Sinonasal Cancer, Occupation, and Tobacco Smoking in European Women and Men

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Background: *In this analysis of European case-control studies on sinonasal cancer, we examined the risk for occupation and smoking, by gender and histological type.*
Methods: *The pooled data included 104 female and 451 male cases, and 241 female and 1,464 male controls. Lifetime smoking and occupational history were recoded following uniform criteria, and job-exposure matrices were applied for wood and leather dust.*
Results: *Wood dust exposure was associated with an excess risk in men (OR = 2.36, 95% CI 1.75–3.2) but not in women (OR = 1.17, 95% CI 0.31–4.47). Exposure to leather dust was associated with an excess risk in both genders. Both wood and leather dust were associated with adenocarcinomas rather than squamous cell carcinomas. Excess risks for smoking were higher for squamous cell carcinomas and higher in men than in women.*
Conclusions: *In these European populations, occupation was associated with about 11% of all sinonasal cancers in women and 39% in men. This difference can, in part, be attributed to variation in exposure patterns between genders.* Am. J. Ind. Med. 36:101–107, 1999.

KEY WORDS: sinonasal cancer; occupational diseases; case-control; wood dust; tobacco; gender

INTRODUCTION

Since the 1960s, a large excess risk for sinonasal cancer (SNC), particularly adenocarcinomas, has been repeatedly documented among workers in wood-related occupations [IARC, 1995; Hayes et al., 1986; Vaughan et al., 1989; Demers et al., 1995]. A high risk for SNC has also been identified among workers in the leather industry, workers exposed to formaldehyde, workers in nickel refining, and those exposed to hexavalent chromium compounds [Roush, 1996]. Besides these exposures, elevated risks for SNC were found in case-control studies for a variety of other occupations [Leclerc et al., 1997]. Most studies, however, lacked the power to demonstrate statistically significant associations of this rare disease with specific jobs.

Few other risk factors have been identified for SNC, apart from occupational exposures. A low excess risk has been associated in some studies with cigarette smoking [Zeng et al., 1993; Fukuda and Shibata, 1988], but results are

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TABLE I. Description of the Case-Control Studies on Sinonasal Cancer Included in the Pooled Analysis

Study center [reference]	Number cases/controls		Year of interview	Year of diagnosis	(%) Adeno-carcinoma/ squamous
	Men	Women			
Italy					
Verona/Vicenza/Siena, [Comba et al., 1992b]	55/184	23/70	1987–88	1982–87	21/44
Brescia, [Comba et al., 1992a]	23/70	11/32	1990	1980–89	18/52
Biella, [Magnani et al., 1993]	22/92	4/19	1988–89	1976–88	42/42
Vigevano, [Merler et al., 1986]	16/29	5/10	1983	1968–82	55/15
France					
[Luce et al., 1992]	167/320	40/89	1986–88	1986–88	42/37
Netherlands					
[Hayes et al., 1986]	91/195	0/0	1982	1978–81	25/55
Germany					
[Bolm-Audorff et al., 1989]	33/33	21/21	1983–85	1983–85	7/37
Sweden					
[Hardell et al., 1982]	44/541	0/0	1979	1970–79	7/71
All	451/1464	104/241	1979–90	1970–89	29/44

not consistent [Roush, 1996]. Radiation exposure, viral infections, and constitutional factors have been associated with the occurrence of this cancer [Roush, 1996], but the evidence is either inconsistent or indicates that only a small proportion of all SNCs may be attributed to them.

Studies that provided results by histology, mostly distinguishing squamous cell carcinoma (SCC) and adenocarcinoma (AC), indicate that some of the identified risk factors for SNC are related with specific histological types. In a number of individual studies and in a pooled analysis of case-control studies [Demers et al., 1995], the higher risk for woodworkers was specifically associated with sinonasal AC. The increased risks for smoking has mostly been found for SCCs and, in some cases, with undifferentiated carcinomas.

In this article, we provide estimates of the attributable risk of SNC for occupational exposures and smoking by gender, and evaluate the differential pattern of the risk by histology. Results of the individual studies [Comba et al., 1992a,b; Magnani et al., 1993; Merler et al., 1986; Luce et al., 1992; Leclerc et al., 1994; Hayes et al., 1986; Bolm-Audorff et al., 1989; Hardell et al., 1982] as well as selected results from a pooled dataset, including extra-European studies particularly on wood dust exposure [Demers et al., 1995] and exposure in other occupations [Leclerc et al., 1997] have been published.

