

Cancer and Construction:

What Occupational Histories in a Canadian Community Reveal

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From 2000 to 2002, male patients at a Canadian cancer treatment center with new-incident head-and-neck or esophageal cancers were invited to participate in a population-based study. The study population included 87 cases and 172 controls. A lifetime-history questionnaire was administered. Odds ratios (ORs) were calculated for occupational groups with a minimum of five cases, adjusted for duration of employment, age, smoking, alcohol, education, and income. A significantly increased risk was shown for construction workers (OR = 2.20; 95% CI 1.25–3.91). This investigation of a set of rare cancers over a limited time period demonstrates the feasibility of this research approach. The increased risk among construction workers supports the need for more comprehensive study of exposures in this occupational group. *Key words:* laryngeal, head-and-neck cancers; esophageal cancers; occupational histories; construction industry; population-based research.

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In Western industrial nations, it is estimated that 4–10% of all head-and-neck cancer cases are caused by occupational exposures.¹ At increased risk are those engaged in some blue-collar occupations, including woodworkers, textile industry workers, and coal

miners²; cooks, waiters, and those in alcohol-related occupations (includes esophageal cancer)^{3,4}; and construction workers.^{2,5–7} Exposures associated with head-and-neck cancers include dust, organic or inorganic agents,⁸ including iron dust, asbestos cement, and coal tar products, paint,⁷ welding fumes,⁹ polycyclic aromatic hydrocarbons (PAHs), cement dust, metal dusts, varnish, and lacquer.⁹ Risk for esophageal cancer has been shown to be elevated for workers exposed to PAHs;¹⁰ for brick makers,¹¹ woodworkers, for workers in finance, insurance, and real estate,¹² administrative support and health service workers,^{12,13} workers in food, beverage and tobacco industries, those in rubber and automotive building industries, waiters;¹⁴ and workers in occupations with exposures to chemical solvents or detergents,¹⁵ to silica dust,¹⁶ and numerous other agents, including sulfuric acid and carbon black.¹⁷ Cancer of the gastric cardia, adjacent to the esophagus, has been shown to be elevated among transportation workers, carpenters, and furniture industry workers.¹² Nasal cancers have been shown to be elevated among metal, foundry, textile, leather, and wood workers.¹⁸ The links between metalworking fluid (MWF) exposures and esophageal, laryngeal, and other head-and-neck cancers have been amply demonstrated.^{19–42}

Construction workers, as a broad occupational group, are potentially exposed to asbestos, wood dust, various oils, man-made mineral fibers (MMMFs), welding fumes, lead, organic solvents, silica, isocyanates, diesel exhaust, concrete dust, and asphalt vapors.⁴³

Since the early 1980s, occupational health research on construction workers in Canada has focused largely on asbestos-related diseases.⁴⁴ Asbestos exposure among construction workers has been documented over the years in such Canadian provinces as British Columbia⁴⁵ and Ontario.^{43,46–48} In the 1980s, drywall workers in Alberta were found to be exposed to asbestos dust at levels as high as 19 fibers per cubic centimeter (f/cc).⁴⁹

Links between occupational exposure to wood dust and stomach cancer were reported in the mid 1980s in British Columbia.⁵⁰ Exposures to a wide range of inorganic dusts have been researched, and some lung cancers were found to be associated with exposures to silica, excavation dust, and concrete dust,⁵¹ which can contain hexavalent chromium, an established human

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lung carcinogen.⁵² Dust-exposed workers such as bricklayers have shown increased risks, not only for pneumoconiosis and lung cancer from silica, but also for stomach cancer.⁵³ The potential problems of construction workers' exposures to diesel exhaust have also been flagged.⁵⁴ Other cancers may be associated with construction work; a recent Canadian study of occupational risk factors for brain cancer, for example, found an increased risk among construction workers.⁵⁵ A study examining cancers among Ontario pipefitters found significantly elevated risks for lung, esophageal, hematologic, and lymphatic malignancies.⁵⁶

In Ontario, exposures to chemical and biological agents are controlled through provincial regulations. These regulations apply to industrial settings, offices, governmental agencies, schools, hospitals, and mines. Construction, however, with the exception of asbestos and silica, is not governed by any occupational exposure limits.⁵⁷

METHODS

This hypothesis-generating case-control study took place in Windsor-Essex County in Ontario, Canada. Approval was obtained from the research ethics committees of both the Windsor Regional Cancer Centre and the University of Windsor, and the study was conducted following approved guidelines. Over a two-and-a-half-year period (January 1, 2000, to May 31, 2002), all male cancer patients at the Windsor Regional Cancer Centre (WRCC) diagnosed with histologically confirmed new-onset head-and-neck or esophageal cancers were invited to participate in a population-based case-control study. Only those who were current residents of Windsor-Essex County were included in the study.

