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RECTAL CANCER AND OCCUPATIONAL RISK FACTORS: A HYPOTHESIS-GENERATING, EXPOSURE-BASED CASE-CONTROL STUDY

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In 1979, a hypothesis-generating, population-based case-control study was undertaken in Montreal, Canada, to explore the association between occupational exposure to 294 substances, 130 occupations and industries, and various cancers. Interviews were carried out with 3,630 histologically confirmed cancer cases, of whom 257 had rectal cancer, and with 533 population controls, to obtain detailed job history and data on potential confounders. The job history of each subject was evaluated by a team of chemists and hygienists and translated into occupational exposures. Logistic regression analyses adjusted for age, education, cigarette smoking, beer consumption, body mass index, and respondent status were performed using population controls and cancer controls, e.g., 1,295 subjects with cancers at sites other than the rectum, lung, colon, rectosigmoid junction, small intestine, and peritoneum. We present here the results based on cancer controls. The following substances showed some association with rectal cancer: rubber dust, rubber pyrolysis products, cotton dust, wool fibers, rayon fibers, a group of solvents (carbon tetrachloride, methylene chloride, trichloroethylene, acetone, aliphatic ketones, aliphatic esters, toluene, styrene), polychloroprene, glass fibers, formaldehyde, extenders, and ionizing radiation. The independent effect of many of these substances could not be disentangled as many were highly correlated with each other. *Int. J. Cancer* 87: 874–879, 2000.
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Although dietary and alcohol-consumption habits have been associated with rectal cancer risk (World Cancer Research Fund, 1997), these factors are probably not sufficient to explain the incidence gap between developed and developing countries as well as the systematically lower risk among women than among men (IARC, 1982, 1987, 1992, IARC, 1997). There must therefore be other, as yet unidentified risk factors for rectal cancer, and it is plausible that some of these are environmental/occupational risk factors. Nevertheless, the evidence on the role of occupational agents in rectal cancer etiology remains scant or non-existent.

In the 1980s, a large hypothesis-generating, population-based case-control study was undertaken in Montreal, Canada, to search for evidence for associations between several types of cancer and hundreds of occupational exposures. Starting from a list of 76 industries, 54 occupations, and 294 substances (Siemiatycki, 1991), the present report describes in-depth analyses of the association between rectal cancer and exposure to the subset of substances, occupations, and industries which have been reported as possible risk factors in previous studies or which appear to be related to rectal cancer.

MATERIAL AND METHODS

The design and data collection methods have been described elsewhere (Gérin *et al.*, 1985; Siemiatycki, 1991; Siemiatycki *et al.*, 1987; Fritschi and Siemiatycki, 1996). In brief, subjects were men, aged 35 to 70 years, residing in Montreal, who were diagnosed with a new, histologically confirmed cancer at 1 of 19 anatomical sites. Between 1979 and 1985, there were 4,576 eligible cancer patients, and 3,730 of these (82%) were successfully interviewed. Eighty-one percent of subjects responded for themselves; proxies provided information for the rest. Not all sites were included in all years of the study; rectal cancer was included in 5 of the 7 years.

Case and control series

There were 257 cases successfully interviewed out of the 304 eligible primary rectal cancer cases (84.5% response rate). Two groups of controls were available. The first group (referred to as cancer controls) included some of the patients with a cancer other than cancer of the rectum. Of the total pool of subjects, we excluded 3 groups: patients interviewed in years in which rectal cancer was not ascertained and interviewed, lung cancer cases, and cases with cancer of other intestinal sites (colon, rectosigmoid junction, small intestine, peritoneum). The remaining 1,295 cancer cases, constituting the group of cancer controls retained for analysis, were distributed by site as follows: esophagus (n = 65), stomach (n = 168), liver (n = 25), gallbladder (n = 20), pancreas (n = 75), pleura (n = 7), prostate (n = 227), testis (n = 19), penis (n = 7), bladder (n = 277), kidney (n = 112), melanoma of skin (n = 77), non-Hodgkin's lymphoma (n = 149), Hodgkin's lymphoma (n = 37), and other (n = 30). Cases and controls were drawn from the 19 major hospitals of the Montreal area, thereby assuring a good representation of the base population. A second control group consisted of 533 successfully interviewed (72% response rate), age-stratified, population-based controls, selected using electoral lists and random-digit dialing.