SUBJECTS AND METHODS

The pooled study incorporates data from four studies in Italy and one each from The Netherlands, France, Germany, and Sweden (Table I). All the studies were selected on the basis of the availability of detailed information on occupational exposures. A detailed description of the process for selection of the studies and the classification of exposures can be found elsewhere [Demers et al., 1995; Leclerc et al., 1997]. The pooled dataset includes 555 cases (451 men, 104 women) and 1,705 controls (1,464 men, 241 women). The cases included 241 SCCs (200 men, 41 women), 160 ACs (149 men, 11 women), 146 of other histology (98 men, 48 women), and 8 of unknown histology. The studies differed concerning the methods for recruitment and interview of the subjects and the vital status of subjects. Subjects were diagnosed between 1970 to 1989 and were interviewed between 1979 and 1990. The case-control ratio ranged from 1 to 12.3, with an overall ratio of 3.1.

Detailed information on lifetime occupational history was collected in all studies included in the pooled analysis. Subjects reported up to 21 occupations held until date of interview, as well as the industries and periods connected to these jobs. The original occupational codes were recoded into standard occupational [ILO, 1969], and industry [ISIC rev. 2 codes, Statistical Office of the United Nations, 1968] coding systems. Subjects in wood- and leather-related occupations or industries were classified as to their level of wood dust and leather dust exposure using a job-exposure matrix (JEM) developed by one of the investigators [Demers et al., 1995]. Formaldehyde exposure was examined through a JEM developed by two of the authors (DB, MG). Industrial hygiene data were used to develop semiquantitative exposure categories for wood dust (nonexposed, 0.01–1 mg/m^3, 1–5 mg/m^3, and >5 mg/m^3). Each unique occupation/industry combination was classified without knowledge of the disease status of the individuals who held the job.

A list of other high-risk occupations/industries was constructed on the basis of results of previous studies, as proposed by Leclerc et al. [1997]. This list includes miners and quarrymen, textile industry workers, cooks, bakers and food industry workers, plumbers and pipe fitters, welders and flame cutters, painters, motor-vehicle drivers, construction workers, and agriculture and livestock production workers [for a complete list with occupation and industry codes, see Leclerc et al., 1997]. A lifetime smoking history was available in all studies and subjects were classified by smoking into non-, ex-, or current smokers.

Statistical Analysis

Unconditional logistic regression was applied for the pooled analysis. Odds ratios and 95% confidence intervals for occupational exposures were adjusted for age (10-year categories), gender (when applicable), study center, and smoking status (when applicable), as well as for the remaining occupational exposures examined. The heterogeneity of effects between study centers was visually examined through Galbraith plots [Galbraith, 1988] and checked in the logistic regression models by testing the significance of the interaction between exposure and center, comparing the deviance of the model with and without the interaction terms. In cases with substantial heterogeneity, logistic regression with mixed effects models was applied [Breslow and Clayton, 1993]. These models are similar to the fixed effects models, but they include as random effects in the linear component of the model the variables, study center, and the exposure effect by study. In this model it is assumed that the effect parameters of each study are randomly distributed around an overall value. Parameters derived from these models have a larger variance than those derived from the more widely used, fixed effects models. We calculated adjusted estimates of the attributable risk [Bruzzi et al., 1985] using a developed software for SAS [Mezzetti et al., 1996]. The attributable risk was calculated using the formula (AR = (proportion of cases exposed) * (OR-1)/OR), which requires knowledge of the OR and the distribution of risk exposure only among cases. AR was calculated subsequently for exposure to different levels of wood, leather dust, and the other specific compounds, and for ‘‘ever’’ having worked in a high-risk occupation or industry.

Mixed effects models were fitted using the GLIMMIX macro for SAS [Littell et al., 1996]. Analyses were done using Stata v5.0 and the SAS System.

TABLE II. Sinonasal Cancer Risk Related to Occupational Exposures by Gender

	$N_{(cases)}$/ $n_{(controls)}$	% Cases exposed	OR	95% CI
Women				
Wood dust	4/9	4.0	1.17	(0.31–4.47)
Formaldehyde	15/41	15.0	0.83	(0.41–1.69)
Leather dust	7/7	7.0	2.71	(0.78–9.43)
Other a priori high risk occupations*	43/104	44.8	1.21	(0.69–2.12)
Men				
Wood dust	168/389	38.4	2.36	(1.75–3.20)
Formaldehyde	229/493	52.3	1.66	(1.27–2.17)
Leather dust	26/42	5.9	1.92	(1.10–3.35)
Other a priori high risk occupations*	165/659	61.1	1.10	(0.82–1.49)

*Excluding all subjects ever worked in a job with wood dust exposure.

