Exhibit 255

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ORIGINAL ARTICLE

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A case-control study of cancer mortality at a transformer-assembly facility

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Summary To address earlier reports of excess cancer mortality associated with employment at a large transformer manufacturing plant, each plant operation was rated for seven exposures: Pyranol (a mixture of polychlorinated biphenyls and trichlorobenzene), trichloroethylene, benzene, mixed solvents, asbestos, synthetic resins, and machining fluids. Site-specific cancer deaths among active or retired employees were cases; controls were selected from deaths (primarily cardiovascular deaths) presumed to be unassociated with any of the study exposures. Using job records, we then computed person-years of exposure for each subject. All subjects were white males. The only unequivocal association was that of resin systems with lung cancer (odds ratio = 2.2 at 16.6 years of exposure, $P = 0.001$, in a multiple logistic regression including asbestos, age, year of death, and year of hire). Certain other odds ratios appeared larger, but no other association was so robust and remained as distinct after considering the multiplicity of comparisons. Study power was very limited for most associations, and several biases may have affected our results. Nevertheless, further investigation of synthetic resin systems of the type used in the study plant appears warranted.

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Introduction

A preliminary proportionate mortality study undertaken by the Massachusetts State Division of Occupational Hygiene in the late 1970s showed a small excess of leukemia and colorectal cancer among Pittsfield, Mass., residents for whom General Electric's name was entered as employer on the death certificate. It was later decided to reevaluate these findings by undertaking a mortality study based on a historical reconstruction of exposures at the plant. During the period covered by this study there were three basic manufacturing divisions at the General Electric facility in Pittsfield: transformers (TD), plastics (PD), and ordnance systems (OS). As of 1982 there were about 3500 workers in TD, 600 in PD, and 3500 in OS. During the mid-1980s the transformer division was phased out.

Although the company did not have the records for a historical reconstruction of the cohort of workers, it did provide a list of insurance pension records for those employee deaths which occurred in 1969 or later and which had resulted in death benefit claims. It was decided to construct a job-exposure matrix and combine this with the work history database to produce a history of job exposures for each worker in the study. A case-control approach was followed: relative-risk estimates were constructed from the exposure histories of cancer cases and a control group consisting of workers who died of causes thought to be unrelated to the exposures under study.

Materials-use records and industrial hygiene records were of limited use for the exposure assessment task since they did not extend far enough back in time. Through interviews with long-term management employees, selected for their historical knowledge of the plant operations, more than 250 chemicals and classes of chemicals were identified as being used at the plant. A toxicological re-

view identified 30 chemicals with possible mutagenic or carcinogenic potential. From these, seven exposures were selected for job exposure rating based on their carcinogenic potential, the quantity of the material used, and the number of operations where the material was used. These exposures were:

1. Pyranol: A transformer oil used from 1936 to 1976, nominally composed of 50% polychlorinated biphenyls (PCBs) (a mixture of isomers but mostly hexachlorobiphenyl), 50% trichlorobenzene (or a mixture of tri- and tetrachlorobenzene), less than 0.25% phenoxypropene oxides and trace amounts of dibenzofurans. The PCB content in Pyranol could in fact vary from 45% to 80%.

2. Benzene: Used from about 1920 to 1950 in various departments for general cleaning during machining and assembly operations.

3. Trichloroethylene (TCE): Used from about 1930 to 1977 as a degreaser.

4. Other solvents: This group includes Varsol (petroleum spirits), CPE 1000 (petroleum spirits and methylene chloride), methylene chloride, kerosene, paint thinners (primarily xylene or toluene based), solvent-based paints, xylene, toluene and naphtha. Some type of solvent exposure occurred in the majority of plant operations from the opening of the plant (1901) to the end of this study (1984).

5. Machining fluids: Used for machining and grinding operations. Straight cutting (mineral) oils predominated before 1940; thereafter soluble fluids were predominant. Synthetic fluids were introduced in the 1970s.