Exposure assessment

The interview questionnaire was in 2 parts: a structured section requesting information on important cancer risk factors, in particular lifestyle characteristics, and a semi-structured section designed to obtain a detailed description of each job the subject had held in his working lifetime (Gérin *et al.*, 1985; Gérin and Siemiatycki, 1991). For each job in each subject's history, questions were asked about the company, its products, the nature of the work site, the subject's main and subsidiary tasks, and any additional information (*e.g.*, equipment maintenance, use of protective equipment, activities of co-workers) that could furnish clues about possible exposures.

A team of chemists and industrial hygienists examined each completed questionnaire and translated each job into a list of potential exposures by means of a checklist that included 294 substances (Siemiatycki *et al.*, 1987; Siemiatycki, 1991). This type of retrospective exposure assessment had not been done previously in the context of a community-based study, and it took several years to develop a satisfactory methodology. The final codes given to a file were based on consensus among the chemists and indus-

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trial hygienists. Chemical coding was carried out completely blind with regard to the subject's disease status.

For each product thought to be present in each job, the coders noted 3 dimensions of information, each on a 3-point scale: degree of confidence that the exposure had actually occurred (possible, probable, definite), frequency of exposure in a normal working week (<5%, 5–30%, >30% of the time), and concentration to which the worker was exposed (low, medium, or high). Given the retrospective nature of the coding, we did not consider it possible to attribute more precise quantitative values to the exposure levels. Non-exposure was not interpreted as absolute 0; rather, it was considered as exposure up to the level that can be found in the general environment and to which all subjects may be exposed.

Occupational circumstances retained for analysis

The present in-depth analysis focused on occupational variables reported to be potential risk factors as well as on a subset of occupational circumstances earmarked from our database. Those retained on the basis of the database were selected by screening analyses looking at rectal cancer in relation to all 54 occupations, 76 industry titles, and 294 occupational substances (Siemiatycki, 1991). We retained those occupational variables which showed, using either population or cancer controls, some evidence of being associated with rectal cancer, *e.g.*, the odds ratio (OR) was at least 1.3 and the level of significance was ≤0.15 when adjusting for 6 literature-based *(a priori)* and data-based (significantly associated with rectal cancer) non-occupational potential confounders: age in years (3 levels), number of years of schooling (2 levels), respondent status (self *vs.* proxy), cigarette smoking as the average number of cigarettes smoked per day times the number of years smoked (3 levels), beer drinking as the average number of beers drank per day times the number of years of drinking (3 levels), body mass index calculated as weight (kg)/height $(m)^2$ (2 levels). We further excluded from analysis any occupational variable that had fewer than 5 subjects in any cell of the contingency (2 × 2) tables based on any exposure level or duration. As a result of these selection criteria, we retained for the present analysis 33 substances, 12 industries, and 12 occupations.

Statistical analyses

Exposure to each substance was analyzed first as a dichotomy of any exposure *vs.* no exposure and then as a dichotomy of substantial *vs.* no exposure. For any given substance, the unexposed group comprised subjects never exposed to that substance plus a small number who were considered possibly exposed or whose exposure occurred only in the 5 years preceding the interview. The substantially exposed group comprised those who the chemists were confident had been exposed (probable or definite exposure) and who had more than 5 years of exposure at medium or high frequency and concentration. Occupation and industry titles were analyzed first as a dichotomy of ever having been employed in the occupation/industry of interest *vs.* not employed and then as a dichotomy of employed for 10 years or more *vs.* not employed. The 5 years preceding the interview were not counted in the duration of exposure.

Occupational circumstances were first included one at a time in the unconditional logistic regression model along with the 6 potential non-occupational confounders. Substances showing evidence of an association with rectal cancer were then examined using regression models that took into account the presence of other occupational substances.

