

# Lung Cancer Mortality and Occupational Exposure to Asbestos Among Telephone Linemen: A Historical Cohort Study in France

Djamila Meguellati-Hakkas, MS

Diane Cyr, MS

Isabelle Stücker, PhD

Joëlle Févotte, MS

Corinne Pilorget, PhD

Danièle Luce, PhD

Pascal Guénel, MD, PhD

**Objective:** The authors studied the mortality by lung cancer in telephone linemen exposed to asbestos at low levels during installation of telephone cables. **Methods:** Three hundred eight lung cancer deaths were identified in the cohort. Exposure to asbestos and to other occupational carcinogens was assessed using a job-exposure matrix. **Results:** The relative risk for lung cancer death associated with an estimated exposure of approximately 2 f/cc-years was 2.1 (95% confidence interval = 1.1–4.0) as compared with workers exposed to less than 0.5 f/cc-years. Mean annual exposure or exposure duration were not clearly related to lung cancer. Adjustment for other occupational lung carcinogens did not change this finding. **Conclusion:** The observed mortality by lung cancer associated with asbestos exposure at low levels is higher than the prediction based on linear downward extrapolations from highly exposed occupational cohorts. (J Occup Environ Med. 2006;48:1166–1172)

Asbestos dust has well-known carcinogenic properties.<sup>1,2</sup> Most studies on asbestos and lung cancer risk have been carried out in populations with elevated occupational exposures such as asbestos miners and millers,<sup>3,4</sup> textile workers,<sup>5</sup> or asbestos–cement factory workers,<sup>6</sup> but the carcinogenic effects of intermittent exposures to low levels of asbestos has not been investigated extensively. It is generally assumed that the risk of lung cancer associated with low exposure to asbestos can be predicted from a downward linear extrapolation of the risk observed in highly exposed cohorts.<sup>7</sup> Several groups of workers operating in asbestos-insulated areas such as telephone linemen may provide empirical observation of lung cancer risk due to sporadic exposure to asbestos. To our knowledge, however, no epidemiologic study on respiratory cancer risk has been specifically carried out in telephone linemen, possibly because occupational exposure levels have been lower and hence more difficult to assess than in other industries and because the variety of exposures to potential lung carcinogens at the workplace makes difficult the study of a particular agent separately.

We conducted a study in the French national telephone utility, the only telephone company operating in France at the time of the study. Because of concern due to a cluster of deaths from respiratory cancers and other cancers sites in a group of telephone linemen, a nationwide historical cohort study of cancer mortality was initiated in this large company. Past exposures to oc-

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From Inserm, U754, IFR69 (Dr Meguellati-Hakkas, Dr Stücker, Dr Guénel), Villejuif, France; Inserm, U687, IFR69 (Dr Cyr, Dr Luce), Saint-Maurice, France; and Université Claude Bernard (Dr Févotte, Dr Pilorget), Lyon, France.

The study was financially supported by Fondation de France and by France Télécom.

Address correspondence to: Pascal Guénel, MD, PhD, INSERM U754, 16 avenue Paul Vaillant-Couturier, 94807 Villejuif Cedex, France; E-mail: guenel@vjf.inserm.fr.

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DOI: 10.1097/01.jom.0000243357.70143.47

cupational hazards were assessed based on recollection of workers with longstanding experience to investigate cancer risks in relation to occupational exposures. We report on lung cancer mortality associated with intermittent asbestos exposure occurring mainly during telephone cable installation in asbestos-insulated areas.

## Materials and Methods

The cohort included workers in activity in a technical branch of the telephone company on January 1, 1978, as well as men newly hired between January 1, 1978, and December 31, 1994. Only men fulfilling these criteria and employed as telephone linemen at any time during their work history were included in the present cohort, representing a total of 34,305 workers.

## Vital Status and Cause of Death

Follow up of individual cohort members started on January 1, 1978, and terminated at the date of death or on December 31, 1996, whichever came first. The deceased workers, and the corresponding date of death, were identified from the National Mortality database collecting information on all deaths in France. This database was provided by the National Institute of Statistics for the years 1978 through 1996 and permitted the identification of the deceased workers in the cohort by matching on name, sex, date of birth, and place of birth. To overcome possible problems due to inaccuracies or missing data in the national mortality database, each cohort member was also sought in the files implemented by the personnel department of the telephone company that keeps track of deaths among active and retired workers. The two mortality registers were first used as independent sources of information and were then compared for identifying duplicates. Based on these two files, a total of 2989 deaths were identified in the cohort.

