IARC; 1990. IARC monographs on the evaluation of carcinogenic risk to humans, vol 49.
40. Nair UJ, Obe G, Friesen M, Goldberg MT, Bartsch H. Role of lime in the generation of reactive oxygen species from betel-quid ingredients. Environ Health Perspect. 1992;98:203–5.
41. Smailyte G, Kurtinaitis J, Andersen A. Mortality and cancer incidence among Lithuanian cement producing workers. Occup Environ Med. 2004;61:529–34.
42. Jakobsson K, Horstmann V, Welinder H. Mortality and cancer morbidity among cement workers. Br J Ind Med. 1993; 50:264–72.
43. Knutsson A, Damber L, Järvholm B. Cancers in concrete workers: results of a cohort study of 33,668 workers. Occup Environ Med. 2000;57:264–7.
44. Fatima SK, Prabhavathi PA, Padmavathi P, Reddy PP. Analysis of chromosomal aberrations in men occupationally exposed to cement dust. Mutat Res Gen Toxicol Environ Mutagenesis. 2001;490:179–86.
45. Jansson C, Johansson ALV, Bergdahl IA, Dickman PW, Plato N, Adami J, et al. Occupational exposures and risk of esophageal adenocarcinoma and gastric cardia cancers among male Swedish construction workers. Cancer Causes Control. 2005;16:755–64.
46. Cauvin JM, Guenel P, Luce D, Brugere J, Leclerc A. Occupational exposure and head and neck carcinoma. Clin Otolaryngol Allied Sci. 1990;15:439–45.
47. Maier H, Fischer G, Sennewald E, Heller WD. Berufliche Risikofaktoren für Rachenkrebs: Ergebnisse der Heidelberger Rachenkrebsstudie [Occupational risk factors for pharyngeal cancer. Results of the Heidelberg Pharyngeal Cancer Study]. HNO. 1994;42:530–40.
48. Pollan M, Lopez-Abente G. Wood-related occupations and laryngeal cancer. Cancer Detect Prev. 1995;19:250–257.
49. Sokic SI, Adanja BJ, Marinkovic JP, Vlajinac HD. Case-control study of risk factors in laryngeal cancer. Neoplasma. 1994;41:43–7.

Received for publication: 1 November 2005