Two parallel sets of analyses were carried out using the 533 population controls and the 1,295 selected cancer controls. To simplify the presentation, we mainly show results based on cancer controls. This set of results was chosen because it constitutes a larger set than that of the population controls and, thus, provides more stable estimates; the response rate for rectal cancer cases was closer to that of cancer controls than to that of population controls, and we surmise that there is a greater opportunity for information bias when using population controls than when using cancer controls. Nevertheless, for substances showing the strongest evidence of an association with rectal cancer based on cancer controls, we also present risk estimates based on population controls.

RESULTS

Table I shows the distribution of selected non-occupational characteristics among rectal cancer cases, cancer controls, and population controls. There were no statistically significant differences for these characteristics between cases and either of the control groups. Population controls tended to be slightly older, to have smoked less, to have consumed less beer, and to have had fewer proxies respond for them than cancer controls.

Substances

Table II presents ORs between rectal cancer incidence and exposure to each of the 33 selected substances at any level of exposure and at the substantial level, using cancer controls. ORs are based on statistical models treating each substance independently and adjusted for the 6 non-occupational potential confounders only.

Among the various substances selected on the basis of previous publications, only rubber dust and rubber pyrolysis products showed any association with rectal cancer. Eighteen of the other substances selected for analysis had statistically elevated (or borderline significant) ORs in either the "ever-exposed" or the "substantially exposed" group. Exposures to cotton dust, wool fibers, and rayon fibers were associated with elevated risks. All of the solvents under analysis, *i.e.*, mononuclear aromatic hydrocarbons (MAHs), toluene, xylene, aliphatic ketones, carbon tetrachloride, aliphatic esters, methylene chloride, trichloroethylene, acetone, and styrene, also showed some excess risks. Other substances associated with elevated risks included formaldehyde, extenders, glass fibers, polychloroprene, and ionizing radiation. For all substances which had reasonable numbers exposed at the substantial level, ORs were higher among subjects substantially exposed than among subjects ever exposed.

Seven of the substances showing some excess risk in Table II had more than 20 exposed cases, and these were subjected to more in-depth analyses with respect to frequency, concentration, and duration of exposure. The dose-response relationships suggested in Table II for cotton dust and wool fibers were primarily due to increasing risk with increasing years of exposure. For aliphatic ketones, the increasing risk with increasing exposure level observed in Table II reflected dose-response trends in all 3 dimensions of exposure. The apparent dose-response relationships seen for MAHs and their constituents, toluene and xylene, appeared to be due primarily to increasing risk with increasing frequency and concentration of exposure. For formaldehyde, the overall dose-

TABLE I – SELECTED CHARACTERISTICS OF RECTAL CANCER CASES AND CONTROLS

Characteristics	Rectal cancer cases (n = 257)	Cancer controls (n = 1,295)	Population controls (n = 533)	P
Mean age (years)	58.7	58.4	59.6	<0.05
Mean number of years of schooling	9.4	9.8	10.1	>0.05
Proxy respondent (%)	15.2	19.7	12.6	<0.01
Mean cigarette-years smoked (cig/day × years)	801	893	802	<0.05
Mean beer-years consumed (beer/day × years)	47	60	33	<0.01
Mean body mass index (kg/m^2)	25.4	25.4	25.6	>0.05

[1]P value for test of the null hypothesis of equality of the 3 groups on this parameter.