6. Asbestos: Used from 1940 to 1975 in wet insulation blankets during brazing and welding. Some insulation pieces were made from asbestos. Also used as powdered additive in some resin operations (this component of asbestos exposure was not rated).

7. Resin systems: Used from 1936 through the end of the study (1984); primarily phenol formaldehyde and polyvinyl formal resin systems.

Several other exposures were not rated by the employees and so were not analyzed: mineral oil (10w oil), used as transformer oil; metal fumes and dust (exposure occurred during welding, brazing, and painting with metal-based pigments); sawdust in woodworking shops; water-based, solvent-based, and epoxy adhesives (the solvent component was rated in group 4 above); and electromagnetic fields.

Materials and methods

Subjects for the analysis were deceased facility employees who met all the following criteria: (1) employment at the facility before 31 December 1984; (2) date of death in the period 1969–1984 (no pension records were available for employees who died before 1969); (3) death reported to and recorded by the company pension office (benefits were available to next-of-kin of employees vested in the pension fund, and next-of-kin of employees who died on the job); (4) possession of a job history record available for exposure rating.

Table 1 Number of subjects by outcome and job-history availability

Outcome	Job history			
	Available		Not available	
	Eligible[a]	Total	No.	%
Orolx[b]	21	21	10	32
Esophagus	13	13	6	32
Stomach	19	19	8	30
Colon	60	60	22	27
Rectum	32	32	4	11
Livbil[b]	9	10	2	17
Pancreas	33	34	11	24
Lung	139	142	68	32
Prostate	58	60	26	30
Bladder	20	22	12	35
Kidney	12	12	4	25
Lymphomas[b]	15	16	5	24
Leukemias	22	23	17	43
Brainp[b]	16	16	9	36
Other cancers	53	53	24	31
Total cases[c]	512	523	220	30
Control causes	1202	1270	719	36
Excluded causes	107	118	68	37
Totals	1821	1911	1003	34

[a] Meeting restrictions given in text
[b] *Abbreviations:* Orolx, oral, laryngeal, pharyngeal; Livbil, liver, gallbladder, and biliary tract; Lymphomas, lymphosarcomas, reticulosarcomas; Brainp, malignant and unspecified brain tumors
[c] Total cases is less than total cancers because ten available and eight unavailable subjects had two primary cancers

Subjects were restricted to white males because there were too few nonwhites or females to allow analytic control for race or sex. Vesting requirements for company workers varied over time, but for most of the study period vesting required 10–15 years' employment with the company. The size of the underlying employee cohort was unknown because work history records did not exist for a large fraction of former employees, especially in the earlier years of death. This lack of histories arose primarily from routine disposal of records over time, along with some misfiling.

After initial data description, the following further restrictions were imposed on the 1911 subjects meeting the above criteria: (1) to eliminate concerns with confounding or diagnostic error at extremes of age, only deaths at ages 21–90 were analyzed; (2) all subjects but one stopped work at the facility in 1946 or later (the single exception, who retired in 1932, was excluded from the analysis); (3) the 36 subjects for whom more than 50% of their work history was unrated for Pyranol exposure were excluded from the analysis. Of the 1821 subjects remaining after these exclusions, those with incomplete ratings had their exposures in unrated periods imputed from a time-weighted average of exposures in rated periods. However, less than 2% of the 51063 person-years of employment had their exposure level assigned by imputation. Subjects also accumulated 16432 person-years from retirement to death. Table 1 displays the distribution of deaths according to job history availability, and the distribution of deaths meeting the final restrictions among those with an available job history. The latter deaths were the subjects for our analysis. (Ten included subjects

were listed as having two primary cancers at death and were thus counted in two site-specific groups; thus the total of the site-specific numbers exceeds the total number of cases, which is 512.)