In order to achieve an adequate sample size, patients with head-and-neck or esophageal cancers, which share similar exposure pathways, were included in the study. The final dataset included new-onset cases ICD-9⁵⁸ coded in patient referral records as cancers of the larynx/larynx glottis (161.00), larynx supraglottis (161.1), larynx subglottis (161.2), larynx in-situ (231.0), submandible (143.1), nasal cavity (160.0), epiglottis (161.2), pharynx (149.0), pharynx bucca (145.0), tongue base (141.9), tongue base dorsal surface (141.0), tongue lateral border (141.2), tongue ventral (141.3), tongue unspecified (141.1), floor of mouth (144.0), retromolar trigone (145.6), piriform sinus (148.1), oropharynx (146.7), nasopharynx (147.2), tonsil (146.0), esophagus (150.3, 150.4, 150.5, 150.8, 151.0), and neck unspecified.

After an in-service training session to explain the purpose of the study and to identify the work instructions applicable to each department, cancer center staff participated in patient recruitment. The medical records department screened all new male head-and-neck cancer and all esophageal cancer patients to confirm pathology and date of diagnosis and then for-

warded a letter outlining the study to each eligible incoming patient. A follow-up telephone call was made by project staff to each patient to schedule an interview at the patient's convenience. During the course of the study, 107 patients were contacted; 12 patients were excluded because their residences were outside the study area or their original diagnoses had been made prior to the study time frame; and two patients declined participation, representing a positive response rate of 98%. Thus, 87 cases were included in the final dataset.

Community controls from the same geographic area as the cases were chosen at random using city directory software⁵⁹ and were recruited by letter and a scripted follow-up telephone call. Of 831 letters sent to randomly selected community controls, 52 were returned as undeliverable; 174 potential controls were excluded because they did not fit within the sampling frame (age, geographic location, prior cancer history). Follow-up phone calls were made to recipients of introductory letters until the targeted number of controls had been interviewed. Of 250 eligible males contacted by telephone, there were 78 refusals, representing a positive response rate of 69%. After exclusions, 172 eligible male controls were included in the final dataset. Controls were recruited and interviewed during the same time period as the cases (2000-2002).

A number of measures were employed to minimize bias. The interviewers were trained to avoid influencing responses. They had no prior education or experience in regard to occupational health or industrial hygiene. As naïve interviewers, they were unlikely to unintentionally influence responses with prior knowledge of potential industrial exposures.

No surrogates (e.g., friends, co-workers, family members) were used to provide work histories in cases where the subject was unavailable (due to death or incapacitation). It is well documented that the use of surrogates in occupational history data gathering can result in a reduction in the quality of data when compared with subjects.⁶⁰

To minimize selection bias among cases, all head-and-neck and esophageal cancer cases referred to the cancer center were invited to participate. Community-based controls were randomly selected from the same population from which the cases arose and for the same time period. The recruitment letter for the community controls did not indicate that this was an occupational cancer study, in order to reduce bias. Occupational and industrial coding of jobs was carried out through a committee process blind to case-control status of the data provided by the interviewers.

Interviews took place between July 26, 2000, and December 13, 2002, and included cases diagnosed from January 1, 2000. All subjects, cases and controls, signed informed consents, and each was given a \$20 stipend. Subjects were informed that their participation was completely voluntary and that they had the

TABLE 1 National Occupational Classification (NOC) Groupings of Study Subjects

Cases (n = 87)	Controls (n = 172)	Occupational Grouping	NOC Codes Included in Grouping
34	61	Agriculture	6234, 8251, 8254, 8431, 8432, 8611, 8253
25	53	Vehicle and transportation equipment assembly, millwrights and mechanics	9482, 7312, 7321, 7315, 7211, 7311
25	27	Construction and related trades	7251, 7252, 7371, 7282, 7217, 7241, 7271, 7272, 7291, 7261, 7281, 7295, 0711, 0712, 7283, 7264, 7281, 7214, 7611, 7621, 7284, 2264, 7293, 7421, 9414
13	19	Food and beverage service, hospitality and casino	0631, 6452, 6453, 6641, 6642, 6443, 6212, 0632, 6241, 6242, 6251, 6252, 6451
16	31	Metal fabrication, machinists, tool and die	7231, 9511, 7232, 9612, 9514, 9516
14	27	Manufacturing managers, utility managers	0912, 0911, 2141
13	41	Retail/wholesale	0611, 0621, 6211, 6411, 6421, 6611, 6622, 6623
20	44	White collar, clerical, financial, office, professional	0012, 0111, 0112, 0121, 1221, 0122, 0414, 0411, 0123, 1111, 1113, 1211, 1224, 1228, 1231, 1241, 4163, 1112, 1221, 1222, 1223, 1225, 1232, 1233, 1242, 1243, 1244, 1423, 1411, 1412, 1413, 1414, 1422, 1431, 1454, 1432, 1433, 1453, 1441, 1442, 1443, 6434, 6435, 6221, 1474, 6672, 6231, 6232, 6431, 6216, 2131, 0811, 0712, 0211, 0651, 2151, 4112, 4211, 2253, 2251, 4155, 4152, 4212, 4154, 4217, 0314, 4151, 4153, 0011, 0412, 4162, 0413, 4168, 4163, 4165, 4166, 4169, 1452, 0512, 5121, 5123, 5124, 5125

right to withdraw themselves and/or their data from the project at any time. To protect participants' confidentiality, study codes with no personal identifiers were included in the database.