RESULTS

Occupation by Gender

In women, an excess risk for SNC was observed for leather dust exposure (Table II), while risks for exposure to wood dust, formaldehyde, and other high-risk occupations were around the null value.

In men, exposure to wood dust was strongly associated with SNC risk (Table II). Analysis by exposure level [results not presented in table, see Demers et al., 1995], showed that excess risk was almost exclusively found among workers in the highest wood dust exposure category of levels higher than 5 mg/m^3 (OR = 6.69). These findings, however, reflect the situation in men. Among women, only four cases and nine controls were evaluated through the job exposure matrix as having been exposed to wood dust. Of the four cases, only one was considered as highly exposed to wood dust. Both leather dust and formaldehyde were associated with an excess risk, although ORs were lower than those observed for wood dust. The risk associated with the list of high-risk occupations and industries was around the null value (OR = 1.10).

Occupation by Histology

The excess risk observed for wood dust was particularly high for ACs, while there was no association between wood dust at any level of exposure and SCC (Table III). Similar to wood dust, exposure to formaldehyde was particularly associated with AC rather than SCCs. Leather dust exposure was principally associated with AC, while lower, statistically nonsignificant excess risk was seen for SCC (Table

TABLE III. Sinonasal Cancer Risk Related to Occupational Exposures by Histology Type

	$N_{(cases)}/n_{(controls)}$	% Cases exposed	OR	95% CI
Adenocarcinoma				
Wood dust	115/398	74.2	12.20	(7.43–20.0)
Formaldehyde	124/534	80.0	3.30	(1.98–5.49)
Leather dust	15/49	9.7	2.99	(1.33–6.73)
Other a priori high risk occupations*	15/763	37.5	0.51	(0.24–1.07)
Squamous cell carcinoma				
Wood dust	35/398	14.7	0.72	(0.47–1.12)
Formaldehyde	80/534	33.6	1.27	(0.92–1.74)
Leather dust	10/49	4.2	1.45	(0.70–2.99)
Other a priori high risk occupations*	133/763	65.5	1.45	(1.03–2.02)

*Excluding all subjects ever worked in a job with wood dust exposure.

TABLE IV. Sinonasal Cancer Risk Related to Smoking by Gender and Histology Type

	$N_{(cases)}/n_{(controls)}$		% Cases exposed	OR	95% CI
All					
Never smoker	160	578	29.7	1.00	
Ex-smoker	197	514	36.6	1.33	(1.00–1.78)
Current smoker	181	595	33.6	1.21	(0.91–1.63)
Women					
Never smoker	80	184	80.0	1.00	
Ex-smoker	11	23	11.0	1.45	(0.63–3.36)
Current smoker	9	28	9.0	0.78	(0.33–1.83)
Men					
Never smoker	80	394	18.3	1.00	
Ex-smoker	186	491	42.5	1.43	(1.03–1.97)
Current smoker	172	567	39.3	1.34	(0.97–1.87)
Squamous cell carcinoma					
Never smoker	66	578	27.7	1.00	
Ex-smoker	69	514	29.0	1.19	(0.78–1.81)
Current smoker	103	595	43.3	1.72	(1.16–2.56)
Adenocarcinoma					
Never smoker	40	578	25.8	1.00	
Ex-smoker	76	514	49.0	1.29	(0.75–2.22)
Current smoker	39	595	25.2	0.65	(0.35–1.20)

III). While no excess risk was associated with the list of high-risk occupations and industries for all SNCs, a statistically significant excess risk (OR = 1.45, 95% CI 1.03–2.02) was observed for SCC. A negative association was found for AC with employment in this group of occupations. In evaluating these occupations and industries, we excluded all subjects who had ever worked in occupations with possible wood dust exposure.

Smoking

A small excess risk among ex-smokers was observed in both men and women, while only male current smokers were at an excess risk (Table IV). Smoking was statistically significantly associated with a higher risk for SCC, while no statistically significant association was observed for AC. The risk for SCC was highest among current smokers (OR = 1.72, 95% CI 1.16–2.56).

