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Gastrointestinal cancer among cement workers

A case-referent study

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Summary. A matched case-referent study was conducted to investigate the risk of gastrointestinal cancer in cement workers. All male cases of cancer in esophagus, stomach, colon and rectum in the period 1922–1988 from two parishes surrounding a cement plant were identified. For each case, four referents were chosen, matched to age and year of death. Employment in the cement plant was confirmed as from plant records or job title noted in the parish books. Of 25 cases with a definite colorectal tumour, 40% had been blue-collar cement workers for 25 years or more, compared with 20% of their referents [odds ratio (OR) 3.2; 95% confidence limits (CI) 1.1–9.4]. There were indications of a dose (time of employment)-response relationship. For 18 cases with an esophageal or stomach tumour, the OR for those ever employed as cement workers was 3.2 (CI 1.0–11), but short-term employment predominated, thus making a causal relationship less likely.

Key words: Cancer – Case-referent study – Cement dust – Colorectal – Stomach

Introduction

In the cement industry, respiratory hazards of inhaled dusts have been long recognized [6, 13]. Recently, an increased risk for gastrointestinal (GI) tumours, especially stomach cancer, has been suggested [7], though there are contradictory data [2, 14]. A parallel to the suggested risk for stomach tumours among workers exposed to high concentrations of other inorganic dusts [10] might be drawn. In portland cement, hexavalent chromium, which is an established carcinogen [5], and other trace metals are present. Furthermore, in a cohort of asbestos-cement workers [1], we have found a surprisingly high risk for gastrointestinal tumours, compared to cohorts of workers in other areas of the asbestos industry. Also, among masons handling cement, lung cancer has been seen in excess in a small study [12]. We present here a case-referent study of gastrointestinal (ICD-7 150–154) tumours among portland cement workers.

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Materials and methods

Cement production. Portland cement production started in 1919 in a little village, where lime kilns have been common since the middle ages.

The annual workforce in the cement plant has numbered between 200 and 400 blue-collar workers, and migration has been low. The cement making process includes quarrying and crushing raw marlstone, calcining and grinding of clinker, blending, packing and shipping of the finished product. A wet method was used until 1970; the dry method was phased in from 1965. The quartz content in the raw material is very low, and thus small amounts of quartz sand are added. Gypsum is added, and during the last two years also ferrous sulphate. Coal has been used to heat the cement ovens except for a few years in the 1940s, when peat fuel was used, and between 1960 and 1982, when oil was used.

Total dust measurements since the mid-1970s (membrane filter method; personal sampling) were generally lower than 10 mg/m^3; but with some exceptions up to 25 mg/m^3. The respirable dust fraction was generally lower than 0.2. The chromium content in clinker dust samples obtained next to a cement oven was 389 mg/kg and in cement dust samples 125 mg/kg. In the finished product, the chromium content was 40 mg/kg.

Silicosis among workers has never been observed. As in all high temperature industrial production, asbestos has been used for insulation. A few cases of pleural plaques, but no case of parenchymal asbestosis, have been found.

Study population. Death records from two parishes in the village and its surroundings were used as the primary source for cases and referents. In these records, the cause of death from the death certificate is entered. The cases included 53 men with a noted GI cancer (esophagus, stomach, small and large intestine), deceased from between 1922 and 1988. For each case four referents of the same age (± 5 years) were chosen with two referents dying before the case, two after him. All but 15 referents had died within ± 5 years; all within 13 years as compared with their case. Those with lung cancer, mental deficiency or uncertain cause of death were rejected.

The National and Regional tumour registries were used as a secondary source of cases. Since 1971, a parish code has been entered for all cancer cases in these registries; thus we obtained all

GI cancers (ICD-7 150–154) reported to the registries in the period 1971–1988 from these parishes. Another three GI-tumours were found, the tumour not being mentioned in the death certificate. Referents were chosen as above. Also, four living cases with a gastrointestinal cancer were found, residing in the present parishes at the time of the diagnosis. For these cases, living referents, age-matched as above, were chosen from the same parish as the case. All referents were searched for in the tumour registries. Thus, we made sure that none of them had had a GI cancer reported to the registry during the period 1958 (when the National Tumour registry was established) to 1971.

Hospital records were searched for verification of diagnosis. Also, the information in the tumour registries concerning type of tumour and grounds for diagnosis was used. One esophageal cancer and seven stomach cancers were verified through histopathology or cytology. Four stomach tumours were seen at autopsy or laparatomy. A clinical diagnosis of stomach cancer was probable in five cases, somewhat doubtful in one. Twenty-one out of 29 colorectal cancers were verified through histopathology or cytology. For five colorectal cases, clinical investigation including X-ray examinations and/or laparatomy findings were judged as warranting the diagnosis. These tumour cases are included in the analyses for cancers of the separated "esophagus and stomach" and "colorectal" sites. Thus the localization of the tumour should be correct as from all sources available, but a microscopic diagnosis is not a prerequisite.