The National Register of Causes of Death (CépiDC-INSERM) was used to obtain the cause of death of the deceased subjects as coded from the death certificate according to the

International Classification of Diseases, 9th Revision.<sup>8</sup> The record of the cause of death was found by matching on gender, date and place of birth, and date and place of death of the deceased employees. In total, 2917 matches were obtained (97.6% of all deaths).

## Occupational History

Individual work histories since the start of employment in the company were obtained from electronic records kept by the personnel department. Jobs were defined by both an occupation and a sector of activity. Occupations were coded according to one of the following six groups: lineman, supervisor, technician, special skill worker, garage worker, or office worker. All cohort members were employed as linemen during at least one job period but may have been employed in any other occupation during their work history in the company. Sectors of activity were one of the following: construction of telephone lines, maintenance of telephone lines, construction of national telephone network, maintenance of the national telephone network, intervention units, garages, or other sectors. For each job period, the date of start and the date of termination of employment were obtained.

## Assessment of Occupational Exposures

Past exposures were assessed by experts using a job-exposure matrix (JEM) approach, ie, a crosstabulation of jobs and occupational exposures. Jobs were coded in the JEM according to both the occupation and the sector of activity, as described previously. Exposures were well-established or suspected occupational carcinogens that occurred at the workplace (Table 1). Different time periods could be defined in the JEM for a given job to account for changes in exposures. For a given exposure in a given job period, semiquantitative indices were used to define exposure probability (P: proportion of exposed workers in a given job), exposure fre-

**TABLE 1**

Occupational Exposures Included in the Job-Exposure Matrix—Telephone Linemen Cohort Study

|   |
|---|
| Acid  |
| Isopropanol   |
| Asbestos  |
| Benzene   |
| Adhesives (PVC material or epoxy)                         |
| Ceramic fibers  |
| Manmade mineral fibers                                    |
| Welding fumes   |
| Car exhaust fumes   |
| Herbicide   |
| Polycyclic aromatic hydrocarbons (PAH)                    |
| Isocyanates   |
| Plastics  |
| Electromagnetic fields (ELF or radiofrequency)            |
| Paints  |
| Lead  |
| Wood dust   |
| Wood preservatives  |
| Ionizing radiations                                       |
| Solvents (chlorinated, oxygenated, or petroleum solvents) |
| Styrene   |
| Ultraviolet radiation                                     |

quency (F: proportion of exposed work time), and exposure intensity (I: estimate of the number of fibers per cubic centimeters for asbestos or appropriate exposure score for other carcinogens).

Exposure assessment was supervised by occupational hygienists and epidemiologists according to a standardized protocol. First, a list of work tasks entailing exposure to the occupational carcinogens was established. Work tasks entailing exposure to asbestos were as follows: using and cutting asbestos sheets as protection equipment during welding, cutting, or drilling asbestos material for insulation purposes, installation of telephone lines, or cable splicing in asbestos-insulated buildings. Experts were asked to assess the proportion of workers within each job who carried out a particular work task and the time duration when the work task was performed in hours per week or month. Exposure probability and exposure frequency were independently obtained from three information sources: a group constituted of occupational phy-

sicians, epidemiologists, and industrial hygienists; a group of experienced or retired workers with good knowledge of the work practices in the company; and a large random sample of current salaried workers who completed a self-administered questionnaire on the work tasks. Whenever a difference occurred between exposure indices, a final consensus was reached during meetings of occupational physicians, experienced workers, and industrial hygienists. The intensity of exposure associated with each task (eg, semi-quantitative exposure indices or estimated fibers per cubic centimeters for asbestos) was determined by the industrial hygienists from the detailed description of the work task and a literature review. When more than one work task caused exposure to the same carcinogen in a given job, the exposures indices attributed to the job were calculated as a weighted average of the exposure indices for the different work tasks.

### Individual Exposure Scores

Annual exposure scores were calculated as the product  $P \times F \times I$  of the exposure indices in the JEM. Cumulative exposure to an occupational carcinogen was then calculated for each worker as the sum of the annual exposure scores over the entire work history. Mean annual exposure was also calculated as the cumulative exposure divided by exposure duration in years.