A comprehensive lifetime-history questionnaire was administered to each subject by trained interviewers, which included the project coordinator and graduate students. Interviews were conducted either in an office at the cancer center, at another location convenient for the patient, or by telephone. On average, the interviews were completed within 30 to 45 minutes. However, interview length depended on the number of residences and jobs the participant had had.

The questionnaire gathered data regarding known or suspected risk factors along with a complete occupational history of all jobs ever worked. Data were gathered regarding marital status, income, education, family cancer history, residential history, and occupational history, including age at the start and end of each job. Jobs were categorized using National Occupational Classification (NOC)⁶¹ codes and the North American Industrial Classification System (NAICS).⁶² NOC codes were used in the analysis rather than NAICS codes to provide a better indication of potential exposure. The NOC codes used in the analysis are shown in Table 1. Because there were relatively few subjects within each of the specific NOC categories, they were grouped within similar or related occupations to provide adequate statistical power.

The statistical program SPSS (version 10) was used to conduct a three-step multivariate analysis. The calculation of descriptive statistics among cases and controls, including stratification and adjustment, employed logistic regression to calculate adjusted odds ratios (ORs) and 95% confidence intervals.⁶³ The logistic model regressed the key dependent variable (case or control) on the key independent variables (occupational groupings), adjusting for confounders. Only occupational groupings with a minimum of five cases and five controls were included; each case or control had worked for a total duration of at least five years within the occupational grouping. OR calculations were based on the mean duration for each occupational grouping.

The model included the following covariates, using the forward conditional method (see Table 2): age as a categorical variable; total cigarette smoking pack-years calculated from data regarding respondents' age at commencement of smoking, age at cessation of smoking (or current age if still smoking), and average number of cigarettes smoked per day⁶⁴; alcohol consumption based on data regarding average number of drinks consumed during a given time period (the alcohol quantification did not include the number of years of alcohol consumption)—cigarette smoking and alcohol consumption are both established risk factors for head-and-neck cancers⁶⁵; income level as a categorical variable; and education as a categorical variable.

RESULTS

The all-male study population dataset included 65 head-and-neck and 22 esophageal cancer cases and 172 community controls (see Table 3). A significantly increased risk was shown for workers in construction (OR = 2.20; 95% CI 1.25–3.91). The following odds ratios were calculated for agriculture (OR = 1.47; 95% CI 0.93–2.30); food, beverage, and hospitality service (OR = 1.41; 95% CI 0.85–2.90); metal fabricators, machinists, and tool and die makers (OR = 1.11; 95% CI 0.65–1.92); vehicle and transportation equipment assembly, millwrights, and mechanical repair (OR = 1.08; 95% CI 0.66–1.73); manufacturing and utilities managers (OR = 1.32; 95% CI 0.61–2.81); white collar, office workers (OR = 0.96; 95% CI 0.54–1.73); retail and wholesale workers (OR = 0.92; 95% CI 0.56–1.53).

When the 22 esophageal cancers were excluded from the analysis, the study power was further limited. When results were obtained for the remaining 65 head-and-neck cancer cases and 172 controls, a significantly increased risk remained for workers in construction (OR = 2.28; 95% CI 1.29–4.00). Odds ratios calculated for the remaining occupational groupings were: agriculture (OR = 1.36; 95% CI 0.86–2.23); food, beverage, and hospitality service (OR = 1.18; 95% CI 0.70–1.98); metal fabricators, machinists, and tool and die makers (OR = 1.08; 95% CI 0.50–1.94); vehicle and transportation equipment assembly, millwrights, and mechanical repair (OR = 1.16; 95% CI 0.71–1.90); manufacturing and utilities managers (OR = 1.16; 95% CI 0.43–3.17); white collar office workers (OR = 0.78; 95% CI 0.38–1.59); retail and wholesale workers (OR = 1.04; 95% CI 0.63–1.74).

A significantly increased risk for male head-and-neck cancers was shown for heavy cigarette smoking history, defined as 20 or more pack years (OR = 4.19; 95% CI 1.63–10.80). Heavy alcohol use, defined as more than one drink per day, increased the risk for male head-and-neck cancer (OR = 6.31; 95% CI 1.54–25.86), albeit with a wide confidence interval.

DISCUSSION

The statistically significant occupational association found for employment in construction is consistent with risks established in prior scientific literature.^{9,38,66} The increased risks shown for heavy cigarette smoking and alcohol consumption are also in keeping with the known risk factors for head-and-neck cancers,^{67–69} thereby adding validity to the overall findings.

