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PROPORTION OF LUNG CANCERS IN MALES, DUE TO OCCUPATION, IN DIFFERENT AREAS OF THE USA

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Occupational data from 5 case-control studies in the United States involving 2,973 male cases and 3,210 controls were analyzed to estimate the percentage of lung cancer attributable to well-known and suspected lung carcinogens. The studies were conducted in areas heterogeneous in terms of industrial activities. The percentage of lung cancers attributable to occupations entailing potential exposure to well-recognized carcinogens ranged, by study area, from 3 to 17%. The further inclusion of occupational groups with suspect carcinogenic exposures changed these estimates very little. Exclusion of data derived from next-of-kin interviews influenced the estimates of attributable risks, but not in a systematic fashion. The estimates also varied according to ethnic group, smoking status and birth cohort, with higher values in non-whites, non-smokers and among members of more recent birth cohorts. Possible errors in exposure classification, which may make these estimates conservative, are discussed.

Some occupational exposures are among the known causes of lung cancer, and, under certain circumstances or in specific geographic areas, their contribution to the total lung cancer burden may be substantial. In order to quantify the role of occupation in lung-cancer development and to examine its variability across several geographic areas, we analyzed data from 5 case-control studies of lung cancer conducted in the 1970s and 1980s in the United States (Blot *et al.*, 1980, 1982 and 1983; Correa *et al.*, 1984; Schoenberg *et al.*, 1987). Each study collected lifetime occupational histories, as well as information on smoking. We estimated the age- and smoking-adjusted proportion of lung cancers due to occupations associated with *recognized* and *suspected* lung carcinogens.

SUBJECTS AND METHODS

Study designs

The designs of the studies, carried out in the states of Louisiana, Florida, Pennsylvania, Virginia and New Jersey, are summarized in Table I. The study areas were originally selected because of high lung-cancer rates and/or because of the presence of certain industries. Subjects with a diagnosis of lung cancer resident in selected geographic areas of the respective states were identified from existing cancer registries, from co-operating hospitals or from death certificates. All the studies included at least some interviews with next-of-kin of the enrolled subjects. Next-of-kin were the only source of information in Pennsylvania and Virginia. Response rates for the studies ranged from 63 to 93%. Overall, 2,973 male cases and 3,210 male controls were included in the analyses. Distributions of the case and control samples according to demographic characteristics and smoking habits are shown in Table II.

Choice and coding of occupations

Information was collected by interview for each job held for 6 months or more, including the place and type of work and the duration of employment. In New Jersey, occupations and industries were coded according to the 1970 Census classification and to the Standard Industrial Classification, respectively (OMB, 1972). In Louisiana, the Dictionary of Occupational Titles (1977) was used. For the other study areas, study-specific job coding schemes were developed.

In a recent review, Simonato and Saracci (1983) classified industries/occupations with well-established carcinogenic exposures (List A) and industries/occupations with suspect carcinogenic exposures (List B). With minor modifications, we have adapted these categories for well-established and suspect lung carcinogens. List A industries or occupations (Table III) are generally associated with specific chemical exposures. List B (Table IV) includes mainly jobs or industrial activities in which an excess of lung cancer has been described in epidemiologic studies, but for which the degree and specificity of the associations with specific exposures are uncertain. Although coding schemes differed across the 5 studies, the industry/occupation dictionaries were used in a standard fashion to identify Lists A and B jobs. The codes were reviewed independently by three of us (P.V., T.T. and R.B.H.). A consensus was reached on all job-history categories except for 20 cases and 28 controls, with uncertain List A exposures, who were deleted from the analysis.