A priori exposure cut points were defined for the exposure variables to take apart the highest possible exposure levels. We defined a reference exposure category that also included workers with low exposure, because only few linemen had never been exposed to asbestos. For cumulative exposure to asbestos, the 10th, 25th, 50th, 75th, and 95th percentiles were chosen as exposure cut points. For mean annual exposure, the variability of exposure was very low, and only the 25th and the 75th percentiles of exposure distribution were used as cut points.

### Statistical Analyses

The expected number of lung cancer deaths was calculated using the French national age- and period-specific mortality rates for lung cancer in men as a standard to obtain standardized mortality ratios (SMRs). Most analyses presented in this article were carried out internally by comparing asbestos exposure groups within the cohort with no reference to external mortality rates. These analyses were conducted using Cox proportional hazards models,<sup>9,10</sup> in which age was used as the time scale and asbestos exposure scores were treated as time-dependent variables. Adjustment for calendar period (in 5-year periods) was used in all the models. Because the exposure that occurs in the years before lung cancer death may be etiologically irrelevant to the disease, cumulative exposure was also calculated by applying a lag time period of 5, 10, or 20 years and was also fitted as a time-dependent variable. Adjustment for other occupational lung carcinogens was carried out in separate analyses to evaluate a potential confounding effect. All analyses were carried out using STATA software.<sup>11</sup>

### Results

A total of 589,162 person-years of follow up were accumulated by cohort members. Among the 2989 deaths from all causes that occurred in the cohort, 308 were from lung cancer. Descriptive data on employment, follow up, and estimated asbestos exposure are shown in Table 2 for all study subjects and for lung cancer cases separately. The mean year of birth was 1931 for the cases of lung cancer and 1945 for the whole cohort. Indices of asbestos exposure at the end of the follow up were slightly higher in cases of lung cancer deaths than in the rest of the cohort.

The expected number of lung cancer deaths in the cohort based on the French national mortality rates was 328.2, leading to a SMR of 0.94 (95% confidence interval = 0.84–

1.05) (figures not shown). In internal analyses, relative risks (RRs) were calculated by comparing lung cancer mortality between exposure groups within the cohort using different variables for estimating asbestos exposure (Table 3). The RR in the highest cumulative exposure category (estimate >1.7 f/cc-years) was 2.1, and it was statistically significant. Slightly increased RRs were observed in the intermediate categories of cumulative exposure, but there was no evidence of a linear dose-response relationship. Because the range of exposure was small, only three exposure categories were used for the mean annual exposure. No significant increase in lung cancer mortality was observed (RR = 1.3) when comparing workers with mean annual exposure above the 75th percentile with workers with mean exposure below the 25th percentile of exposure distribution. However, the exposure gradient between exposure groups was too small for a meaningful analysis. Duration of asbestos exposure was associated with some increase in risk, particularly in workers exposed more than 32 years, but none of the RRs were statistically significant.

Cumulative exposures were calculated allowing for latency periods of 5, 10, or 20 years before cancer death, ie, not accruing cumulative exposure by annual exposure scores during these periods (Table 4). Elevated exposure to asbestos was more strongly associated with lung cancer mortality when accounting for a latency period of 5 years. Some increase in risk of lung cancer death was still apparent after allowing for a latency period of 20 years.

Results of the multivariate analysis are shown in Table 5. Exposures to asbestos and to other lung carcinogens selected among occupational exposures that occurred in company are included in the models. Adjusting for PAH, engine exhaust and arc welding fumes did not modify substantially the RR associated with asbestos exposure, although it was no longer statistically

**TABLE 2**

Selected Characteristics of Employment, Follow Up, and Asbestos Exposure—Telephone Linemen Cohort Study, 1978–1996