TABLE II – OR AND 95% CI FOR THE RELATION BETWEEN RECTAL CANCER AND SELECTED SUBSTANCES, USING CANCER CONTROLS

Substances	Exposure level					
	Any			Substantial		
	N[1]	OR[2]	95% CI	N	OR	95% CI
Selected from the literature						
Metal						
Metallic dust	69	0.9	0.7–1.2	30	0.8	0.5–1.3
Iron compounds	63	0.9	0.7–1.3	23	0.7	0.4–1.1
Mild steel dust	46	1.0	0.7–1.5	17	0.7	0.4–1.2
Stainless steel dust	10	0.8	0.4–1.7	2	0.4	0.1–1.5
Iron dust	9	0.7	0.4–1.5	5	0.9	0.4–2.5
Rubber						
Rubber dust	12	1.7	0.9–3.4	2	1.8	0.4–9.0
Rubber pyrolysis products	10	3.0	1.4–6.5	1	1.7	0.2–17.3
Combustion products						
Soot	17	0.8	0.5–1.3	4	0.8	0.3–2.4
Coal	8	0.6	0.3–1.3	6	1.3	0.5–3.3
Wood	9	0.7	0.3–1.4	4	1.0	0.3–3.2
Others						
Asbestos (chrysotile)	30	0.7	0.5–1.0	3	0.5	0.2–1.6
Asbestos (amphiboles)	11	0.7	0.3–1.2	2	1.5	0.3–7.6
Ink	12	1.3	0.7–2.6	6	1.2	0.5–2.9
Added based on screening analyses						
Textile						
Fabric dust	28	1.0	0.7–1.6	19	1.3	0.8–2.2
Cotton dust	25	1.0	0.6–1.6	18	1.5	0.9–2.6
Wool fibers	26	1.6	1.0–2.6	19	1.9	1.1–3.3
Rayon fibers	10	1.6	0.8–3.2	7	2.6	1.0–6.7
Solvents						
MAHs[3]	101	1.2	0.9–1.6	34	1.6	1.0–2.5
Toluene	50	1.4	1.0–2.0	17	1.7	1.0–3.0
Xylene	39	1.3	0.9–1.9	7	2.9	1.1–7.3
Aliphatic ketones	21	1.4	0.8–2.3	15	2.4	1.3–4.6
Carbon tetrachloride	16	2.0	1.1–3.5	2	0.5	0.1–2.0
Aliphatic esters	12	1.4	0.7–2.6	10	3.0	1.4–6.8
Methylene chloride	7	1.2	0.5–2.8	5	3.8	1.1–12.9
Trichloroethylene	12	2.0	1.0–3.9	3	0.9	0.3–3.2
Acetone	11	2.3	1.1–4.7	8	4.8	1.8–13.0
Styrene	6	1.7	0.7–4.5	5	3.9	1.2–12.9
Others						
Formaldehyde	36	1.2	0.8–1.9	13	2.4	1.2–4.7
Extenders	20	1.4	0.8–2.3	8	3.6	1.4–9.2
Glass fibers	14	0.9	0.5–1.6	8	4.3	1.7–11.3
Hypochlorites	17	1.0	0.6–1.7	12	1.3	0.7–2.6
Polychloroprene	14	2.0	1.1–3.8	2	7.1	1.0–51.8
Ionizing radiation	6	3.8	1.3–11.4	0	—	—

[1]N, number of exposed cases.–[2]OR adjusted for age, education, respondent status, cigarette smoking, beer consumption, and body mass index.–[3]MAHs include all aromatic compounds that have only 1 benzene ring, including substituted products such as xylene, toluene, styrene, phenol, and ethyl benzene.

response pattern appeared to reflect increases by concentration and by duration.

The results in Table II ignore the possible confounding among occupational risk factors. Since there are no well-established occupational risk factors for rectal cancer, it is not clear that any other occupational variables should be included in any model for a given substance. However, if 1 or more of these substances is in fact a risk factor for rectal cancer, then the correlated exposures could lead to confounding and misleading inferences. In such a case, it might be appropriate to include the occupational risk factor in all models. Since the carcinogenicity of these substances is not established, the results in Table II, unadjusted for occupational variables, represent our best estimates of the relative risks for each substance. Still, it is of some interest to include multiple occupational variables in the same model, to see which variables seem the most susceptible to confounding by other occupational variables. To accomplish this, we selected a subset of variables in Table II, namely, those with a lower confidence limit of the OR $\geq$1.0 at the "any exposure" level. The 8 variables which satisfied this criteria were included, together with the standard 6 non-occupational co-variates, in a single model. This was done only at the "any exposure" level, and the results are shown in Table III. Among this set of variables, those that seem least affected by the presence of others in the model are rubber pyrolysis products, wool fibers, polychloroprene, and ionizing radiation.