A subset of the death-certificate diagnoses was validated. To reduce the false-positive rate, we requested hospital records for those cancer diagnoses that were reported to have less than a 90% confirmation rate in a study by Percy et al. [1]. Among the 1911 subjects with job history, 257 required validation; 75% of the validation inquiries yielded responses. Among the responses, 94% confirmed or adjusted the cancer diagnosis from the death certificate, with 87% of diagnoses validated by means such as histology, imaging, surgery, or autopsy. Individual diagnoses were corrected according to the validation substudy. Further details have been reported elsewhere [2, 3].

Eligible controls were noncancer deaths from the same underlying cohort as the cases, with the exclusion of certain diagnoses based on their possible associations with the exposure under study [4]: diseases of blood and blood-forming organs (ICD8 280–289), because of the benzene exposure; mental disorders (290–315), because of the exposure to benzene, TCE, and other solvents; diseases of the digestive system (520–577), because of the exposure to PCBs and their reported effects on liver metabolism; genitourinary diseases (580–629), because of the solvent exposure; and ill-defined conditions. One hundred and seven noncancer deaths were thus excluded from the control group. The remaining 1202 controls were 78% circulatory, 10% respiratory, 6% injury, and 6% other causes of death.

The work histories of cases and controls generated over 1000 job titles from 50 separate departments occupying approximately 100 buildings. The job exposure matrix had over 5500 entries; each was identified by a combination of job title, department, and building. In a series of on-site interviews with 18 long-term knowledgeable employees, our industrial hygienist (M.F.H.) obtained ratings of each matrix entry for seven selected exposures from 1901 to 1984. Initially, a four-point categorical rating scale was used to rate jobs. Wherever the experience of the employees overlapped, multiple ratings of jobs were obtained. If the ratings differed by no more than one rank, they were averaged and rounded to the nearest integer; if the difference was larger, a further assessment was made to resolve the conflict. The overall agreement of employees was predominantly good to excellent, depending on the exposure being rated. Further details of the exposure assessment and interview procedure have been presented elsewhere [5].

For analysis of rated jobs, some exposure-rating categories were combined, depending on the nature of the exposure and the numbers of jobs in each category. For Pyranol, benzene, and solvents, the analysis categories were: 0 = no exposure; 1 = indirect exposure (chemical in work area but worker does not perform tasks using it); 2 = direct exposure. For TCE, asbestos, resins, and machining fluids, the categories were: 0 = no exposure; 1 = any exposure. For each individual, various cumulative exposure scores were computed by applying these ratings to the individual job histories. Results presented below were based on

score = years at level 2

for Pyranol, benzene, and solvents, and

score = years exposed

for the other four exposures. Each score was lagged by 2 years, so that exposures accumulated in the last 2 years before death could not contribute to the score. This was done to limit the counting of exposures after onset of the disease that led to death.

For each exposure score and cancer site involving more than eight cases, we examined the crude and age-stratified contingency table of the two variables. We also computed the Mantel trend test [6], both unstratified and stratified on potential confounders, such as age. Various exposure score-cancer pairs were subjected to further contingency table analyses, including stratification on other exposure scores. Cancer sites involving four to eight cases were screened using crude tables; sites involving fewer than four cases were not examined. We also examined binary indicators of exposure. For all but solvents level 1, these were coded 1 = ever exposed, 0 = never exposed. Because most subjects had at least several years of solvents level 1 exposure, the solvent level 1 indicator was coded 1 = over 20 years, 0 = 20 or fewer years of exposure.