|  | All Cohort Subjects (n = 34,305) |        |         |         | Lung Cancer Deaths (n = 308) |        |         |         |
|--|----------------------------------|--------|---------|---------|------------------------------|--------|---------|---------|
|  | Mean                             | (SD)   | Minimum | Maximum | Mean                         | (SD)   | Minimum | Maximum |
| Year of birth                            | 1945                             | (12.4) | 1913    | 1974    | 1931                         | (7.6)  | 1917    | 1959    |
| <b>Mortality follow up</b>               |                                  |        |         |         |                              |        |         |         |
| Age at start of follow up (yr)*          | 34                               | (11.3) | 17      | 65      | 47                           | (7.5)  | 19      | 60      |
| Age at end of follow up (yr)†            | 51                               | (12.1) | 19      | 84      | 59                           | (8.0)  | 34      | 74      |
| Duration of follow up (yr)               | 17                               | (3.6)  | <1      | 19      | 12                           | (4.6)  | <1      | 19      |
| <b>Employment</b>                        |                                  |        |         |         |                              |        |         |         |
| Year at start of employment              | 1970                             | (10.1) | 1937    | 1994    | 1959                         | (8.2)  | 1943    | 1985    |
| Age at start of employment (yr)          | 25                               | (5.1)  | 15      | 59      | 29                           | (5.5)  | 19      | 52      |
| Duration of employment (yr)              | 23                               | (7.4)  | <1      | 45      | 26                           | (6.1)  | 4       | 40      |
| Proportion of work time as a lineman (%) | 85                               | (30)   | <1      | 100     | 93                           | (21)   | <1      | 100     |
| <b>Asbestos exposure‡</b>                |                                  |        |         |         |                              |        |         |         |
| Mean annual exposure (f/cc)§             | 0.05                             | (0.01) | 0.01    | 0.42    | 0.05                         | (0.01) | 0.03    | 0.06    |
| Cumulative exposure (f/cc-yr)            | 0.87                             | (0.54) | <0.01   | 12.53   | 1.13                         | (0.43) | 0.01    | 2.04    |
| Exposure duration (yr)                   | 17.0                             | (9.5)  | <0.1    | 42.0    | 22.0                         | (7.9)  | 0.3     | 37.0    |

\*Start of follow up: January 1, 1978, or date of start of employment if it occurred later.

†End of follow up: date of death or December 31, 1996, at the latest.

‡Among workers ever exposed to asbestos (30,625 in the total cohort and 305 lung cancer deaths). Exposure values are estimated as explained in the text.

§Excluding periods with no exposure to asbestos.

SD indicates standard deviation.

**TABLE 3**

Relative Risk of Lung Cancer Death According to Exposure Scores for Asbestos—Telephone Linemen Cohort Study, 1978–1996

|   | Exposure Percentile | Exposure Limits | Mean Exposure in the Exposure Group | No. of Lung Cancer Deaths | Relative Risk | 95% Confidence Interval |
|---|---------------------|-----------------|-------------------------------------|---------------------------|---------------|-------------------------|
| Cumulative asbestos exposure (f/cc-yr*) | 0% to 10%           | 0.00–0.50       | 0.36                                | 32                        | 1.0           | —                       |
|   | >10% to 25%         | >0.50–0.86      | 0.76                                | 48                        | 1.2           | 0.7–1.8                 |
|   | >25% to 50%         | >0.86–1.23      | 1.10                                | 76                        | 1.4           | 0.9–2.1                 |
|   | >50% to 75%         | >1.23–1.46      | 1.39                                | 75                        | 1.3           | 0.8–2.1                 |
|   | >75% to 95%         | >1.46–1.73      | 1.59                                | 61                        | 1.3           | 0.8–2.0                 |
|   | >95%                | >1.73           | 1.91                                | 16                        | 2.1           | 1.1–4.0                 |
| Mean annual asbestos exposure (f/cc*)   | 0% to 25%           | 0.00–0.05       | 0.048                               | 45                        | 1.0           | —                       |
|   | >25% to 75%         | >0.05–0.055     | 0.051                               | 187                       | 1.2           | 0.9–1.6                 |
|   | >75%                | >0.055          | 0.057                               | 76                        | 1.3           | 0.9–1.8                 |
| Exposure duration (yr)                  | 0% to 10%           | 0.0–10.0        | 7.5                                 | 35                        | 1.0           | —                       |
|   | >10% to 25%         | >10.0–17.1      | 15.2                                | 45                        | 1.0           | 0.6–1.6                 |
|   | >25% to 50%         | >17.1–23.5      | 21.5                                | 75                        | 1.4           | 0.9–2.1                 |
|   | >50% to 75%         | >23.5–27.8      | 26.6                                | 77                        | 1.2           | 0.8–1.9                 |
|   | >75% to 95%         | >27.8–32.3      | 30.3                                | 60                        | 1.1           | 0.7–1.8                 |
|   | >95%                | >32.3           | 33.7                                | 16                        | 1.6           | 0.9–3.0                 |

\*Number of fibers per cubic centimeter estimated from semiquantitative exposure indices as explained in the text.

significant. There was no indication of an association between occupational exposures to these carcinogens and lung cancer death.