Most substances showing excess risk based on cancer controls also showed elevated risk based on population controls, but the precision in risk estimates was lower using the latter group of referents. Risk estimates based on population controls for those 8 substances with a lower confidence limit of the OR $\geq$1.0 at the "any exposure" level based on cancer controls are shown in Table IV.

Occupations and industries

We investigated the effect of having been employed in 24 selected occupations or industries. Occupations retained for analysis included metal processors, metal machinists, sheet-metal workers, welders and flame cutters, general machinists, metal products fabricators, service station attendants, printers, masons and tile setters, leather workers, performing and graphic artists, and police and firefighters, whereas industries included iron, steel mills and foundries, metal fabricating and machining, rubber prod-

TABLE III – OR AND 95% CI FOR THE RELATION BETWEEN EVER EXPOSURE TO SELECTED SUBSTANCES AND RECTAL CANCER, USING 2 STATISTICAL MODELS, BASED ON CANCER CONTROLS

Substances	Model 1 Adjusted for non-occupational factors		Model 2 Adjusted for non-occupational and occupational factors	
	N[1]	OR1[2]	OR2[3]	95% CI
Rubber pyrolysis products	10	3.0	2.6	1.2–5.9
Wool fibers	26	1.6	1.6	1.0–2.6
Carbon tetrachloride	16	2.0	1.4	0.7–2.7
Trichloroethylene	12	2.0	1.6	0.8–3.5
Acetone	11	2.3	1.6	0.7–3.6
Toluene	50	1.4	1.1	0.8–1.7
Polychloroprene	14	2.0	1.8	0.9–3.7
Ionizing radiation	6	3.8	3.4	1.1–10.5

[1]N, number of exposed cases.–[2]Adjusted for age, education, respondent status, cigarette smoking, beer consumption, and body mass index.–[3]Adjusted for the non-occupational variables listed above as well as for all of the other substances in this table.

ucts, petroleum and coal products, mineral fuels, printing and publishing, pulp and paper mills, sawmills, clothing, leather goods, defense services, and household furniture. Results based on occupations and industries were less directly informative about the carcinogenicity of the occupational substances *per se,* but they were more comparable to results from other studies based on job titles. The number of exposed cases was often very low, resulting in imprecise OR estimates. None of the occupations exhibited very strong and persuasive associations with rectal cancer. There were suggested associations for prolonged employment among leather workers [$OR_{\geq 10\ years} = 1.6$, 95% confidence interval (CI) 0.9–2.8, 16 exposed cases] and performing and graphic artists ($OR_{\geq 10\ years} = 2.3$, 95% CI 0.9–6.1, 6 exposed cases). Four industries exhibited some elevated risks: household furniture ($OR_{ever} = 2.7$, 95% CI 1.3–5.8, 11 exposed cases; $OR_{\geq 10\ years} = 4.8$, 95% CI 1.6–14.0, 7 exposed cases), rubber products ($OR_{ever} = 3.6$, 95% CI 1.1–11.7, 5 exposed cases), iron, steel mills and foundries ($OR_{ever} = 1.8$, 95% CI 0.9–3.7, 12 exposed cases), and sawmills ($OR_{ever} = 2.7$, 95% CI 0.9–8.1, 5 exposed cases).

DISCUSSION

Our study was based on a reliable, case-by-case, expert-performed, detailed exposure assessment (Siemiatycki *et al.,* 1997) regarded as the best approach for population-based retrospective studies (Bouyer and Hémon, 1993). In addition, we studied histologically confirmed incident cases and obtained information on several potential confounders. Nevertheless, these had a relatively modest impact on risk estimates. The choice of control group is often problematic as different methods have advantages and disadvantages (Hennekens and Buring, 1987). We had access to population and cancer controls, and both led to the same conclusions.