Analysis

Adjusted odds ratios (OR) were calculated using Gart's maximum-likelihood method (Martin and Pickle, 1980; Gart, 1970) as measures of the relative risk of lung cancer associated with ever-employment in the selected occupations. The odds ratios were adjusted for age, in 4 groups, birth cohort, in 4 groups, and usual cigarette use, in 5 groups (Table II). In 3 areas (Louisiana, New Jersey and Pennsylvania) it was possible to use 9 categories of smoking, but this did not materially affect the estimates. Ninety-five percent confidence limits were computed using Gart's maximum likelihood method (Gart, 1970). Population-attributable risks (PAR), adjusted for age, birth cohort and cigarette use, were estimated using the procedure of Whittemore (1983): $AR = 1 - (\Sigma_k y_k x_{2k}/n y_{2k})$, where n = total number of cases, y_k = number of controls in stratum k, x_{2k} and y_{2k} = number of unexposed cases and controls in stratum k.

RESULTS

Twenty-five per cent of the cases and 20% of the controls had ever held a job included in List A. Among those with List A jobs (Table III), the highest odds ratios (OR) were for boilermakers (1.8) and for asbestos production workers (2.7). Approximately 30% of both cases and controls had held a job included in List B. Among individual List B occupations, significantly elevated ORs were reported for talc miners, welders and leather workers (Table IV).

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TABLE I - STUDY DESIGNS OF THE 5 STUDIES INCLUDED IN THE ANALYSIS

State and year	Louisiana 1979–1983	Florida 1976–1979	Pennsylvania 1974–1977	Virginia 1976	New Jersey 1980–1981
Study base	Hospital-based in 29 parishes	Hospital-based and death certificates in four counties	Death certificates in three counties	Death certificates in one county	Cases ascertained through hospital records, cancer registry and death certificates in 6 areas
Controls and exclusions	Randomly selected from hospital patients—lung disease and tobacco related cancers excluded	Randomly selected from: a) hospital records and b) death certificates—lung and psychiatric diseases excluded	Randomly selected decedents—lung disease and suicide excluded	Randomly selected from death certificates	NJ licensed drivers, death certificates
Matching	Sex, age, hospital, ethnic group	Age, county, source of ascertainment	Sex, age, county, year of death	Ethnic group, age, year of death and county	Age, area of residence, ethnic group, vital status
Response rates	76% (cases) 89% (controls)	84% (cases) 84% (controls)	93% (cases) 82% (controls)	89% (cases) 82% (controls)	70% (cases) 63% (controls)
Number of cases and controls	Subject: 760/937 NOK: 294/134	Subject: 163/290 NOK: 178/166	Subject: 0/0 NOK: 335/332	Subject: 0/0 NOK: 347/308	Subject: 542/666 NOK: 378/377
References	Correa *et al.*, 1984	Blot *et al.*, 1982	Blot *et al.*, 1983	Blot *et al.*, 1980	Schoenberg *et al.*, 1987

NOK = next-of-kin.

Table V reports the population-attributable risks (PAR) by study area for occupations in List A and in List A or B. For jobs included in List A, estimates, by study area, were between 3 and 17%. Inclusion of List B jobs as exposed had no substantial effect on the PARs. The 95% upper confidence limits of the PARs ranged from 8 to 35%. The lower confidence limit for each PAR reached 0%. The high variability of the attributable risk among study areas suggests that it may be an oversimplification to produce a single estimate for all 5 study sites combined.

Table VI shows the ORs and PARs, for List A and List A or B according to respondent type. For New Jersey and Florida, both the ORs and PARs were higher for data collected in personal subject interviews, compared to next-of-kin interviews. For Louisiana, the opposite occurred.

Race and smoking status influenced the estimates (Table VII). Non-whites tended to have higher ORs and population-attributable risks than did whites. Non-smokers consistently showed higher ORs than did smokers, with the PAR as high as 42% for non-smokers in Pennsylvania. However, the numbers of non-smokers were small. An effect of birth cohort seemed to be present (Table VIII), since higher attributable risks were found in people born in 1930 or later.