Heterogeneity of Effects Between Centers

There was considerable heterogeneity in effects between the centers participating in the study, particularly for wood dust exposure (chi-square for heterogeneity = 20.74 on 5 degrees of freedom, *P*-value = 0.0009). The risk related to a high level of wood dust exposure was lowest for the study in Sweden (OR = 1.73) and highest for the French study (OR = 18.12), with ORs for the other centers being around 4. The heterogeneity of the relative risks for wood dust were evaluated by fitting mixed effects models. The ORs obtained for exposure to wood dust from a random (mixed) effects model (OR = 1.65, 95% CI 0.85–3.43) was lower and the confidence limits wider than those obtained for the fixed effects models (OR = 2.33, 95% CI 1.75–3.11). A similar pattern was observed for the other exposures examined, with OR being slightly lower and confidence intervals wider. Given the small differences in the estimates derived from both models, we present all the relative and attributable risk estimates on the basis of the more widely used fixed effects models.

Attributable Risk Estimates

The attributable risk for SNC in relation to occupation was 33% (95% CI = 12–89%). Considerable differences in attributable risk estimates were observed between men and women (Table V). In men, up to 39% (95% CI = 18–92%) of all SNC cases could be attributed to occupational exposure, while in women occupation explained only 11% (95% CI = 0.1–100%). This was mainly caused by the low prevalence of wood-related occupations in women (4% of female cases vs. 38% of male cases). Different patterns of the attributable risk were observed by histological type. For AC, occupational exposures were related to 77% (95% CI = 60–100%) of all cancers. A lower attributable risk for occupational exposures, 22% (95% CI = 3–100%), was observed for SCC, mainly due to exposures in other than wood- or leather-related occupations. Fifteen percent of all SNCs could be attributed to smoking, this proportion being

TABLE V. Attributable Risk for Sinonasal Cancer Related to Occupational Exposures and Smoking, by Gender and Histology Type

Exposure	AR (%) All	AR (%) Women	AR (%) Men	AR (%) Squamous cell carcinoma	AR (%) Adeno-carcinoma
Wood	18	1	22	−6	68
Leather	3	4	3	1	6
Other a priori high risk occupations[a]	8	8	6	20	−36
All occupational exposures[b]	33	11	39	22	77
Smoking	15	1	23	23	−3

[a]Excluding all subjects ever worked in a job with wood dust exposure.
[b]AR calculated using OR and prevalence for ever having worked in at least one risk occupation (those involving exposure to wood dust, leather dust, formaldehyde, and other a priori risk occupations as listed in Materials and Methods). This AR was not derived by adding up ARs of separate exposure groups.

TABLE VI. AR for Wood Dust, Leather Dust, and Formaldehyde for All Centers, Consecutively Omitting From the Pooled Results Each Study in Turn

	AR (%) Related to wood dust	AR (%) Related to leather dust
All	18	3
Without Germany	18	3
Without Verona/Vicenza/Siena	20	3
Without The Netherlands	20	3
Without France	5	5
Without Biella	18	3
Without Brescia	19	5
Without Sweden	21	7
Without Vigevano	19	1

higher for SCC (23%). The AR-related to smoking was higher in men (23%) than in women (1%).

The prevalence of exposure differed considerably between centers, particularly because some of the case-control studies were conducted selectively in areas with a high concentration of leather or wood industries. The overall attributable risks for wood dust and leather dust were therefore estimated excluding each participating center consecutively (Table VI). Attributable risks for wood dust varied considerably, depending on whether the study from France was included. Smaller variations were observed for attributable risk related to leather dust, where exclusion of the Swedish study and the study in Vigevano made the largest difference.

DISCUSSION

The findings of this study indicate that the two major risk factors for SNC are some occupations related to about 33% of all SNCs, and tobacco smoking related to about 15%. These proportions were clearly higher in men than in women. These results also indicate that in the case of SNC, specific exposures can be linked to morphology. Wood dust and, to a lesser extent, leather dust and formaldehyde were predominantly associated with ACs, a pattern which appears to be specific to this tumor.

Smoking and occupational exposures entailing a variety of exposures, including PAHs, asbestos, and other organic and inorganic fibers and dusts, metal fumes, solvents, and dyes, were primarily associated with sinonasal SCCs. For lung cancer, previous reports have shown an excess for SCCs and not AC, related to exposure to PAHs at work and from cigarette smoking [Zahm et al., 1989]. Individual studies on SNC had previously shown an exposure-cell-type specificity for wood dust and AC. The inconsistent results obtained in some earlier individual studies concerning the role of smoking and SNC risk are probably due to lack of an analysis by separate histologies in those studies.