Table 2 Summary of covariate distributions (means and standard deviations in years)

	Cases		Controls	
	Mean	SD	Mean	SD
Year of birth	1909	12	1907	12
Age at death	68	12	70	12
Year of death	1978	4	1978	4
Year of hire	1937	14	1935	15
Year stopped work	1968	8	1968	8
Duration of employment (leaves excluded)	27	10	28	10
Time from retirement to death	8	8	9	8
Foreign born	18%		15%	
Vested 5 years before death	93%		92%	
Employed 5 years before death	44%		37%	

SD, Standard deviation

For most analyses, certain sites with few cases were combined based on the assumption that any carcinogenic effect of the exposures should be similar at these sites because of anatomic proximity, tissue similarity, similarity of exposure routes, or similarity of diagnostic categories: liver, gallbladder, and biliary cancers (ICD8 155–156) were combined into a single category, "livbil"; buccal pharyngeal, and laryngeal cancers were combined into a single category, "orolx" (ICD8 140–149, 161); malignant and unspecified brain tumors were combined into a single category, "brainp" (ICD8 191–192, 238); all lymphosarcomas and reticulosarcomas were combined into a single category, "lymphomas" (ICD8 200–202); and all leukemias were combined (ICD8 204–207). We also performed tabular analyses on the separate component sites as well; these are reported in detail elsewhere [2, 3].

Computations were carried out using the EGRET software package [7], which implements analyses described in Breslow and Day [6]. Age and death year were entered in all regressions displayed below, and other covariates were entered when their inclusion altered an estimate by more than 20%. As shown in Table 2, there were only small differences between cases and controls with respect to measured potential confounders, so that for the most part adjustment made little difference.

We also applied several multiple-comparisons techniques to our results in order to obtain an overall summary of the degree to which our findings fall within chance expectations, including *P*-value plots [8] and empirical-Bayes methods [9, 10]. The results of these analyses are reported elsewhere [2, 3, 10].

Results

Contingency table analyses

Most of the stratified data analyses involved fine categorizations of covariates and multiple-level exposure categorizations. Since the purely categorical analyses led to the same results as the regression analyses with dichotomous (exposed/not exposed) exposures, we present results from the regressions only. Also, because level 1 (indirect exposure) for Pyranol, benzene, and solvents showed no discernible associations with any outcomes, we combined

Table 3 Logistic regression estimates obtained using binary exposures (exposure = 1 if any time spent above level 1)

	Pyranol	Benzene	Solvents
	Odds ratio (95% limits)	Odds ratio (95% limits)	Odds ratio (95% limits)
Orolx[a]	1.12 (0.38, 3.36)	1.03 (0.30, 3.58)	5.32 (1.54, 18.3)*
Esophagus	0.90 (0.20, 4.12)	1.23 (0.26, 5.72)	3.60 (0.94, 13.8)
Stomach	0.89 (0.26, 3.08)	0.32 (0.04, 2.42)	0.78 (0.31, 1.93)
Colon	0.63 (0.28, 1.41)	0.74 (0.33, 1.66)	1.09 (0.65, 1.85)
Rectum	0.88 (0.33, 2.31)	0.85 (0.29, 2.47)	0.51 (0.25, 1.06)
Pancreas	1.05 (0.43, 2.59)	0.58 (0.18, 1.93)	0.61 (0.30, 1.24)
Livbil[a]	2.40 (0.59, 9.71)	2.76 (0.68, 11.2)	0.69 (0.18, 2.60)
Lung	0.99 (0.62, 1.58)	0.58 (0.31, 1.07)	1.57 (1.08, 2.27)
Prostate	0.80 (0.37, 1.71)	1.02 (0.49, 2.12)	0.84 (0.49, 1.42)
Bladder	0.53 (0.12, 2.29)	1.02 (0.29, 3.51)	1.21 (0.49, 2.98)
Kidney	0.43 (0.06, 3.35)	4.29 (1.33, 13.8)	1.64 (0.49, 5.50)
Lymphomas	3.26 (1.14, 9.32)*	1.00 (0.22, 4.53)	1.97 (0.65, 5.95)
Leukemias	0.48 (0.11, 2.05)	0.90 (0.26, 3.08)	1.26 (0.53, 2.99)
Brainp[a]	1.09 (0.31, 3.88)	2.11 (0.66, 6.73)**	2.65 (0.84, 8.36)