TABLE II - SELECTED CHARACTERISTICS OF THE STUDY POPULATION

	Cases		Controls	
	Number	%	Number	%
Cigarette use[1]				
Never	111	4	783	24
<1/2 pack/day	198	7	376	12
1/2–2	2.177	73	1.748	54
>2	435	14	256	8
Unknown	52	2	47	2
Birth cohort				
<1910	888	30	950	30
1910–1919	1.129	38	1.193	37
1920–1929	726	24	786	24
1930+	230	8	281	9
Ethnic group				
White	2.339	78	2.526	79
Non-white	644	22	684	21
Interview type				
Subject	1.444	49	1.893	59
Next-of-kin	1.529	51	1.317	41
Total	2.973	100	3.210	100

[1]In 3 studies (Louisiana, New Jersey and Pennsylvania) more detailed information for smoking was available.

The occupations showing the highest ORs among non-smokers were all asbestos-related, including asbestos production workers, pipefitters, boilermakers and shipyard workers. For these occupations combined, the OR among smokers was 1.4 (1.1–1.9), and among non-smokers 2.4 (0.9–6.8), considering data from personally interviewed subjects only. The corresponding PARs were 5.6% and 15.9%.

DISCUSSION

The population-attributable risk is a function of the risk of disease associated with exposure, as here measured by odds ratio, and the proportion of exposed subjects in population, as here estimated by the proportion of exposed controls. Differences in PARs among study areas are due to variability in either of these measures. The proportion of controls who ever held any of the occupations in List A was 14% in Louisiana, 27% in Virginia, 20% in New Jersey, 13% in Florida, and 34% in Pennsylvania. These results primarily reflect differences in industry and occupation patterns between the study areas. In fact, the study areas were initially selected in part because of their employment patterns. Some further differences in occupational patterns may be associated with the study group sources and inclusion criteria. Only the control series in New Jersey was population based. Controls selected from hospital patients and among the deceased may be over-represented with subjects who have held hazardous occupations. This possibility is, however, not supported by available data (McLaughlin *et al.*, 1985). The insignificant contribution of suspect exposures in List B is probably due, at least in part, to the uncertainties of exposure classification for occupations in which the actual carcinogens have not been well characterized.

Variation in the odds ratio for industry/occupation groupings among study areas may be due to a number of factors. The distribution of jobs within occupational groups may vary from area to area. For example, the increased risk associated with List A jobs for Virginia reflects in large part exposures in shipyards, while for Pennsylvania the excess risk mainly reflects exposures in steel production. The other study areas also have unique occupational distributions.

Carcinogenic exposures associated with the same industries and occupations may be more prevalent or intense in some study areas. Such differences could be due to variation in

TABLE III – OCCUPATIONS IN LIST A: CASES/CONTROLS, ODDS RATIOS AND 95% CONFIDENCE LIMITS

	All subjects		
	Case/control	OR[1]	(95% CL)
Vineyard workers	—[2]	—	—
Roofers/asphalt workers	45/37	1.4	(0.9–2.3)
Coke plant workers	1/2	0.7	(0.02–21.2)
Gas workers	—	—	—
Asbestos production workers	9/4	2.7	(0.7–12.0)
Insulators	15/20	0.9	(0.4–2.0)
Shipyard/dockyard workers	307/254	1.4	(1.1–1.7)
Steamfitters	44/41	1.2	(0.8–2.0)
Boilermakers	26/17	1.8	(0.9–3.7)
Pipefitters	53/51	1.2	(0.8–1.8)
Arsenic, iron ore, asbestos, uranium miners	13/12	1.3	(0.5–3.3)
BCME/CMME or chromate pigment production workers	—	—	—
Mustard gas production workers	—	—	—
Copper smelter workers	—	—	—
Chromium plating workers	1/1	0.8	(0.02–20.1)
Ferrochromium production workers	—	—	—
Steel production workers	181/166	1.2	(1.0–1.6)
Nickel refining workers	—	—	—
Ferrous foundry workers	16/10	1.2	(0.5–3.0)
Demolition workers	3/3	0.8	(0.1–5.2)
Locomotive building and repair workers	21/21	1.0	(0.5–2.0)
Automobile brake workers	98/90	1.2	(0.9–1.7)
Non-exposed[3]	1,383/1,662	1.0	—

[1]Adjusted for age, birth cohort, and smoking.–[2]No cases or controls.–[3]Not included in either List A or List B.

industrial processes and in the condition of plant facilities. We, however, had no direct data on this aspect of exposure.