The association between exposure to formaldehyde and SNC persisted even after adjusting for other occupational exposures, including wood and leather dust. However, when the risk for a potential confounder (particularly wood dust) is very sharp, it may not be possible to assess the separate effects of another agent. This is exemplified by the fact that out of 126 AC cases exposed to formaldehyde, 115 were also exposed to wood dust. Luce et al. [1993] found a higher risk among those exposed to both wood dust and formaldehyde than among those exposed to wood dust alone, suggesting that both exposures may be additive or multiplicative.

The percentage of adenocarcinomas in men was 3 to 4 times higher than in women. This difference, however, rather than being evaluated as a sex-specific response, is, in part, a reflection of the differences in exposure. The prevalence of exposure to wood dust was clearly higher in men (approximately 38%) compared to women (approximately 4%).

Furthermore, among those exposed a lower percentage of women were evaluated as being exposed to a high level (1 out of 4 female exposed cases) compared to 72% in men.

Pooling results of different studies conveys a considerable advantage by increasing the power of the study and also by verifying the presence of similar risks in different populations. There are, however, complexities both concerning the methodology to be used and also the interpretation of the findings of pooled studies. The observed differences between studies concerned mainly the magnitude of the risk, particularly for wood dust, rather than the pattern of the risk. Nearly all studies identified an increased risk for wood and leather dust for AC and an increased risk for SCC associated with smoking. There was variation, however, in the magnitude of the effect, which could be due to chance, differences in study design, differences in time period, differences in exposure patterns, confounding by other exposures, or genetic factors. The latter is probably an unlikely explanation for this specific tumor, since the study subjects were nearly exclusively Caucasian, and the inconsistencies were not smaller among geographically related centers, e.g., Italy, than among distant centers. Differences in exposure assessment were reduced in the pooled analysis by recoding all occupational information and by evaluating de novo exposure to specific agents and by using JEMs, providing semiquantitative estimates of exposure for specific agents. Other differences, however, in the design of individual studies, such as control selection, matching, or potential study-specific confounding effects, could only in part be taken into account in the pooled analysis. The application of random (mixed) effects models may control for random variation of the results between centers by appropriate modeling techniques. However, even in the case of substantial heterogeneity of the risk as found for wood dust, these models did not considerably modify the risk parameter, although they did result in wider confidence intervals.

The attributable risks were shown to differ considerably between centers, particularly because some of the case-control studies were conducted selectively in areas with a high concentration of leather or wood industries. This is because the attributable risk depends not only on the RR (which can be assumed to be biologically constant between European populations) but also by the prevalence and level of exposure. We repeated the calculations excluding these centers but for some centers this resulted in major changes in the overall estimates of the attributable risk. This occurred mainly in the AR calculations related to wood dust, where AR depended on whether or not France was included. This study is, however, population-based. It was not especially focused on a woodworkers population, as reflected in the proportion of controls exposed to wood dust, which is comparable with that of the other studies. The proportions of cases exposed to wood dust in this study is high, leading (together with the high OR) to a relatively high AR for the French study. In view of these findings, one has to consider heterogeneity of effects within Europe regarding wood dust exposure and SNC, possibly caused by differences in exposure.

In conclusion, the results of this pooled analysis indicate that exposure to wood and leather dust are associated predominantly with adenocarcinomas of the nose and sinuses, while tobacco smoke and other occupational exposures are associated with squamous cell carcinomas. Both occupational exposures and smoking were associated with higher excess risks in men than in women.

REFERENCES

Bolm-Audorff U, Vogel C, Woitowitz HJ. 1989. Berufliche und außerberufliche Risikofaktoren von Nasen-Rachentumoren. (Occupational and environmental risk factors of nasal and nasalpharyngeal cancer.) Staub-Reinhaltung der Luft 49:389–393.

Breslow EN, Clayton DG. 1993. Approximate inference in generalized linear mixed models. J Am Stat Assoc 88:9–25.

Bruzzi P, Green SB, Byar DP, Brinton LA, Schairer C. 1985. Estimating the population attributable risk for multiple risk factors using case-control data. Am J Epidemiol 122:904–914.

Comba P, Barbieri PG, Battista G, Belli S, Ponterio F, Zanetti D, Axelson O. 1992a. Cancer of the nose and paranasal sinuses in the metal industry: a case-control study. Br J Ind Med 49:193–196.

Comba P, Battista G, Belli S, de Capua B, Merler E, Orsi D, Rodella S, Vindigni C, Axelson O. 1992b. A case control study of cancer of the nose and paranasal sinuses and occupational exposures. Am J Ind Med 22: 511–520.