There is value in such population-based case-control studies in that they tend to be generalizable to the population as a whole.⁷⁰ However, there were limitations to this study: it was small for a community-based case-control study;⁷¹ it did not control for marijuana smoking⁷² or environmental tobacco smoke exposure⁷³; recall bias

TABLE 2 Descriptive Profile of Male Head-and-neck/Esophageal Cancer Cases and Community Controls

	Number in Analysis (Total 259)	
	Cases (n = 87)	Controls (n = 172)
Current age (mean)	65 years	58 years
Age categories		
> 40 years	2 (2%)	19 (11%)
41–50 years	7 (8%)	30 (17%)
51–60 years	18 (21%)	53 (31%)
61–70 years	29 (33%)	37 (22%)
> 70 years	31 (36%)	33 (19%)
Household income		
< \$40,000	21 (24%)	29 (17%)
≥ \$40,000	66 (76%)	143 (83%)
Education		
Less than high school	34 (39%)	41 (24%)
Completed high school or higher	53 (61%)	131 (76%)
Alcohol consumption		
Never used alcohol	3 (3%)	19 (11%)
1 drink per month to 1 drink per day	47 (54%)	112 (65%)
More than 1 drink per day	37 (43%)	41 (24%)
Cigarette smoking		
≤ 1 pack-year	8 (9%)	45 (26%)
< 20 pack years (but > 1)	8 (9%)	49 (28%)
≥ 20 pack years	71 (82%)	78 (45%)

may have affected the quality of self-reported data regarding jobs held in the past; the study used occupational groupings as a surrogate for exposure (misclassification of exposure is likely when subjects with less exposure are aggregated with the more highly exposed—non-differential misclassification of exposure may have decreased the probability of detecting associations^{63,74}); the use of industry or job titles and self-reported exposures has not been found to be sufficiently accurate to adequately evaluate likely exposures.^{75–77}

Carcinogens exist in many occupational environments.^{65,78–92} More specificity in regard to exposures would provide a clearer understanding of causality. A more accurate assessment of exposures and dose could be accomplished with the use of open-ended occupational questions and an expert panel to evaluate data in relation to existing knowledge of occupational exposures along with any additional information provided by hygiene reports and other available sources.^{71,75,93–105} However, while it is without question of significant value to identify through research the risks associated with specific exposures, nonetheless the identification of risks associated with occupational groups can provide important knowledge in itself. It is not always the case that a specific exposure in a particular work environment is the sole cause of a disease. While many carcinogens are independently sufficient to cause disease, co-carcinogens or incomplete carcinogens may require

TABLE 3 Results of Analysis by Occupational Group (Based on Mean Duration)*

	OR	95% CI		Mean Duration (Years)
		Lower	Upper	
Occupation				
Agriculture	1.47	0.93	2.30	25.61
White collar, clerical, financial, office	0.96	0.54	1.73	18.48
Manufacturing and utilities managers	1.32	0.61	2.81	21.16
Food and beverage, hospitality, casino	1.41	0.85	2.34	12.38
Construction	2.20	1.25	3.91	17.51
Vehicle assembly, mechanics, millwrights	1.08	0.66	1.73	15.57
Metal fabricators, machinists, tool and die	1.11	0.65	1.92	14.87
Retail, wholesale	0.92	0.56	1.53	13.46
Age (years)				
41–50	1.19	0.19	7.52	
51–60	1.73	0.32	9.46	
61–70	4.95	0.93	26.34	
≥ 70	3.94	0.71	21.50	
Cigarette smoking				
< 20 pack years (but > 1)	0.90	0.27	2.96	
≥ 20 pack years	4.19	1.63	10.80	
Alcohol use				
1 drink per month to 1 drink per day	3.07	0.78	12.03	
More than 1 drink per day	6.31	1.54	25.86	

*Odds ratios (ORs) were calculated for occupational groups with a minimum of five cases and five controls adjusted for duration of employment, age, smoking, alcohol, education, and income.

combinations of factors.¹⁰⁶ To narrow causality down to a specific agent in a workplace where multiple carcinogenic exposures may occur would require an accurate record of all agents used in the work process over at least a 30-year period (considering latency).

In many ways, our level of scientific knowledge regarding the interactions of the thousands of potential chemical agents and conditions found in industrial workplaces is in its infancy. In Ontario, for example, occupational cancer research has been constrained by the limitations of occupational histories obtained from death certificates or records indicating only predominant occupation.^{107,108} While laboratory-based research can identify isolated carcinogenic agents, a population-based study, such as the research described herein, has the advantage of being able to evaluate interactive and cumulative effects in a real-world environment. Institutional prevention efforts should focus more attention on potential occupational causes of cancer.¹⁰⁹ Our understanding of cancer causality would be enhanced by the adoption of occupational data collection on a system-wide basis by provincial cancer centers and by conducting focused case-control studies to research specific exposures and cancers. Such information has the potential to advance our understanding of cancer etiology in a practical manner and may ultimately facilitate the formulation of public health interventions.^{110,111} Moreover, Ontario and other jurisdictions need to enact regulations to specifically protect construction workers from exposures to carcinogenic agents as well as other chemical and biological hazards.