Sampling error could well account for the variability in odds ratios across the study areas, since confidence intervals for the ORs for the 5 study areas overlap considerably. The precision of statistical estimates of population-attributable risks involve further uncertainty, as statistical variability in the odds ratio and in the estimate of the proportion of the population exposed must be considered in the calculation of confidence intervals for the PAR (Whittemore, 1983).

TABLE IV – OCCUPATIONS IN LIST B: CASES/CONTROLS, ODDS RATIOS AND 95% CONFIDENCE LIMITS

	All subjects		
	Case/control	OR[1]	(95% CL)
Pesticide applicators	8/6	2.1	(0.5–8.5)
Aluminum production workers	4/8	0.5	(0.1–1.9)
Beryllium refining workers	—[2]	—	—
Smelter (other than copper) workers	8/15	0.6	(0.2–1.5)
Butchers	35/50	0.8	(0.5–1.3)
Painters	201/193	1.1	(0.9–1.4)
Welders	148/127	1.3	(1.0–1.8)
Oilfield workers	0/4	—	—
Laundry and dry cleaning workers	68/78	1.0	(0.7–1.4)
Zinc-lead miners	1/0	—	—
Talc miners	17/8	2.8	(1.0–7.7)
Truck drivers (other than delivery and routemen)	433/438	1.1	(0.9–1.3)
Printing pressmen	15/26	0.7	(0.3–1.3)
Machine room workers in printing	—	—	—
Rubber workers	26/27	1.2	(0.7–2.2)
Leather workers	42/29	1.8	(1.0–3.1)
Pesticide production workers	4/4	1.4	(0.3–8.2)
Man-made mineral fiber producers	—	—	—
Chimney sweeps	—	—	—
Cadmium workers	—	—	—
Non-exposed[3]	1,383/1,662	1.0	—

[1]Adjusted for age, birth cohort and smoking.–[2]No cases or controls.–[3]Not included in either List A or List B.

In a study of this type, misclassification of exposure may have an important impact on the estimated ORs. Random misclassification of exposure would tend to bias ORs toward 1, *i.e.*, no effect (Copeland *et al.*, 1977). Next-of-kin probably do not report as complete or detailed occupational histories as are obtained from interviews of the subjects themselves. Indeed, in these data the number of jobs reported from next-of-kin interviews was somewhat less, on average, than that reported in the first-person interviews. Prior comparisons (Pickle *et al.*, 1983) showed that the number of jobs reported also varied by type of respondent. The mean number of jobs held as reported by subjects, subjects' wives, their children, siblings and other respondents was 8.6, 6.3, 5.4, 4.4 and 4.2, respectively. Although usual occupation reported by next-of-kin is generally comparable to usual occupation reported by subjects (Blot and McLaughlin, 1985), the shorter lists of jobs from next-of-kin interviews would reduce the opportunity for categorization as ever-employed in List A or List B jobs. As this under-reporting probably affects cases and controls equally, the net effect would be to lower the OR. Comparing the results for direct first-person interviews with those for next-of-kin interviews in the present study, the ORs were lower for next-of-kin interviews from New Jersey and Florida, while for Louisiana they were higher.

The need to attribute occupational exposures from historical job titles is a common limitation of occupational case-control studies, in which little or no information is generally available about on-the-job exposure (Gerin *et al.*, 1985). Even though a subject worked in a shipyard or steel mill, we cannot be certain whether and to what extent he was exposed to asbestos, polycyclic aromatic hydrocarbons, and other lung carcinogens.