Four cases had, according to these sources, a doubtful malignant primary gastrointestinal (GI) tumour. These are still included in the "all GI-cancer" group but excluded from the separate sites. Four cases with the diagnosis "cancer ventriculi", five cases with "stomach cancer", one case with "disseminated abdominal cancer", two cases with colorectal tumours and one "ca intestini" were treated in the same manner, as no hospital records or tumour registry notes were available to confirm the diagnosis in the death certificate. Thus, in the "all GI cancer" group, some abdominal cancers other than those which should be classified into ICD-7 150–154 may be included.

Only one GI cancer case out of 23 was verified through histopathology/cytology before 1960. After 1960, 14 out of 18 tumours among blue-collar cement workers and 13 out of 19 tumours among the other men were microscopically verified.

In summary, we found 60 "all GI" cancer cases. Eighteen cases had a verified esophageal or stomach cancer and 25 had a verified colorectal cancer. In a few instances, a full set of referents was not available, due to the matching criteria. Thus, there are 234 referents to the GI cancer cases.

Exposure. In the parish books, occupational titles of all deceased men were regularly noted. "Cement workers" began to appear in the records in the mid-twenties. All "cement workers" among cases and referents were noted, with the first ones appearing among them in 1940.

Since 1945, data were kept on all employees terminating their employment at the cement plant. Cases and referents were searched for in these files. For a few men, these data were completed with information from a long-term personnel manager at the plant. Relatives to "cement workers" not found in the files, and to employees whose tenure still was uncertain, were interviewed by telephone in order to obtain more information on employment. In 12 such interviews, the aim of the present study was stated as an investigation of possible health risks from cement work. GI-tumours were not explicitly mentioned, and the interviewer was not aware of the case/referent status of the subject.

All information available was used to classify cases and referents into "< 5 years", "5 to 24 years" or "25 years or more". For a referent, only employment time and time since first employment until the year of death for his case was considered. For seven employees or "cement workers", the duration of exposure could not be exactly established. They were placed in the lowest reasonable exposure and latency time group that could be considered, thus making dose-response estimates conservative.

All occupational titles for cases and referents were classified into five groups for comparison of socio-economic status. An unmatched analysis displayed a similar socio-economic spectrum.

Statistics. Matched analyses were performed using conditional logistic regression [3] as implemented in the PECAN program (Storer B. PECAN User's notes, version 2.2 1984). Given a certain level of exposure, the risk for cancer is thus assumed to be constant within each matched set. Crude trend tests were performed by assigning the four employment categories the numbers 0, 1, 2, 3.

Table 1. Employment in a cement plant among cases with gastrointestinal tumours and their referents.

Employment/latency	All gastrointestinal cancers				Esophagus and stomach[a]				Colon and rectum[a]			
	Cases		Referents		Cases		Referents		Cases		Referents	
	N	%	*N*	%	*N*	%	*N*	%	*N*	%	*N*	%
Employed	21	35	73	31	7	39	15	21	12	48	43	45
Blue collar												
−5 yrs employment												
Latency −5 yrs	1	2	1	0.4	1	6	–	–	–	–	1	1
5–24 yrs	1	2	3	1	1	6	–	–	–	–	2	2
25+ yrs	2	3	4	2	1	6	–	–	–	–	4	4
5–24 yrs employment												
Latency 5–24 yrs	2	3	12	5	1	6	2	3	1	4	7	7
25+ yrs	3	5	20	8	2	11	7	10	–	–	8	8
25+ yrs employment	11	18	27	12	1	6	4	6	10	40	20	21
White collar												
Latency −5 yrs	–	–	–	–	–	–	–	–	–	–	–	–
5–24 yrs	1	2	–	–	–	–	–	–	1	4	–	–
25+ yrs	–	–	6	3	–	–	2	3	–	–	1	1
Not employed	39	65	161	69	11	61	57	79	13	52	53	55
Total	60	100	234	100	18	100	72	100	25	100	96	100

[a] Verified cases from hospital records and/or tumour registry notes

Results

The distribution of *"all GI"* cancer cases over employment time and latency strata was comparable to that of the referents (Table 1). Thus, in the matched analysis there was no association between "all GI" cancer and employment as a blue-collar worker as compared to non-employees and white-collar workers at the plant (OR = 1.3; $P = 0.4$).

For verified *"esophageal and stomach cancer"*, the corresponding figure was OR = 3.2, (95% CI, 0.96–10.7; $P = 0.06$). However, short-time blue-collar employment in the cement plant was more common among cases than among referents (Table 1).

For verified *"colorectal cancer"*, there was no association (OR = 1.1; $P = 0.9$) with employment as a blue-collar worker at the plant. In contrast to the distribution of tumours in the upper GI tract over employment and latency strata, long-term employment predominated among the colorectal cancer cases (Table 1).