CONCLUSION

This preliminary population-based investigation of a set of relatively rare cancers over a limited time period has demonstrated the feasibility of this approach. The finding of a statistically significant increased risk among construction workers supports the need for more comprehensive study of an occupational group that has received relatively little attention.

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References

1. Maier H, Tisch M. Occupation and cancer of the head-neck area. *HNO*. 1999;47:1025-37.
2. Haguenoer JM, Cordier S, Morel C, Lefebvre JL, Hemon D. Occupational risk factors for upper respiratory and upper digestive cancers. *Br J Ind Med*. 1990;47:380-3.
3. Kjaerheim K, Andersen A. Incidence of cancer among male waiters and cooks: two Norwegian cohorts. *Cancer Causes Control*. 1993;4:419-26.
4. Herity B, Moriarity M, Bourke GJ, Daly L. A case-control study of head-and-neck cancer in the Republic of Ireland. *Br J Cancer*. 1981;43:1977-82.
5. Maier H, Tisch M, Dietz A, Conradt C. Construction workers as an extreme risk group for head-and-neck cancer? *HNO*. 1999; 47:730-6.
6. Stern FB, Ruder AM, Chen GC. Proportionate mortality among unionized roofers and waterproofers. *Am J Ind Med*. 2000; 37:478-92.
7. Maier H, Fischer G, Sennewald E, Heller WD. Occupational risk factors for pharyngeal cancer. Results of the Heidelberg Pharyngeal Cancer Study. *HNO*. 1994;42:530-40.
8. Maier H, de Vries N, Weidauer H. Occupation of the oral cavity, pharynx and larynx. *HNO*. 1990;38: 271-8.

