Cancer mortality among magazine printing workers

Danièle Luce, Marie-France Landre, Thierry Clavel, Isabelle Limousin, Sylvie Dimerman, Jean-Jacques Moulin

Abstract

Objectives—After an inquiry from the employees of an offset printing plant, a historical cohort study was conducted to investigate cancer mortality among these workers.

Methods—The cohort comprised 262 men, who contributed 2771 person-years of observation. 16 deaths were identified during the follow up period (1980–91). Expected numbers of deaths were derived from age specific regional rates. Standardised mortality ratios (SMR) and 95% confidence intervals (95% CIs) were calculated.

Results—An increased cancer mortality was found after 10 years of employment (SMR 213, 95% CI 98 to 405, based on nine deaths), mainly due to a high mortality from lung cancer (SMR 381, 95% CI 104 to 975, four deaths), and from oesophageal cancer (SMR 1049, 95% CI 216 to 3065, three deaths). For workers with at least 20 years since the start of employment, the SMR was 262 (95% CI 105 to 540) for all cancer sites, 447 (95% CI 92 to 1306) for lung cancer, and 1094 (95% CI 132 to 3952) for oesophageal cancer. The increased cancer mortality was concentrated among pressmen.

Conclusion—Although based on small numbers, the findings suggest an increased risk of cancer among these workers, which should be further investigated.

Keywords: printing workers; cancer

Several investigators have reported an increased cancer mortality among workers in the printing industry. Studies have been conducted among various groups of printing workers, and excess risks have been found for different cancer sites, particularly for lung cancer, haematopoietic neoplasms, and colorectal cancer. Increased risks have also been detected for melanoma, cancers of the buccal cavity and pharynx, pancreas, kidney, and liver. However, the mortality experience of workers in offset printing has not been specifically studied.

In 1991, employees in an offset printing plant expressed concern about a high number of cancer deaths. We thus initiated a mortality cohort study, to find whether cancer mortality was increased among these workers relative to the general population and to examine mortality patterns which could suggest an occupational aetiology.

Material and methods

The plant has produced weekly and monthly magazines, and the four colour offset printing process has been used since the start of printing in 1956. Printing was carried out until 1972 with sheet fed offset printing machines, then with web fed offset printing machines, then with web fed offset litho presses. Ink mists may be generated when the presses are running. Printing inks may contain polycyclic aromatic hydrocarbons, including benz(a)pyrene, and other known or suspected carcinogens, such as dichlorobenzidine derived dyes. In the plant, the offset litho machines included a damping system, which used an isopropanol solution. The presses were cleaned after each operating cycle, and various solvents (mainly petroleum solvents) were used during cleaning operations.

Reliable personnel records were available only since 1980. The study population was then defined as all male workers still employed on 1 January 1980 (211 men) or employed since then (51 men). The cohort included only workers who had been employed for at least one year. For each worker, the contribution to person-years at risk started after they had been employed for one year or at the beginning of the follow up period (1 January 1980), whichever occurred later, and was ended on the date of death or at the end of study (31 December 1991).

Vital status was ascertained up to 31 December 1991 from information provided by the registry office of the subject’s birthplace, and by the national file of the French National Institute for Statistics (INSEE). Both sources were used for French citizens. As deaths of foreigners are also recorded in INSEE files, if they died in France, foreign workers who had left work and who were not identified as dead were assumed to be alive at the end of follow up. Date and place of birth, date of start and end of employment, and job title were collected for each subject from company’s records. For most workers, only the last job title was recorded. However, workers’ job mobility has been limited over time, and changes, if any, were usually from an unskilled to a skilled job within the same category. It was therefore possible to categorise each worker according to the following groups: pressmen, packers and forwarders, binders,
Results
The cohort comprised 262 men, who contributed 2771 person-years of observation. At the end of the follow up, 228 men were known to be alive, 16 had died, and 18 men who were not identified as dead were assumed to be alive. Table 1 shows the mortality for selected causes. Although slightly increased, all cause mortality was not significantly above that expected. An increased mortality from cancer was found, which was mainly due to an excess of deaths from lung cancer and oesophageal cancer. The other cancer deaths were one from kidney cancer (v 0·10 expected), one from colon cancer (v 0·22 expected), and one from cancer of an unknown site. Also, a high SMR was found for cirrhosis of the liver.