A matched analysis with duration of employment divided into <5 years, 5 to 24 years, 25+ years was undertaken. The relative risk for colorectal cancer in these employment strata, compared to no employment or white-collar employment, was OR <0.1 (no case), 0.25 and 2.3 respectively. Due to the small number of tumours, significance testing of the risk estimates in the individual strata was not meaningful. Nor did the numerical trend for dose-response reach statistical significance.

The matched analysis was repeated, comparing instead blue-collar workers with 25+ years of employment with all those with a shorter employment time, all white-collar workers and those not employed. Colorectal cancer was then significantly associated with long-term blue-collar employment (OR = 3.2; CI 1.1–9.4; $P = 0.03$).

Discussion

Our case-referent study is small and its statistical power accordingly low. The power for detection of a three-fold risk elevation of colorectal cancer in 25 cases, with no restrictions as to employment time and latency, is only 64%.

We found a significant association between *"long-term employment"* as a cement worker and colorectal cancer. The relative risk was three-fold; also, there were indications of a dose-response relationship, though not significant. A numerically elevated odds ratio for cancer in the upper GI tract, almost reaching significance, was also found. The distribution of cases indicated that this was due to an excess among short-time employees and thus not related to the cement exposure.

The use of deceased referents might, theoretically, introduce a bias if deceased referents are overrepresentative of "unhealthy" lifestyle or medical conditions associated with premature death. This might particularly be a risk in younger ages. The main point of interest is if such possible health and behavioural differences between cases and referents before death would affect the possibility of being exposed to cement, i.e. to become employed, and to stay employed, in the industry. We excluded potential referents with debility or mental deficiency as a cause of death, but not those with alcohol-related diagnoses. However, when, in a recent study [9], living and dead referents were compared, dead referents were, as expected, heavier cigarette smokers and consumers of alcohol, but no differences concerning usual industry of employment were demonstrated. Neither could we, in this study, demonstrate any differences between cases and referents as to job title/profession. Thus, a severe distorting bias in this respect is probably not present. Nor is there any reason to suspect marked dietary differences.

Potential referents with lung cancer were also rejected, as some asbestos exposure was known to have occurred at the cement plant. Also, lung cancers in excess have been reported in a cohort of masons using cement [12]. As a separate part of the present study (not reported here) the association between lung cancer and cement work was also investigated. No evidence of an excess risk was demonstrated. The number of cases was, however, very small.

A recent Swedish study [16] showed an excess of esophageal and stomach tumours and fewer colorectal cancers than expected among blue-collar workers and farmers. In contrast, an excess of colorectal cancers was seen among white-collar workers. Thus, distorting factors linked to socio-economic differences would act in the opposite direction to that which our results suggests.

Much effort was made to validate the diagnoses. However, especially for the cases appearing early in the observation period, histopathology seldom had been performed. This particularly influences stomach tumours, whereas the colorectal tumour diagnoses should be quite reliable. There was no marked difference in quality of diagnosis between exposed and unexposed men. Thus, a misclassification bias should not be operating.

In an recent registry-based study of adenocarcinomas in the stomach [17], an occupational title in the register indicating dust exposure was related to an increased risk, especially for tumours in antrum/pylorus and for exposure to mineral dust. In a British registry study of men, denoted a cement workers in 1939, McDowall [7] found an excess of stomach cancer with an SMR of 175. Preliminary results from a cohort study [14], following the initial one, showed a slightly raised, but not significant SMR of 119 for stomach cancer. No data on duration of exposure were presented. In a cohort [2] of 4231 cement workers from six cement plants in the USA, 27 stomach cancer deaths were found. No association with tenure could be demonstrated, keeping latency, age at follow-up and year of birth under control. The number of cases was, however, small, thus yielding low statistical power.

Our results regarding stomach cancer are in agreement with these two latter studies. The numerical excess of upper GI tumours among short-time employees, indicated in our study, might be compared to the excess of cancers often seen in short-time employees [4, 8]. Also, among Danish asbestos-cement workers, stomach cancer has been noted in excess among short-time employees [11].

A raised risk for colorectal tumours in cement workers, as we found, has not previously been described. Interestingly, however, in the McDowall study [7] a non-significantly raised SMR of 188 for rectal tumours was noted. Also, in a cohort study of asbestos-cement workers [1], we have seen an elevated risk for colorectal tumours that was higher than expected. Moreover, workers exposed to dust from stainless steel grinding had an increased risk for colorectal cancer [15]. Thus, there are suspicions that several types of dust are associated with colorectal cancer.

Our results, together with earlier studies, do not provide enough evidence for either acceptance or refutation of a hypothesized risk for malignant tumours in the cement industry. However, it suggests a risk for colorectal cancer after long exposure. A cohort study of cement workers is now in progress.

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