**Discussion**

In this historical cohort study, lung cancer risk was investigated in tele-

phone linemen exposed to asbestos mainly from telephone cable installation in asbestos-insulated buildings. We found that the risk of lung cancer increased twofold in workers with an estimated cumulative exposure of approximately 2 f/cc-years, a low exposure value. A linear dose-

response relationship between cumulative exposure and lung cancer mortality was not apparent. Exposure to other lung occupational carcinogens did not confound the results.

The study was based on a large cohort of telephone linemen with a long duration of follow up (median

**TABLE 4**

Relative Risk of Lung Cancer Death According to Cumulative Exposure to Asbestos Allowing for Different Lag Time Periods Between Exposure and Death—Telephone Linemen Cohort Study, 1978–1996

| Lag Time | Cumulative Exposure (f/cc-yr*) | No. of Deaths | Relative Risk | 95% Confidence Interval |
|----------|--------------------------------|---------------|---------------|-------------------------|
| 5 yr     | 0–0.43                         | 34            | 1.0           | —                       |
|          | >0.43–0.77                     | 46            | 1.0           | 0.6–1.6                 |
|          | >0.77–1.11                     | 76            | 1.1           | 0.7–1.8                 |
|          | >1.11–1.38                     | 76            | 1.3           | 0.8–2.0                 |
|          | >1.38–1.72                     | 61            | 1.0           | 0.6–1.6                 |
| 10 yr    | >1.72                          | 15            | 2.3           | 1.2–4.5                 |
|          | 0–0.35                         | 38            | 1.0           | —                       |
|          | >0.35–0.62                     | 44            | 1.1           | 0.7–1.7                 |
|          | >0.62–0.93                     | 76            | 1.1           | 0.7–1.6                 |
|          | >0.93–1.23                     | 75            | 1.0           | 0.7–1.6                 |
| 20 yr    | >1.23–1.60                     | 60            | 0.9           | 0.6–1.4                 |
|          | >1.60                          | 15            | 1.4           | 0.7–2.7                 |
|          | 0–0.16                         | 69            | 1.0           | —                       |
|          | >0.16–0.32                     | 40            | 1.0           | 0.6–1.5                 |
|          | >0.32–0.57                     | 66            | 0.8           | 0.6–1.2                 |
|          | >0.57–0.88                     | 68            | 0.7           | 0.4–1.0                 |
|          | >0.88–1.26                     | 51            | 0.7           | 0.5–1.2                 |
| >1.26    | 14                             | 1.4           | 0.7–2.8       |                         |

\*Number of fibers per cubic centimeter estimated from semiquantitative exposure indices as explained in the text.

**TABLE 5**

Multivariate Analysis of Relative Risk for Lung Cancer Death According to Cumulative Exposure to Asbestos After Adjustment for Exposure to Arc Welding Fumes, Engine Exhaust, and PAHs—Telephone Linemen Cohort Study, 1978–1996

| Cumulative Exposure          | No. of Deaths | Relative Risk | 95% Confidence Interval |
|------------------------------|---------------|---------------|-------------------------|
| Asbestos (f/cc-yr*)          |               |               |                         |
| 0–0.50                       | 32            | 1.0           | —                       |
| >0.50–0.86                   | 48            | 1.4           | 0.8–2.5                 |
| >0.86–1.23                   | 76            | 1.6           | 0.8–3.0                 |
| >1.23–1.46                   | 75            | 1.4           | 0.7–2.9                 |
| >1.46–1.73                   | 61            | 1.3           | 0.6–2.8                 |
| >1.73                        | 16            | 2.1           | 0.9–5.3                 |
| Arc welding (yr of exposure) |               |               |                         |
| 0                            | 54            | 1.0           | —                       |
| >0–0.03                      | 127           | 1.2           | 0.8–1.6                 |
| >0.03–0.04                   | 64            | 1.3           | 0.8–2.2                 |
| >0.04                        | 63            | 1.4           | 0.7–2.8                 |
| Engine exhaust (ppm CO-yr†)  |               |               |                         |
| 0–20.4                       | 30            | 1.0           | —                       |
| >20.4–39.0                   | 124           | 0.8           | 0.4–1.5                 |
| >39.0–46.0                   | 77            | 0.8           | 0.4–1.7                 |
| >46.0                        | 77            | 0.7           | 0.3–1.5                 |
| PAH (unit-yr‡)               |               |               |                         |
| 0–1.8                        | 30            | 1.0           | —                       |
| >1.8–15.2                    | 124           | 0.7           | 0.4–1.1                 |
| >15.2–24.6                   | 77            | 0.7           | 0.4–1.2                 |
| >24.6                        | 77            | 1.0           | 0.5–2.0                 |

\*Number of fibers per cubic centimeter estimated from semiquantitative exposure indices as explained in the text.