Discussion
Standardised mortality ratios based on small numbers are subject to wide fluctuations, and should be regarded with caution. Nevertheless, several findings suggest that this cohort presents an increased cancer mortality, particularly for lung cancer, which could be related to occupation. The largest SMRs were found among workers with the longest duration of employment and time since first employment, younger age at hire, and the excess was limited to pressmen. No increased cancer mortality was found among workers who began employment after 1970. This may reflect changes in work exposures. However, a sufficient latent period may not yet have passed to show an excess of cancer in this group.

Table 1  Observed deaths and SMRs for selected causes

<table>
<thead>
<tr>
<th>Causes of death (ICD-9)</th>
<th>Obs</th>
<th>Exp</th>
<th>SMR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All causes (000–999)</td>
<td>16</td>
<td>14·48</td>
<td>110 (63 to 178)</td>
</tr>
<tr>
<td>All cancer sites (140–239)</td>
<td>10</td>
<td>5·37</td>
<td>186 (89 to 342)</td>
</tr>
<tr>
<td>Oesophagus (150)</td>
<td>3</td>
<td>0·36</td>
<td>843 (174 to 2463)</td>
</tr>
<tr>
<td>Liver cirrhosis (571)</td>
<td>4</td>
<td>1·31</td>
<td>305 (83 to 781)</td>
</tr>
<tr>
<td>Oral cancer (140)</td>
<td>3</td>
<td>0·83</td>
<td>360 (74 to 1052)</td>
</tr>
</tbody>
</table>

Maintenance workers, platemakers, and others (mainly administrative workers). Data on smoking were abstracted from the occupational physician’s medical records. Causes of death were obtained from the national file of the French National Institute of Health and Medical Research (INSERM), which is responsible for the coding of all French death certificates. Expected numbers of deaths were calculated from regional rates, with 10 year age group specific rates. The reference rates of 1985 were applied to the whole study period. Analyses with national rates and two calendar periods (1980–6, 1987–91) produced similar results and are not presented in this report. Comparisons of observed and expected numbers of deaths were expressed as standardised mortality ratios (SMRs) and exact 95% confidence intervals (95% CIs) based on the Poisson distribution were calculated.11 The SMRs were also computed according to years since first employment, employment duration, year of hire, age at hire, and job category.

Table 2  Cancer mortality by job category, year of hire, age at hire, duration of employment, and time since first employment

<table>
<thead>
<tr>
<th>Job category</th>
<th>All sites</th>
<th>Lung</th>
<th>Oesophagus</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Obs</td>
<td>Exp</td>
<td>SMR (95% CI)</td>
</tr>
<tr>
<td>Pressmen</td>
<td>2·13</td>
<td>328 (132 to 676)</td>
<td>3</td>
</tr>
<tr>
<td>Others</td>
<td>3·23</td>
<td>93 (19 to 272)</td>
<td>1</td>
</tr>
<tr>
<td>&lt; 1970</td>
<td>2·09</td>
<td>268 (116 to 528)</td>
<td>4</td>
</tr>
<tr>
<td>≥ 1970</td>
<td>2·38</td>
<td>84 (10 to 303)</td>
<td>0</td>
</tr>
<tr>
<td>&lt; 30</td>
<td>2·22</td>
<td>225 (73 to 525)</td>
<td>3</td>
</tr>
<tr>
<td>≥ 30</td>
<td>3·14</td>
<td>159 (52 to 371)</td>
<td>1</td>
</tr>
<tr>
<td>&lt; 10 y</td>
<td>1·14</td>
<td>88 (2 to 490)</td>
<td>0</td>
</tr>
<tr>
<td>≥ 10 y</td>
<td>4·22</td>
<td>213 (98 to 405)</td>
<td>4</td>
</tr>
<tr>
<td>&lt; 20 y</td>
<td>2·69</td>
<td>111 (23 to 324)</td>
<td>3</td>
</tr>
<tr>
<td>≥ 20 y</td>
<td>2·67</td>
<td>262 (105 to 540)</td>
<td>3</td>
</tr>
</tbody>
</table>
Tobacco smoking is a potential confounder for lung cancer. Smoking habits were available for only 75% of the workers. It was nevertheless possible to assess the potential confounding effect of smoking on lung cancer by performing the indirect adjustment proposed by Axelson. Among workers with known smoking habits, 20% had never smoked, 15-3% were former smokers, 25-9% were moderate smokers (<20 cigarettes a day), and 38-8% were heavy smokers (20 cigarettes a day). The proportions of the various categories of smokers in the French male population in 1980-1, adjusted for age distribution of the cohort, were 25% never smokers, 26-4% former smokers, 21-6% moderate smokers, and 26-3% heavy smokers. Assuming relative risks of 1, 5, 10, and 20 among never, former, moderate and heavy smokers respectively, differences between the smoking habits of the cohort and those of the French population would lead to an SMR of 126 for lung cancer. When data on smoking in France in 1991-2 were used for reference, the lung cancer SMR due to smoking alone would be 139. Given the magnitude of the SMRs found in this study, it seems that the increase in risk of lung cancer could not be entirely explained by confounding with smoking.