* $P > 0.40$ by Mantel trend test; ** $P < 0.01$ by Mantel trend test

[a] *Abbreviations:* Orolx, Oral cavity, larynx, and pharynx; Livbil, liver, gallbladder, and biliary tract; Brainp, brain and unspecified brain tumors

Table 4 Logistic regression estimates obtained using binary exposures (exposure = 1 if any time spent above level 0)

	TCE	Asbestos
	Odds ratio (95% limits)	Odds ratio (95% limits)
Orolx	1.26 (0.51, 3.08)	0.83 (0.32. 2.15)
Esophagus	0.95 (0.29, 3.17)	1.87 (0.62, 5.65)
Stomach	0.70 (0.25, 1.95)	[a]
Colon	0.83 (0.47, 1.46)	0.89 (0.50, 1.58)
Rectum	0.78 (0.35, 1.69)	1.27 (0.61, 2.64)
Pancreas	1.64 (0.82, 3.29)	0.80 (0.37, 1.75)
Livbil	0.54 (0.11, 2.63)	1.15 (0.28, 4.65)
Lung	1.01 (0.69, 1.47)	1.10 (0.76, 1.60)
Prostate	0.82 (0.46, 1.46)	1.02 (0.57, 1.83)
Bladder	0.85 (0.32, 2.23)	0.93 (0.35, 2.47)
Kidney	0.99 (0.30, 3.32)	2.99 (0.94, 9.56)*
Lymphomas	0.76 (0.24, 2.42)	1.88 (0.68, 5.25)
Leukemias	1.10 (0.46, 2.66)	0.83 (0.32, 2.15)
Brainp	0.93 (0.32, 2.69)	1.61 (0.59, 4.37)

* $P = 0.01$ by Mantel trend test

[a] Stomach cancer excluded because there were no exposed cases

Table 5 Logistic regression estimates obtained using binary exposures (exposure = 1 if any time spent above level 0)

	Resins	Machining fluids
	Odds ratio (95% limits)	Odds ratio (95% limits)
Orolx	1.42 (0.55, 3.71)	0.98 (0.41, 2.36)
Esophagus	1.66 (0.51, 5.48)	0.96 (0.31, 2.96)
Stomach	0.44 (0.10, 1.90)	1.22 (0.48, 3.14)
Colon	0.85 (0.43, 1.65)	0.74 (0.44, 1.24)
Rectum	1.02 (0.43, 2.38)	0.92 (0.45, 1.88)
Pancreas	0.36 (0.11, 1.20)	0.75 (0.37, 1.49)
Livbil	[a]	0.86 (0.23, 3.24)
Lung	1.72 (1.17, 2.52)*	0.86 (0.60, 1.23)
Prostate	0.55 (0.24, 1.22)	0.90 (0.53, 1.54)
Bladder	0.41 (0.09, 1.78)	1.27 (0.50, 3.21)
Kidney	1.20 (0.32, 4.48)	2.10 (0.56, 7.83)*
Lymphomas	1.34 (0.42, 4.26)	1.19 (0.41, 3.43)
Leukemias	0.59 (0.17, 2.02)	1.24 (0.51, 2.99)
Brainp	1.18 (0.38, 3.71)	0.73 (0.28, 1.98)

* $P = 0.01$ by Mantel trend test

[a] Livbil excluded because there were no exposed cases

levels 0 and 1 of these exposures into a single reference level. Tables 3–5 summarize the exposure-cancer associations using the odds ratios estimated from the logistic regressions including age, death year, and one exposure at a time. (Similar results were obtained putting all exposures in one model.) For comparison to the logistic P values, we also computed the P values from the age + death-year stratified Mantel trend test using the continuous exposures, with five age strata (21–40, 41–60, 61–70, 71–80, 81–90) and two death-year strata (1969–1976, 1977–1984). These results are given in footnotes whenever they convey additional useful information.