†Estimated cumulative exposure to carbon monoxide in ppm-yr used as an indicator of exposure to engine exhaust fumes.

‡Cumulative exposure values using arbitrary exposure scores.

19 years), including over 300 lung cancer deaths. All workers were state-employed, representing a stable population in which the follow up in mortality registers was highly facilitated. Because we used two mortality registers, one national and one specific to the company, a crossvalidation could be carried out and virtually no worker was lost to follow up for mortality. Moreover, the causes of deaths were obtained from another national database for 98% of the deceased workers.

### Exposure Misclassification

Because measurement data of past occupational exposures were only very sparse or inexistent in the company, exposure to carcinogens was assessed using the expertise of company workers, occupational physicians and occupational hygienists with longstanding experience. These work groups rated the proportion of workers within each job who had executed well-defined work tasks and the frequency with which the work tasks were carried out. Three independent sources of information permitted an internal validation of the exposure assessment process, and a final consensus was obtained. Despite considerable effort to obtain valid exposure estimates, nondifferential exposure misclassification was likely to occur. To examine the effect of exposure classification on the results, we conducted sensitivity analyses in which jobs were considered as nonexposed to asbestos if the exposure probability and/or the exposure frequency were below different thresholds. However, the findings were not changed importantly (results not shown).

Besides employment in the company, exposure to asbestos may have occurred in other companies or outside work, but this information was not available. It is possible that some workers were exposed to asbestos in jobs held before, rather than after, their employment in the telephone company, because almost all workers leave the company at the age of

retirement. Although the unidentified sources of asbestos exposure outside the company may contribute to exposure misclassification, we believe that this problem is of relatively minor importance because most of the workers' careers were completed in the company. Moreover, these additional sources of exposure are most likely distributed equally across cases and noncases of lung cancer death, leading to nondifferential exposure misclassification.

### Confounding

Like most occupational cohort studies, individual data on tobacco smoking were not available in our study on telephone linemen. Confounding due to tobacco smoke occurs when a marked difference in the prevalence of tobacco smoking exists between compared exposure groups. It has been recognized that relative risks above 1.4 between occupation and lung cancer are unlikely due to uncontrolled confounding from tobacco smoking.<sup>12</sup> It has been demonstrated recently that systematic or chance differences in lifestyle habits such as tobacco smoking between exposure groups in occupational cohort studies is unlikely to cause a more than 20% change in relative risk.<sup>13</sup> In the present study, the observed number of lung cancer deaths in the cohort was slightly lower than the expected number based on national mortality rates (SMR = 0.94), indicating that the tobacco consumption in telephone linemen is similar or slightly lower than in the general population. In addition, because we carried out internal analyses within a group of socially homogeneous workers with identical lifestyles, the differences in prevalence of tobacco smoking between exposure groups should be modest, making a strong confounding effect very unlikely.

Confounding from other occupational lung carcinogens was investigated using the exposure data collected for the JEM. We selected exposure to polycyclic aromatic hydrocarbons

(PAH), arc welding fumes, and engine exhaust as potential confounders. For other lung carcinogens such as ionizing radiations, only a small number of subjects were exposed at low levels, and they were not introduced in the multivariate models. The adjustment for the potential confounders did not modify the relation between exposure to asbestos and lung cancer. In addition, none of these exposures was independently associated with lung cancer mortality in our data.

### Comparison With Previous Studies

Asbestos is not known to be a major occupational hazard in telephone linemen. These workers, however, may have been exposed during installation or maintenance of telephone cables in asbestos-insulated buildings or areas or when using protective equipment containing asbestos, eg, for welding. These activities are not specific to telephone linemen, and similar exposures in other occupations performing comparable work tasks may occur. We found a significantly twofold increased RR of lung cancer mortality for linemen in the highest category of cumulative exposure estimate. The average asbestos exposure in this group was estimated to be approximately 2 f/cc-years. The association between asbestos exposure and lung cancer was strengthened after allowing for a 5-year latency period, indicating that the most recent exposure was unimportant to cancer risk. These findings suggest a possible causal association between intermittent exposure to asbestos and lung cancer mortality.