Demers PA, Kogevinas M, Boffetta P, Leclerc A, Luce D, Gérin M, Battista G, Belli S, Bolm-Audorf U, Brinton LA, Colin D, Comba P, Hardell L, Hayes RB, Magnani C, Merler E, Morcet J-F, Preston-Martin S, Matos E, Rodella S, Vaughan TL, Zheng W, Vainio H. 1995. Wood dust and sino-nasal cancer: pooled reanalysis of twelve case-control studies. Am J Ind Med 28:151–166.

Fukuda K, Shibata A. 1988. A case-control study of past history of nasal diseases and maxillary sinus cancer in Hokkaida, Japan. Cancer Res 48:1651–1652.

Galbraith RF. 1988. A note on graphical presentation estimated odds ratios from several clinical trials. Stat Med 7:889–894.

Hardell L, Johansson B, Axelson O. 1982. Epidemiological study of nasal and nasopharyngeal cancer and their relation to phenoxy acid or chlorophenol exposure. Am J Ind Med 3:247–257.

Hayes RB, Gerin M, Raatgever JW, de Bruyn A, Gerin M. 1986. Wood-related occupations, wood dust exposure, and sinonasal cancer. Am J Epidemiol 124:569–577.

IARC (International Agency for Research on Cancer) Working group on the Evaluation of Carcinogenic Risks to Humans. 1995. IARC monographs on the evaluation of carcinogenic risks to humans, vol. 62. Wood dust and formaldehyde. Lyon: WHO.

ILO (International Labour Office). 1969. International standard classification of occupations. Revised edition 1968. Geneva.

Leclerc A, Martinez Cortes M, Gérin M, Luce D, Brugere J. 1994. Sinonasal cancer and wood dust exposure: results from a case control study. Am J Epidemiol 140:340–349.

Leclerc A, Luce D, Demers PA, Boffetta P, Kogevinas M, Belli S, Bolm-Audorff U, Brinton LA, Colin D, Comba P, Gérin M, Hardell L, Hayes RB, Magnani C, Merler E, Morcet J-F, Preston-Martin S, Vaughan TL, Zheng W. 1997. Sinonasal cancer and occupation. Results from the reanalysis of twelve case-control studies. Am J Ind Med 31:153–165.

Littell CR, Milliken GA, Stroup WW, Wolfinger RD. 1996. SAS systems for mixed models. Cary, NC: SAS Institute.

Luce D, Leclerc A, Morcet JF, Casal-Lareo A, Gérin M, Brugere J, Haguenoer JM, Goldberg M. 1992. Occupational risk factors for sinonasal cancer: a case-control study in France. Am J Ind Med 21:163–175.

Luce D, Gérin M, Leclerc A, Morcet J-F, Brugère J, Goldberg M. 1993. Sinonasal cancer and occupational exposure to formaldehyde and other substances. Int J Cancer 53:224–231.

Magnani C, Comba P, Ferraris F, Ivaldi C, Menighin M, Terracini B. 1993. A case-control study of carcinomas of the nose and paranasal sinuses in the woolen textile manufacturing industry. Arch Environ Health 48:94–97.

Merler E, Baldasseroni A, Laria R, Faravelli P, Agostini R, Pisa R, Berrino F. 1986. On the causal association between exposure to leather dust and nasal cancer: further evidence from a case-control study. Br J Ind Med 43:769–774.

Mezzetti M, Ferraroni M, Decarli A, La Vecchia C, Benichou J. 1996. Software for attributable risk and confidence interval estimation in case-control studies. Comput Biomed Res 29:63–75.

Roush GC. 1996. Cancers of the nasal cavity and paranasal sinuses. In: Schottenfeld D, Fraumeni J Jr, editors. Cancer Epidemiology and prevention, second ed. New York: Oxford University Press. p 587–602.

Statistical Office of the United Nations. 1968. International standard industrial classification of all economic activities. Statistical papers series M, number 4, revision 2. New York: United Nations.

Vaughan TL. 1989. Occupation and squamous cell cancers of the pharynx and sinonasal cavity. Am J Ind Med 16:493–510.

Zahm SH, Brownson RC, Chang JC, Davis JR. 1989. Study of lung cancer histologic types, occupation, and smoking in Missouri. Am J Ind Med 15:565–578.

Zeng W, McLauhglin JK, Chow WH, Chien HT, Blot WJ. 1993. Risk factors for cancers of the nasal cavity and paranasal sinuses among white men in the United Sates. Am J Epidemiol 138:965–972.