9. 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.
10. Maceir H, Tisch P. Epidemiology of laryngeal cancer: results of the Heidelberg Pharyngeal Cancer Study. *Acta Otolaryngol.* 1997;527 suppl:160-4.
11. Xu Z, Pan GW, Liu LM, et al. Cancer risks among iron and steel workers in Anshan, China, Part I: Proportional mortality ratio analysis. *Am J Ind Med.* 1995;30:1-6.
12. Engel LS, Vaughan TL, Gammon MD, et al. Occupation and risk of esophageal and gastric cardia adenocarcinoma. *Am J Ind Med.* 2002;42:11-22.
13. Ward MH, Dosemeci M, Cocco P. Mortality from gastric cardia and lower esophagus cancer and occupation. *J Occup Med.* 1994;36:1222-7.
14. Chow WH, McLaughlin JK, Malfer HS, Linet MS, Weiner JA, Stone BJ. Esophageal cancer and occupation in a cohort of Swedish men. *Am J Ind Med.* 1995;27:749-57.
15. Cucino C, Sonnenberg A. Occupational mortality from squamous cell carcinoma of the esophagus in the United States during 1991-1996. *Dis Dis Sci.* 2002;47:568-72.
16. Pan G, Takahashi K, Feng Y, Liu L, et al. Nested case-control study of esophageal cancer in relation to occupational exposure to silica and other dusts. *Am J Ind Med.* 1999;35:272-80.
17. Parent ME, Siemiatycki J, Fritschi L. Workplace exposures and oesophageal cancer. *Occup Environ Med.* 2002;57:325-34.
18. Comba P, Barberi PG, Battista G, et al. Cancer of the nose and paranasal sinuses in the metal industry: a case control study. *Br J Ind Med.* 1992;49:193-6.
19. Occupational Disease Panel, Toronto. Ontario. Workers' Compensation Board. Report to the Workers' Compensation Board on the health effects of occupational exposure to fluids used for machining and lubricating metal in manufacturing: cancer of the esophagus. Government Reports Announcements & Index (GRA&I), Issue 06. 1997.
20. Flanders WD, Cann CI, Rothman KJ, Fried MP. Work-related risk factors for laryngeal cancer. *Am J Epidemiol.* 1984;119:23-32.
21. Olsen J, Sabroe S. Occupational causes of laryngeal cancer. *J Epidemiol Community Health.* 1984;38:117-21.
22. Vena JE, Sultz HA, Feidler RC, Barnes RE. Mortality of workers in an automobile engine and parts manufacturing complex. *Br J Ind Med.* 1985;42:85-93.
23. Mallin K, Berkeley L, Young Q. A proportional mortality study of workers in a construction equipment and diesel engine manufacturing plant. *Am J Ind Med.* 1986;10:127-41.
24. Zargraniski RT, Kelsey JL, Walter SD. Occupational risk factors for laryngeal carcinoma: Connecticut, 1975-1980. *Am J Epidemiol.* 1986;124:67-76.
25. Brown LM, Mason TJ, Pickle LW, et al. Occupational risk factors for laryngeal cancer on the Texas Gulf Coast. *Cancer Res.* 1988; 48:1960-4.
26. Tola S, Kalliomaki PL, Pukkala E, Asp S, Korkala ML. Incidence of cancer among welders, platers, machinists, and pipe fitters in shipyards and machine shops. *Br J Ind Med.* 1988;45:209-18.
27. Bravo MP, Espinosa J, Calero JR. Occupational risk factors for cancer of the larynx in Spain. *Neoplasm.* 1990; 37:477-81.
28. Haguenoer JM, Cordier S, Morel C, Lefebvre JL, Hemon D. Occupational risk factors for upper respiratory tract and upper digestive tract cancers. *Br J Ind Med.* 1990;47:380-3.
29. 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.
30. Eisen EA, Tolbert PE, Monson RR, Smith TJ. Mortality studies of machining fluid exposure in the automobile industry. I: A standardized mortality ratio analysis. *Am J Ind Med.* 1992; 22:809-24.
31. Tolbert PE, Eisen EA, Pothier LJ, Monson RR, Hallock MF, Smith TJ. Mortality studies of machining-fluid exposure in the automobile industry. II: Risks associated with specific fluid types. *Scand J Work Environ Health.* 1992;18:351-60.
32. 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.
33. Zheng W, Blot WJ, Shu X, et al. Diet and other risk factors for laryngeal cancer in Shanghai, China. *Am J Epidemiol.* 1992; 136:178-91.
34. Delzell E, Macalusa M, Honda Y, Austin H. Mortality patterns among men in the motor vehicle manufacturing industry. *Am J Ind Med.* 1993;24:471-84.
35. Eisen EA, Tolbert PE, Hallock MF, Monson RR, Smith TJ, Woskie SR. Mortality studies of machining fluid exposure in the automobile industry. III: A case-control study of larynx cancer. *Am J Ind Med.* 1994;26:185-202.
36. Park RM, Mirer FE. A survey of mortality at two automobile engine manufacturing plants. *Am J Ind Med.* 1996; 30:664-73.
37. Goldberg P, Leclerc A, Luce D, Morcet JF, BrugSre J. Laryngeal and hypopharyngeal cancer and occupation: results of a case-control study. *Occup Environ Med.* 1997;54:477-82.
38. 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.
39. National Institute for Occupational Safety and Health (NIOSH). Occupational Exposure to Metalworking Fluids. DHHS (NIOSH) Publication No. 98-102. 1998.
40. Eisen EA, Bardin J, Gore R, Woskie SR, Hallock MF, Monson RR. Exposure-response models based on extended follow-up of a cohort mortality study in the automobile industry. *Scand J Work Environ Health.* 2000;27:240-9.
41. Tolbert PE. Oils and cancer. *Cancer Causes Control.* 1997; 8:386-405.
42. Calvert GM, Ward E, Schnorr TM, Fine LJ. Cancer risks among workers exposed to metalworking fluids: a systematic review. *Am J Ind Med.* 1998;33(3):282-92.
43. Verma DK, Kurtz LA, Sahai D, Finkelstein MM. Current chemical exposure among Ontario construction workers. *Appl Occup Environ Hyg.* 2003;18(10):131-47.
44. Churg A. Malignant mesothelioma in British Columbia in 1982. *Cancer.* 1985;55:672-4.
45. Teschke K, Morgan MS, Checkoway H, et al. Mesothelioma surveillance to local sources of exposure to asbestos. *Can J Public Health.* 1997;88:163-8.
46. Gennaro V, Finkelstein MM, Ceppi M, et al. Mesothelioma and lung tumors attributable to asbestos among petroleum workers. *Am J Ind Med.* 2000;37:275-82.
47. Keith MM, Brophy JT. Identification of work-related asbestos disease in a Canadian community. *Ann NY Acad Sci.* 2006; in press.
48. Keith M and Brophy JT. Participatory mapping of occupational hazards, disease, and injury among asbestos-exposed workers from a foundry and insulation complex in Southwestern Ontario, Canada. *Int J Occup Environ Health.* 2004;10: 144-53.
49. Verma DK, Middleton CP. Occupational exposure to asbestos in the drywall taping process. *Am Ind Hyg Assoc J.* 1980; 41:264-9.
50. Gallagher RP, Threlfall WJ, Band PR, Spinelli JJ. Cancer mortality experience of woodworkers, loggers, fishermen, farmers and miners in British Columbia. *Natl Cancer Inst Monograph.* 1985; 69:163-7.
51. Siemiatycki J, Dewar R, Lakhani R, Nadon L, Richardson L, Gerin M. Cancer risk associated with 120 inorganic dusts: results from a case-control study in Montreal. *Am J Ind Med.* 1989; 16:547-67.
52. U.S. Department of Labor. Chemical Sampling Information: Chromium (VI) (Hexavalent Chromium). 2006.
53. Finkelstein MM, Verma DK. Mortality among Ontario members of the International Union of Bricklayers and Allied Craftworkers. *Am J Ind Med.* 2005;47:4-9.
54. Verma DK, Finkelstein MM, Kurtz L, Smolyneec E, Eyre S. Diesel exhaust exposure in the Canadian railroad work environment. *Appl Occup Environ Hyg.* 2003;18:25-34.
55. Pan SY, Ugnat AM, Mao Y. Occupational risk factors for brain cancer in Canada. *J Occup Environ Med.* 2005;47:704-17.
56. Finkelstein MM, Verma DK. A cohort study of mortality among Ontario pipe trades workers. *Occup Environ Med.* 2004;61:736-42.
57. Ontario Ministry of Labour. Control of Exposure to Biological or Chemical Agents, R.R.O. 1990, Reg. 833: <<http://www.canlii.org/on/laws/regu/1990r.833/20060412/whole.html>>.
58. Percy C, Van Holten V, Muir C. International Classification of Diseases for Oncology. 2nd ed. Geneva, Switzerland: World Health Organization, 1990.