Because cumulative exposure is the product of the mean annual exposure dose and the exposure duration, these later exposure parameters were studied separately. The variability of the mean annual exposure was small and did not permit a comparison between clearly contrasted exposure groups, but it is suggested

that lung cancer mortality rises slowly with mean annual exposure. For exposure duration, a more distinct, although not significant, pattern was observed in workers exposed more than 32 years. It is therefore possible that the conjunction of both a high exposure duration and a relatively high annual exposure is necessary for lung cancer risk to be apparent.

Our finding of an increased lung cancer mortality associated with cumulative exposure contrasts with risk predictions based on downward extrapolations from highly exposed occupational cohorts. In these studies, a wide range of exposure was observed, from 1 f/cc<sup>14</sup> to more than 250 f/cc.<sup>15</sup> These values are clearly distinct from the mean exposure of 0.05 f/cc estimated for all workers in the present study, although this value is based on experts' judgment. Based on the findings reported in the highly exposed cohorts, most authors assume a linear relationship between relative risk of lung cancer mortality and cumulative exposure to asbestos.<sup>7,16</sup> It can also be estimated from a literature review that 1-f/cc-year increase in exposure is associated with approximately 1% increase in RR.<sup>16,17</sup> Under this assumption, a RR of 1.02 should be observed for workers in the highest cumulative exposure group of our study, a value much lower than the RR of 2.1, which was actually observed in the present study.

Although it is possible that the exposure to asbestos is slightly underestimated in our study, it cannot fit the prediction for lung cancer mortality derived from studies in highly exposed populations. In these studies, asbestos was usually present permanently in the workers environment,<sup>16</sup> and hence the results provide information on lung cancer risk associated with continuous exposures only. Conversely, in the present cohort, a sporadic pattern of asbestos exposure exists. For example, exposure peaks may occur in workers installing telephone lines in asbestos-

insulated buildings but will not for the same task at other work places. It is therefore possible that peak exposures are the most etiologically relevant exposures for lung cancer in workers who are intermittently exposed to asbestos. Unfortunately, peak exposures could not be observed directly in the present study because of its retrospective design.

Several other studies in occupations with low exposure and lung cancer point to the same direction. In a population-based case-control study carried out in Sweden,<sup>18</sup> the cumulative exposure dose of 4 f/cc-years was associated with a RR of 1.9 (95% confidence interval = 1.3–2.7). In a cohort study conducted among gas and electricity utility workers in France,<sup>19</sup> an RR of 2.0 (95% confidence interval = 1.3–3.2) for lung cancer was reported for exposure as low as 8.1 f/cc-years. No further increase in risk was observed in higher exposure group. In another recent cohort study among pulp and paper industry workers,<sup>20</sup> the RR for lung cancer was 1.44 (95% confidence interval = 0.85–2.45) for exposure to asbestos greater or equal to 0.78 f/cc-years, and there was an indication of a dose-response relationship.

In the present study, it was suggested that lung cancer mortality in telephone linemen with low asbestos exposure may be higher than what can be suspected from previous studies in highly exposed workers. This finding was not likely due to uncontrolled confounding from tobacco smoking or from occupational carcinogens other than asbestos. The increased lung cancer mortality in the highest cumulative exposure group was possibly related to peak exposures to asbestos. This assumption requires confirmation, but it is potentially important because it may concern several occupational groups performing the same work tasks as telephone linemen and having simi-

lar low asbestos exposures. These findings provide additional support that even low levels of exposure to asbestos in occupational settings should be strictly avoided.

### Acknowledgments

The authors are indebted to the groups of experts who participated in the occupational exposure assessment process, in particular the MATCOM group: Dr Bernard Siano, Dr Martine Aucomte, Dr Annie Barreau, Dr Bruno Jahan, Dr Nicole Honorat, M. Marc Mougel, and Dr Maryse Ville-goureix; and the group of employees who provided information of invaluable interest on occupational exposures and work practices: Alain Jubeau, Michel Robert, Yves Le Gohalen, Charles Mauzale, Daniel Duclos, Robert Tressard, Philippe Perrault, Jean-Pierre Kolczik, Jacky Poingt, Alain Guérodé, Bernard Lerat, and Jean-Claude Martzloff. The authors also thank Alice Guéguen for her help in data analysis.

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