Misclassification could also have occurred due to the use of different coding schemes in the study areas. Although the sensitivity and specificity of exposure classification will vary depending upon the level of detail provided in the occupational coding scheme, our panel review of the different coding systems used in the studies helped to standardize categorization

TABLE V – OCCUPATIONS HELD IN LIST A AND LIST A OR B: EXPOSED CASES/CONTROLS, ODDS RATIOS (OR) AND ATTRIBUTABLE RISK IN THE POPULATION (PAR)

	Louisiana	Virginia	New Jersey	Florida	Pennsylvania
List A					
Exposed cases/controls	173/154	110/83	238/211	86/87	144/114
OR[1]	1.2	1.3	1.4	1.4	1.4
95% CI	(0.9–1.5)	(0.9–2.0)	(1.1–1.8)	(1.0–2.1)	(0.9–2.1)
PAR[1]	3.0	10.0	11.0	10.0	17.0
95% upper confidence limit of PAR	8	23	19	20	34
List A or B					
Exposed cases/controls	440/390	192/152	541/566	176/215	222/197
OR[1]	1.2	1.3	1.2	1.1	1.4
95% CI	(1.0–1.4)	(0.9–1.8)	(1.0–1.4)	(0.8–1.5)	(0.9–2.0)
PAR[1]	6.0	9.0	9.8	6.1	17.3
95% upper confidence limit of PAR	13	25	20	20	35

[1]Adjusted by smoking status, age, and birth cohort.

TABLE VI – OCCUPATION IN LIST A AND LIST A OR B BY TYPE OF INTERVIEW. ODDS RATIO (OR) AND ATTRIBUTABLE RISK IN THE POPULATION (PAR)

Type interviewed	Louisiana	New Jersey	Florida
List A			
Living subjects			
Cases/controls	126/138	149/137	45/61
OR (95% CI)	1.2 (0.9–1.6)	1.7 (1.2–2.4)	1.8 (1.1–3.3)
PAR	3.8	18.7	18.3
Next-of-kin			
Cases/controls	47/16	89/74	41/26
OR (95% CI)	1.6 (0.8–3.8)	1.1 (0.7–1.7)	1.5 (0.7–3.0)
PAR	7.7	3.1	9.8
List A or B			
Living subjects			
Cases/controls	328/353	338/380	98/139
OR (95% CI)	1.2 (1.0–1.5)	1.3 (1.0–1.6)	1.6 (1.0–2.5)
PAR	7.2	15.1	22.8
Next-of-kin			
Cases/controls	112/37	203/186	78/76
OR (95% CI)	1.4 (0.8–2.4)	1.1 (0.8–1.5)	0.8 (0.5–1.3)
PAR	10.8	4.9	—

TABLE VII – OCCUPATION IN LIST A BY ETHNIC GROUP AND SMOKING STATUS. ODDS RATIOS (OR) AND ATTRIBUTABLE RISK IN THE POPULATION (PAR)

	Louisiana	Virginia	New Jersey	Florida	Pennsylvania
Ethnic group					
Non-whites					
Cases/controls	42/45	27/23	37/32	15/16	—
OR (95% CI)	1.1 (0.6–1.9)	1.4 (0.6–3.6)	1.9 (0.9–4.6)	1.8 (0.7–5.4)	—
PAR	4.4	15.0	21.9	21.5	—
Whites					
Cases/controls	131/109	83/60	201/179	71/71	—
OR (95% CI)	1.2 (0.9–1.7)	1.3 (0.7–2.1)	1.3 (1.0–1.8)	1.4 (0.8–2.3)	—
PAR	3.9	8.9	9.1	10.2	—
Smoking status[2]					
Non-smokers					
Cases/controls	3/29	8/16	10/52	5/19	4/23
OR (95% CI)	1.4 (0.3–5.8)	1.8 (0.5–6.5)	1.5 (0.6–4.0)	1.9 (0.5–8.9)	2.7 (0.4–24.8)
PAR	4.3	19.7	12.3	19.7	42.0
Smokers					
Cases/controls	170/125	102/67	228/159	81/68	140/91
OR (95% CI)	1.2 (0.9–1.5)	1.3 (0.8–1.9)	1.4 (1.0–1.8)	1.4 (0.9–2.1)	1.4 (0.9–2.1)
PAR	3.7	8.3	11.6	9.8	16.0