Note that the dichotomous-regression results and the trend-test results sometimes conflict. The causes of such conflicts are that more controls than cases are exposed but exposed cases are more highly exposed than controls (in which case the dichotomous regression results appear negative but the trend test can be positive), or more cases than controls are exposed but exposed controls are more highly exposed than cases (in which case the dichotomous regression results appear positive but the trend test can be negative). P value plots and empirical-Bayes analyses revealed only one consistently very small P value, that of resins and lung cancer [10].

Logistic regressions with continuous regressors

Twenty-eight exposure-cancer associations identified as important in the binary-exposure regressions *or* in the contingency table screening were further modelled using continuous exposure scores. For the most suspicious associations in Tables 3–5, Table 6 summarizes the odds ratios for the 97th control percentile of the exposure score ver-

Table 6 Summary of logistic regressions with continuous regressors including age at death, death year, and hire year. Odds ratios are estimated relative risks for the 97th percentile of control exposure relative to no exposure

Abbreviations OR, odds ratio; CL, confidence limits; Mach fl, machine fluids; Orolx, oral cavity, larynx, and pharynx; Livbil, liver, gallbladder, and biliary tract; Brainp, brain including unspecified brain

Site	Exposure	OR	95% CL	*P* (two-sided)
Orolx	Solvents	2.8	0.68, 11	0.15
Livbil	Pyranol	2.2	0.76, 6.5	0.15
Lung	Resins	2.2	1.4, 3.6	0.001
Kidney	Benzene	1.9	0.92, 4.0	0.08
	Asbestos	1.5	0.37, 6.5	0.55
	Mach fl	3.2	0.57, 18	0.19
Lymphomas	Pyranol	1.5	0.55, 4.3	0.42
	Solvents	4.5	0.99, 21	0.05
Leukemia	Benzene	1.4	0.64, 3.2	0.38
Brainp	Benzene	2.1	1.00, 4.4	0.05
	Solvents	2.1	0.36, 12	0.41

sus no exposure, based on logistic regressions that included age, death year, and hire year. The 97th percentile was chosen because it was usually close to the maximum case exposure at each site, and so estimates based on it would be rough estimates of the maximum possible effect among these subjects. Only the estimates, and not the *P* values, would change if a different percentile was used. More extensive tabulations of the models we fit are given elsewhere [2, 3].

In comparing the continuous-exposure results in Table 6 to the dichotomous-exposure results in Tables 3–5, we note that the Pyranol-lymphomas and solvents-orolx associations are greatly reduced in apparent importance in the continuous regressions. For these associations, this discrepancy may be attributed to the fact that a much higher proportion of cases than controls are exposed, but the difference between cases and controls in average time exposed is not large. On the other hand, the positive associations of benzene with brain tumors, other solvents with lymphomas, and resins with lung cancer remain consistent between the dichotomous and continuous-exposure analyses.

Other analyses

In regressions with multiple exposures, we attempted to enter product terms to check for departures from the linear-logistic model. Where such terms could be fitted, none approached significance and all had extremely large variance, no doubt reflecting the small number of cases available for all sites (except the lung). Similar results occurred when we attempted to fit quadratic terms for single exposures. We also modelled the resins-lung cancer association using restricted cubic-spline logistic regression but again detected no important departures from the linear-logistic model.

As numbers permitted, we examined subtypes within the compound sites of orolx, livbil, lymphomas, and leukemias. Most subtypes had too few cases to produce any new results. However, the solvents' association with lymphomas (lymphosarcomas + reticulosarcomas) was entirely concentrated in the reticulosarcomas. In fact, five of the six reticulosarcomas were exposed to solvents level 2, compared to 654 of 1202 controls (54%), yielding a crude odds ratio of 4.2 and a (two-sided) *P* value of 0.01 in an age-death-year stratified trend test. Furthermore, all the exposed cases had at least 8 years of exposure. Pyranol level greater than 1 and benzene level 2 also showed associations with reticulosarcoma ($P = 0.02$ and 0.04 in age-death-year stratified trend tests), but both results were entirely attributable to two cases who had long-term exposures to Pyranol, benzene, solvents, and asbestos. Two of four cases with multiple myeloma were exposed to benzene level 2 compared to 178 of 1202 controls (crude odds ratio = 5.7, exact lower *P* value = 0.11).