59. Polk City Directory Infotyme Software for Leamington, Windsor, Windsor Suburban, Ontario Multi-Dimensional Intelligence. Southfield, MI: R.L. Polk, 1999.
60. Hennenburger PK. Collection of occupational epidemiologic data. *Occupational epidemiology*. *Occup Med*. 1996;11:393-401.
61. Human Resources Development Canada. National Occupational Classification. Ottawa, Canada, 1992.
62. Statistics Canada. North American Industrial Classification System. Ottawa, Canada, 1998.
63. Checkoway H, Pearce N Crawford-Brown D. *Research Methods in Occupational Epidemiology*. New York: Oxford Press, 1989.
64. Rothman KJ, Greenland S. *Modern Epidemiology*. 2nd ed. New York: Lippincott, Williams & Wilkins, 1998.
65. Tomatis L, Aitio A, Day NNE, Heseltine E, et al. *Cancer: Causes, Occurrence and Control*. Lyon: IARC Scientific Publications, No. 100. 1990:170.
66. Stern FB, Ruder AM, Chen GC. Proportionate mortality among unionized roofers and waterproofers. *Am J Ind Med*. 2000; 37:478-92.
67. Sankaranarayanan R, Masuyer E, Swaminathan R, Ferlay J, Whelan S. Head-and-neck cancer: a global perspective on epidemiology and prognosis. *Anticancer Res*. 1998;18(6B):4779-86.
68. Johnson N. Tobacco use and oral cancer: a global perspective. *J Dent Educ*. 2001;328-39.
69. Lewin F, Norell SE, Johansson H, et al. Oncologic Center, Karolinska Hospital, Stockholm, Sweden. Smoking tobacco, oral snuff, and alcohol in the etiology of squamous cell carcinoma of the head-and-neck: a population-based case-referent study in Sweden. *Cancer*. 1998;82:1367-75.
70. Blair A, Hayes B, Stewart P, Zahm SH. Occupational epidemiologic study design and application. *Occupational epidemiology*. *Occup Med*. 1996;11:403-19.
71. Siemiatycki J. Future etiologic research in occupational cancer. *Environ Health Perspect*. 1995;103 suppl 8:209-16.
72. Zhang ZF, Morgenstern H, Spitz MR, et al. Marijuana use and increased risk of squamous cell carcinoma of the head-and-neck. *Cancer Epidemiol Biomarkers Prev*. 1999;8:1071-8.
73. Zhang ZF, Morgenstern H, Spitz MR, et al. Environmental tobacco smoking, mutagen sensitivity, and head-and-neck squamous cell carcinoma. *Cancer Epidemiol Biomarkers Prev*. 2000; 9:1043-9.
74. Blair A, Linos A, Stewart PA, et al. Evaluation of risks for non-Hodgkin's lymphoma by occupation and industry exposures from a case-control study. *Am J Ind Med*. 1993; 23:301-12.
75. Siemiatycki J, Fritschi L, Nadon L, Gerin M. Reliability of an expert rating procedure for retrospective assessment of occupational exposures in community-based case-control studies. *Am J Ind Med*. 1997;31:280-6.
76. Teschke K, Olshan AF, Daniels JL, et al. Occupational exposure assessment in case-control studies: opportunities for improvement. *Occup Environ Med*. 2002;59:575-94.
77. Fritschi L, Siemiatycki J, Richardson L. Self-assessed versus expert-assessed occupational exposures. *Am J Epidemiol*. 1996; 144:521-7.
78. Nicholson WJ. Quantitative Estimates of Cancer in the Workplace. *Am J Ind Med*. 1984;5:341-2.
79. International Agency for Research on Cancer (IARC). *Monographs on the Evaluation of Carcinogenic Risks of Chemicals to Humans*. Lyon, France: IARC, 1987.
80. Kraut A. Estimates of the extent of morbidity and mortality due to occupational diseases in Canada. *Am J Ind Med*. 1994;25: 267-78.
81. Infante P. Cancer and blue-collar workers: who cares? *New Solutions*. 1995;5(2):52-7.
82. Ward E. Overview of preventable industrial causes of occupational cancer. *Environ Health Perspect*. 1995;103 suppl 8:197-203.
83. Steenland K, Loomis D, Shy C, Simonsen N. Review of occupational lung carcinogens. *Am J Ind Med*. 1996;29:474-90.
84. Stellman JM and Stellman SD. *Cancer and the workplace*. *CA Cancer J Clin*. 1996; 46:70-92.
85. Epstein S. Winning the war against cancer? Are they even fighting it? *The Ecologist*. 1998;28(2):69-80.
86. Epstein S. *Politics of Cancer Revisited*. New York: East Ridge Press, 1998.
87. Herbert R, Landrigan P. Work-related death: a continuing epidemic. *Am J Pub Health*. 2000 90:541-54.