[1]Estimates adjusted by age, birth cohort and smoking.–[2]Estimates adjusted by birth cohort and age.

TABLE VIII - OCCUPATION IN LIST A AND LIST A OR B BY BIRTH COHORT. ODDS RATIOS (OR) AND ATTRIBUTABLE RISK IN THE POPULATION (PAR)

Birth cohort	List A	List A or B
< 1930		
Exposed cases/controls	706/609	1,446/1,398
OR	1.3	1.3
95% CI	1.1–1.6	1.1–1.4
PAR	10.9	8.8
1930+		
Exposed cases/controls	45/40	125/122
OR	1.9	1.8
95% CI	1.0–3.7	1.2–2.9
PAR	14.3	24.2

of jobs in Lists A and B.

Contrary to the effects of random misclassification as described above, response bias, in the form of more accurate or detailed reporting from cases or controls, could either increase or decrease ORs. A major impact of such differential misclassification seems unlikely, since the total number of jobs reported was similar for cases and controls. Such response bias is more likely when specific chemical exposures, rather than occupations, are ascertained by interview. Furthermore, the odds ratios associated with List A occupations were generally lower than those reported from other published studies. Low risks for well-known carcinogenic exposures are more consistent with random misclassification, *i.e.*, an error in exposure assessment evenly distributed among the cases and the controls, than with differential response bias.

PARs for List A occupations were higher for non-smokers than for smokers. This was due to higher ORs for occupation among non-smokers, and not to a higher proportion of occupationally exposed subjects. Because of the small number of non-smokers among lung-cancer cases, these differences must, however, be interpreted cautiously. A statistical test for interaction between the risk associated with cigarette use and with ever-employment in List A occupations was not significant ($p = 0.14$).

Non-whites had higher attributable risks for List A occupations, mainly due to higher ORs. Although the ethnic differences were not large, the findings raise the possibility that our exposure categories imply higher occupational exposures for non-whites than for whites. As Table VIII shows, subjects born in 1930 or after had higher ORs and PARs both for List A and List A or B jobs. This could be due to better recall and more accurate information for this group, or due to different exposures introduced in more recent years.

Doll and Peto (1981) have suggested that 15% of lung cancers in the United States may be due to occupational exposure. Our data are consistent with this overall estimate, although the PARs in this study varied considerably according to geographic area. One advantage of our estimates is that they are based on empirical data from studies containing information about confounding variables and effect modifiers.

In highly industrialized areas of Norway and northern Italy, the population-attributable risk was as high as 30–40% (Kjuus *et al.*, 1986; Pastorino *et al.*, 1984). Clearly, such small and homogeneous areas with heavy industrialization may yield PARs which are higher than those found for the entire US or for the diverse locations included in the present analysis. PAR estimates may also be higher from studies which rely on direct exposure assessment, as in the Norwegian and Italian studies, rather than on indirect categorization based on job titles (Vineis *et al.*, 1986).

Even if our estimate that 3 to 17% of lung cancers in the study areas are related to exposure to recognized occupational carcinogens is conservative, the number of lung cancers induced by occupational exposures would be large, since lung cancer is the most common cancer in the United States, with nearly 100,000 new cases expected among males in 1987 (Silverberg and Lubera, 1987). The estimates of 3 to 17% refer only to well-known carcinogenic exposures. Estimating the contribution of other, still suspected or unknown exposures, is more uncertain. Additional associations could only add to the calculated population-attributable risk.

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