Although this study benefited from an expert's translation of lifetime occupational history into specific exposures, it is still subject to exposure misclassification. The misclassification was non-differential, leading to attenuation of OR estimates (Hennekens and Buring, 1987). Another limitation of our study was the limited statistical power due to the low exposure prevalence for some substances and a modest number of study subjects. Still, notwithstanding these limitations, this is the largest case-control study of rectal cancer with relatively high-quality exposure assessment.

We included several non-occupational variables as co-variates, but we had very limited data on dietary variables, some of which are likely risk factors for rectal cancer (World Cancer Research Fund, 1997). In the last 3 years of field work, our questionnaire included a brief checklist of foods rich in β-carotene. We used this information to derive a β-carotene index and analyzed it in relation to rectal cancer. There was no association in our data, and the β-carotene index was not retained as a confounder. In future work attempting to replicate our findings, it would be desirable to collect data on dietary variables. Nevertheless, the associations between various dietary habits and rectal cancer, on the one hand, and occupational exposures, on the other, are unlikely to be strong enough to greatly distort the association between occupational exposure and rectal cancer. One dietary co-variate retained in our analyses was beer consumption, which has shown some weak association with rectal cancer risk (IARC, 1988; Stemmermann *et al.,* 1990; Dean *et al.,* 1979; Kabat *et al.,* 1986; Kato *et al.,* 1990; Pickle *et al.,* 1984).

We have examined the relation between rectal cancer and 54 occupations, 76 industries, and 294 substances. For the present in-depth analysis, we examined a subset of pertinent occupational circumstances because of suggestive evidence from other research or from our preliminary screening analyses. It can therefore be inferred that for all those substances, occupations, and industries not listed here, our evidence was either negative or insufficiently precise to warrant presentation. A full list of the substances, occupations, and industries examined in our screening analyses has been published (Siemiatycki, 1991).

A full assessment of causality for each of the substances examined would also require consideration of experimental evidence of carcinogenesis, and physiological–metabolic evidence concerning the fate of these substances in the body. Examination of these lines of evidence is beyond the scope of this report.

We found higher risks of rectal cancer among workers exposed to rubber dust and rubber pyrolysis products and in the rubber industry. Excess risks of rectal cancer have been reported among rubber workers in Massachusetts (Dubrow and Wegman, 1984) but not in the German and British rubber industries (Sorahan *et al.,* 1986; Weiland *et al.,* 1996). We also noted elevated ORs among leather workers, who are exposed to rubber dust through rubber sole-buffing activities.

There were indications of excess risk for exposure to cotton dust, wool fibers, and rayon fibers. These substances were strongly correlated with one another, therefore preventing mutual adjustments. A study in the Nordic countries found higher incidence rates of rectal cancer among textile workers (Andersen *et al.,* 1999). We also noted significantly raised risks in the household furniture industry, where workers are exposed to fabric dusts through upholstery activities.

All 10 solvents subjected to the present in-depth analyses exhibited excess risk of rectal cancer to various degrees. However, in several instances, their effects could not be disentangled due to strong intercorrelations. As a result, mutual confounding is likely to occur among various subsets. One previous study indicated an elevation in risk for solvents as a class (Berlin *et al.,* 1995), but others found no such association (Gerhardsson de Verdier *et al.,* 1992; Peters *et al.,* 1989). Our data are compatible with a slight increase in risk associated with toluene exposure, though adjustment for other occupational factors suggests that the observed risk estimate might be subject to confounding by 1 or more of the occupational exposures. The excess risk observed among styrene-exposed workers corroborates results from a Finnish cohort study (Anttila *et al.,* 1998). Many workers potentially exposed to MAHs have been found to be at excess risk of rectal cancer, including Canadian metal mill workers (Gallagher and Threlfall, 1983), US stationary engineers and firefighters (Decoufle *et al.,* 1977), and service station attendants (Gerhardsson de Verdier *et al.,* 1992).