88. Dupre JS. Report of the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario. Ontario Ministry of the Attorney General. Toronto, ON: Queens Printer for Ontario, 1984.
89. Miller A. Recommendations for the Primary Prevention of Cancer. Report of the Ontario Task Force on the Primary Prevention of Cancer, 1995.
90. Toronto Cancer Prevention Coalition. *Preventing Occupational and Environmental Cancer: A Strategy for Toronto*. Toronto, ON, 2001.
91. Ham J. Report of the Royal Commission on the Health and Safety of Workers in Mines: Ontario Royal Commission on the Health and Safety of Workers in Mines. Ministry of the Attorney General, Toronto, ON, 1976.
92. Aronson K, Howe G, Carpenter M, Fair M. Surveillance of potential associations between occupations and causes of death in Canada, 1965-91. *Occup Environ Med*. 1999;56:265-9.
93. McGuire V, Nelson LM, Koepsell TD, Checkoway H, Longstreth WT. Assessment of occupational exposure in community-case-control studies. *Annu Rev Public Health*. 1998;19:35-53.
94. Benke G, Sim M, Fritschi L, Aldred G, Forbes A, Kauppinen T. Comparison of occupational exposure using three different methods: hygiene panel, job exposure matrix (JEM), and self reports. *Appl Occup Environ Hyg*. 2001;16:84-91.
95. Sieber WK, Sundin DS, Frazier TM, Robinson CF. Development, use, and availability of a job exposure matrix based on national occupational hazard survey data. *Am J Ind Med*. 1991;20:163-74.
96. Kauppinen T, Toikkanen J, Pukkala E. From cross-tabulations to multipurpose exposure information systems: a new job-exposure matrix. *Am J Ind Med*. 1998;33:409-17.
97. Gilks W, Richardson S. Analysis of disease risks using ancillary risk factors, with application to job-exposure matrices. *Stat Med*. 1992;11:1443-63.
98. Goldberg M, Kromhout H, Guenel P, et al. Job exposure matrices in industry. *Int J Epidemiol*. 1993;22:S10-S15.
99. Kauppinen TP, Mutanen PO, Seitsamo JT. Magnitude of misclassification bias when using a job-exposure matrix. *Scand J Work Environ Health*. 1992;18:105-12.
100. Kauppinen TP. Assessment of exposure in occupational epidemiology. *Scand J Work Environ Health*. 1994;20(special issue):19-29.
101. Weston TL, Aronson KJ, Siemiatycki J, Howe GR, Nadon L. Cancer mortality among males in relation to exposures assessed through a job-exposure matrix. *Int J Occup Environ Health*. 2000;6:194-202.
102. Plato N, Steineck G. Methodology and utility of a job-exposure matrix. *Am J Ind Med*. 1993;23:491-502.
103. Dosemeci M, Cocco P, Gomez M, Stewart PA, Heineman EF. Effects of three features of a job-exposure matrix on risk estimates. *Epidemiology*. 1994; 5:124-7.
104. Messing K, Dumais L, Courville J, Seifert AM, Boucher M. Evaluation of exposure data from men and women with the same job title. *J Occup Med*. 1994; 36:913-7.
105. Siemiatycki J, Dewar R, Richardson L. Costs and statistical power associated with five methods of collecting occupation exposure information for population-based case-control studies. *Am J Epidemiol*. 1989;130:1236-46.
106. Goldsmith DF. Importance of causation for interpreting occupational epidemiology Research: A case of quartz and cancer. *Occupational epidemiology*. *Occup Med*. 1996;11:433-49.
107. Marrett LD and Weir E. Occupation and cancer in Ontario: review of the options for establishing a cancer-occupation data base for Ontario. An occasional paper for the Industrial Disease Standards Panel, 1989.
108. Carpenter DO, Arcaro K, Spink DC. Understanding the human health effects of chemical mixtures. *Environ Health Perspect*. 2002;110 suppl 1:25-42.
109. Clapp RW, Howe GK, Jacobs M. Environmental and occupational causes of cancer re-visited. *J Public Health Policy*. 2006; 27:61-76 .
110. Doll R, Peto R. *Avoidable causes of cancer*. Oxford, U.K.: Oxford University Press, 1981.
111. Firth M, Brophy J, Keith M. *Workplace Roulette: Gambling with Cancer*. Toronto, On, Canada: Between the Lines, 1997.