We used a modification of an induction-latency analysis method of Thomas [11] (1983) to further analyze certain associations. The only notable finding was that the association of machining fluids with kidney cancer became significant in these analyses ($P = 0.001$ at an 8-year mode for the induction-latency period). We also performed certain analyses in which the exposure scores were divided into two epoch components, one measuring exposure up to 31 December 1950, the other measuring exposure after the date. Both components were entered in logistic regressions in place of the original variable. The associations of resins with rectal and lung cancer, machining fluids with kidney cancer, and TCE with leukemias appeared to be largely or entirely concentrated in the post-1950 exposures. Cross-tabulations showed that the association of solvents level 2 with reticulosarcoma was also concentrated in the post-1950 exposures, with crude odds ratios of 1.4 for pre-1950 exposure and 8.3 for post-1950 exposure.

All lung cancer analyses were repeated after deleting all deaths with ICD codes for nonmalignant respiratory diseases from the control group. This deletion had only a trivial impact on the results.

Discussion

We did not find any unambiguous associations with leukemia or colorectal cancer (the diseases whose reported excess led to this study). Nonetheless, we did see a fair

degree of consistency between earlier literature and the positive associations in our data. For example, there is some previous evidence for the positive association of polychlorinated biphenyls with liver/biliary cancer [12–14] and lymphomas [14], other solvents with lymphomas [15], asbestos with kidney cancer [16], and formaldehyde (used in resins operations) with lung cancer [17]. We are unaware, however, of previous links between machining fluids and kidney cancer, as seen in our study. As for the associations established by previous literature but not apparent in our data (asbestos-lung cancer and benzene-leukemia), the confidence intervals we observed were wide enough to include previously reported relative risks, and so our failure to observe the associations may only be due to statistical error.

The only unequivocally positive association in our analyses involved resins and lung cancer. It has been suggested that the observed resins-cancer associations may be due to exposure to hazardous levels of asbestos dusts in the operations in which asbestos-filled phenol-formaldehyde resin was produced. To test this hypothesis, we examined the associations of resins with esophageal, rectal, and lung cancer, separating resins within operations involving uncoded asbestos exposure (exposure not coded by our industrial hygienist) from resins within operations involving only coded asbestos exposure. If a resins-cancer association was due to asbestos confounding, we should expect the association to be concentrated in operations involving uncoded asbestos exposure. This appeared to be the case for lung cancer. For example, the logistic regression odds ratio for resins exposure without uncoded asbestos was 1.8 (95% CL = 0.68, 4.7; $P = 0.24$), while the odds ratio for resins exposure potentially with uncoded asbestos was 2.4 (95% CL = 1.4, 4.1; $P = 0.002$) (both odds ratios are computed at the 97th percentile of total resins exposure). On the other hand, the resins-esophageal and resins-rectal cancer associations were *not* concentrated in the operations with uncoded asbestos; in fact, they appeared more significant in operations without uncoded asbestos.

There are, of course, numerous other potential sources of bias that may have had an important effect on our results. As in most studies, uncontrolled confounding, selection bias, and misclassification need to be considered in interpreting our data. Obviously, the potential for bias from loss to follow-up and exposure misclassification is large; nonetheless, because we have little or no direct evidence regarding such problems, we omit a discussion of them here. Interested readers may consult our full reports [2, 3] for detailed consideration of possible biases and their likely impact on the present data.

Although our results are exploratory and suggestive only, they should be of value in comparison to findings of other, similar studies at other facilities. Because of the clear association of resins with lung cancer, and the concentration of this association in specific operations, we recommend that further research be conducted on the possible association of lung cancer with resin systems, especially phenol-formaldehyde systems.

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