Excess risks of rectal cancer have been reported among workers exposed to inks (Gerhardsson de Verdier *et al.,* 1992), as well as among printers (Andersen *et al.,* 1999; Minder and Beer-Porizek, 1992). In our study, there was some weak evidence of higher risks among workers exposed to inks ($OR_{subst} = 2.3$, 95% CI 0.5–2.9,

TABLE IV – OR AND 95% CI FOR THE RELATION BETWEEN EXPOSURE TO SELECTED SUBSTANCES AND RECTAL CANCER, BASED ON POPULATION CONTROLS

Substances	Exposure level					
	Any			Substantial		
	N[1]	OR[2]	95% CI	N	OR	95% CI
Rubber pyrolysis products	10	2.1	0.8–5.5	1	2.2	0.1–36.4
Wool fibers	26	2.2	1.2–3.9	19	3.9	1.8–8.4
Carbon tetrachloride	16	1.5	0.8–2.9	2	0.7	0.1–3.3
Trichloroethylene	12	2.4	1.0–5.6	3	1.1	0.3–4.6
Acetone	11	1.6	0.7–3.4	8	2.0	0.8–5.3
Toluene	50	1.5	1.0–2.3	17	1.6	0.8–3.0
Polychloroprene	14	1.6	0.8–3.3	2	343.4	0.0–∞
Ionizing radiation	6	2.3	0.7–7.4	0	—	—

[1]N, number of exposed cases.–[2]Adjusted for age, education, respondent status, cigarette smoking, beer consumption, and body mass index.

6 exposed cases) but only when population controls were used. We found moderately elevated ORs for performing and graphic artists. This supports results from other studies on professional artists (Miller and Blair, 1985), painters, potters, musicians, actors (Minder and Beer-Porizek, 1992), and artistic workers (Andersen *et al.,* 1999).

Our observation of raised risks among workers in the pulp and paper mills and sawmills concurs with that from the Swedish paper industry (Arbman *et al.,* 1993).

Working with leather was associated with cancer of the rectum in our data. Excess risks have been observed previously for shoe/leather workers (Gerhardsson de Verdier *et al.,* 1992). Polychloroprene, when combined with toluene, produces contact cement, which is used by shoemakers. Our results are consistent with a significant 2-fold excess in risk among workers exposed to polychloroprene.

An elevation of rectal cancer risk among subjects exposed to combustion gases from coke, coal, and wood has been reported in Sweden (Gerhardsson de Verdier *et al.,* 1992), but our data did not replicate this finding.

A review of metal-working fluids concluded that straight oil might be associated with an increased risk of rectal cancer (Calvert *et al.,* 1998). There was no excess risk associated with exposure to cutting fluids in our database nor was there an excess risk of rectal cancer in relation to any of the metallic dusts under analysis, in contrast with a previous report indicating excess risk in relation to non-ferrous metals and metal dust or fumes (Peters *et al.,* 1989). There have been reports of excess rectal cancer in the metal industry (Arbman *et al.,* 1993; Gallagher and Threlfall, 1983) and related occupations (Dubrow and Wegman, 1984; Jakobsson *et al.,* 1997; Minder and Beer-Porizek, 1992), but our findings, based on different types of metal worker, do not support those results. One exception was the iron, steel, and foundry industry, for which there was some evidence of excess risk in our data.

Higher risks of rectal cancer were associated with substantial exposure to formaldehyde. This gas is mainly used for plastic and resin manufacture. Evidence of an increased risk of cancer of the rectum was observed in a group of workers in a plastic-producing plant (Marsh, 1983).

We found increased risks among workers exposed to extenders, a group of multipurpose additives often found in the same work environment as styrene. No other evidence concerning that association was found in the literature.

Lastly, glass fibers are used mainly in insulation, plastic reinforcing material, and special textiles. An excess risk of rectal cancer was associated with substantial exposure to glass fibers in our data set. Exposures to glass fibers and styrene were moderately correlated.

In summary, this hypothesis-generating study earmarked a number of possible occupational risk factors for rectal cancer. However, the available evidence remains too limited to justify firm conclusions, especially since, for most of these occupational circumstances, there was no or very little corresponding literature with which to compare our results.

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