The risk of lung cancer among workers in the printing industry has been investigated in several studies. These studies are difficult to compare because they involved different occupational groups, different sections of the industry, and different printing processes. Increased lung cancer risks were found among workers classified as pressmen, printers, typographers, and lithographers. Significant or non-significant excess mortality from lung cancer has been reported among newspaper pressmen. Proportionate mortality studies within other sections of the printing industry failed to show a significant increase in risk, although slightly increased proportional mortality rates were found. Only a few studies have provided information on the printing process. A significant increase in risk of lung cancer was found among newspaper pressmen operating rotary letterpress machines in Manchester. In a recent study from Denmark, factory workers in newspaper and magazine production showed an excess risk of lung cancer, which was attributed to work with rotary letterpress printing.

Exposure to ink mists, and more specifically to polycyclic aromatic hydrocarbons such as benzo(a)pyrene, was suggested to be a possible cause for the excess in lung cancer among letterpress printers. Our results indicate that pressmen in magazine production with offset technology may also have had an increased occupational risk of lung cancer. These workers were also exposed to ink mists which contain benzo(a)pyrene. However, the concentration of benzo(a)pyrene in offset inks is lower than in letterpress inks, and offset inks are less liable to produce ink mist. In a recent analysis of offset inks used in France in printing plants similar to the present one, concentrations of benzo(a)pyrene were generally low (<100 μg/kg), although in some inks relatively high concentrations of about 1 mg/kg were found. The same study found low atmospheric concentrations of benzo(a)pyrene (<1 g/1000 m³), equivalent to those usually found in urban areas (M Lafontaine, personal communication). On the other hand, although no objective data were available, it is generally admitted that the concentration of polycyclic aromatic hydrocarbons in offset inks has decreased over the past 20 years. Furthermore many tasks which are now automated were performed in the past in the vicinity of unenclosed presses without exhaust ventilation. Consequently, relatively higher exposures may have occurred in the past. Thus the role of ink mist in the excess of lung cancer found in the present study may not be ruled out.

The increase in deaths from oesophageal cancer is more likely to be related to non-occupational factors. Alcohol consumption is a known risk factor for this disease, and the increased SMR found for cirrhosis of the liver indicated a high proportion of heavy drinkers among these workers. However, mortality from cirrhosis of the liver and from oesophageal cancer did not show similar patterns, so that factors other than smoking and alcohol could not be excluded. Several studies have provided data on the risk of oesophageal cancer among printing workers, none of which have shown a significantly increased risk. Conversely, it is interesting to note that an excess of cirrhosis of the liver has been previously reported in two studies among newspaper pressmen.

Because of the small size of the cohort, the short follow up, and the lack of information on occupational exposures, definitive conclusions could not be reached. Nevertheless, despite its limitations, this study suggests an increased risk of cancer, especially lung cancer, among workers in magazine printing with offset technology. Further investigations of this group of workers, including complete occupational histories and characterisation of specific exposures, are needed to confirm and to clarify the present findings.

We acknowledge the cooperation of the company management and Personnel Department (JP Pegen). We thank Dr JP Raymond for providing data on smoking.

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CORRECTIONS

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The bottom row of table 2 should read Total II-V, not I-V. The address for S E Wilcock should be only the Hyperbaric Research Unit, Robert Gordon University, School Hill, Aberdeen AB9 1FR.

Cancer mortality among magazine printing workers Daniele Luce, Marie-France Landre, Thierry Clavel, Isabelle Limousin, Sylvie Dimernan, Jean-Jacques Moulin (1997;54:264-7).

Page 266 column 2 line 4 should read <1 µg, not <1 g. Also in table 2 for oesophagus <1970, the expected number should be 0.20, not 2.20.

BOOK REVIEWS


This book represents a collection of reviews written by the participants in a workshop on the mechanisms of fibre carcinogenesis, held at the IARC in Lyon on 9-11 January, 1996. The goals of the workshop were twofold; to review and discuss the current knowledge on the mechanisms of fibre carcinogenesis, and to use this knowledge in the assessment of carcinogenic risks to humans or animals.

The primary outcome of the workshop was the consensus report, which is presented in the first part of the book, and was agreed by all the workshop participants. This report brings to light a surprising number of weaknesses and data gaps in the available literature on fibre characterisation, genotoxicity, cell proliferation or activation, and animal studies. A prime example of such shortcomings is the general lack of information on the characterisation of fibre dose—that is, fibre numbers, dimensions, surface area, chemistry, durability, and biopersistence—for most in vitro and in vivo studies. The report also discusses the relevance of mechanistic data from in vitro and in vivo assays for the evaluation of carcinogenic risk to humans and concludes with several recommended experimental studies which would provide additional data for the future assessment of fibre carcinogenicity.

The remainder of the book focuses on various aspects of mineral fibre carcinogenicity which were outlined in the consensus report, and such reviews express the opinions of their authors. Briefly, the paper by Kane provides a good discussion of the proposed five mechanistic hypotheses for fibre carcinogenesis. Fubini follows up on these hypotheses by examining the interactions between fibres and cells through the analysis of fibre parameters such as crystallinity, micromorphology, elemental analysis, solubility, and adsorption, which are often not considered by most investigators. The report by Jaurand provides cautious consideration to the limitations and feasibility of mutation and cell transformation assays for investigating the mechanistic effects of fibres. Topics presented by Jeyaratnam and Gordon focus on providing a contribution to the neoplastic effects of various fibres and current issues such as signal transduction pathways, oxidative stress, antioxidant mechanisms, and protooncogene expression. Donaldson describes in detail the role of reactive oxygen species, cytokines, and growth factors in preneoplastic and fibrotic changes. The advantages and disadvantages of in situ hybridisation, intratracheal instillation and intravenous injection are reviewed by Oberdörster. The final review, by Davis, discusses the interactions of inhaled particulate matter along with fibres and the potential effects of mixed doses on fibre pathogenicity.

Overall this book is a collection of concise and up to date reviews on the subject of fibre (mainly asbestos) carcinogenesis. It is generally readable, clear, and informative. Its comprehensive tables and references provides a very good introduction for newcomers to the subject, as well as being an excellent resource for examination candidates. Unfortunately, the most appropriate readers (students) will be unable to afford its high price. The sections in the reviews on recommended experimental studies and unanswered questions are worthwhile to the professional audience. These sections state clearly the directions that research should take to close gaps in data and strengthen current information. There are several similar books on the market today which deal with the health effects and potential effects of mineral dusts, but this book will be of interest to those investigators who work predominately with asbestos fibres.

KELLY ANN BÉRUBÉ


This is the first comprehensive text book on the immune responses of the lungs I have read. Immunopathology of the Lung and Upper Respiratory Tract, edited by John Bien- enstode, was published in 1984. The book