Renal Cell Cancer among Paperboard Printing Workers

Thomas Sinks,[1] Boris Lushniak,[1] B. J. Haussler,[1] Joseph Sniezek,[1] Jou-Fang Deng,[1] Paul Roper,[1] Patricia Dill,[1] and Ralph Coates[2]

A physician's alert prompted us to investigate workers' cancer risk at a paperboard printing manufacturer. We conducted a retrospective cohort mortality study of all 2,050 persons who had worked at the facility for more than 1 day, calculated standardized incidence ratios (SIRs) for bladder and renal cell cancer, and conducted a nested case-control study for renal cell cancer. Standardized mortality ratios (SMRs) from all causes [SMR = 1.0, 95% confidence interval (CI) = 0.9–1.2] and all cancers (SMR = 0.6, 95% CI = 0.3–1.0) were not greater than expected. One bladder cancer and one renal cell cancer were included in the mortality analysis.

Six incident renal cell cancers were observed, however, compared with less than two renal cell cancers expected (SIR = 3.7, 95% CI = 1.4–8.1). Based on a nested case-control analysis, the risk of renal cell cancer was associated with overall length of employment but was not limited to any single department or work process. Although pigments containing congeners of dichlorobenzidine and o-toluidine had been used at the plant, environmental sampling could not confirm any current exposure. Several limitations and a potential selection bias limit the inferences that can be drawn. (Epidemiology 1992;3:483–489)

In June 1986, the National Institute for Occupational Safety and Health (NIOSH) received a request for technical assistance from the Occupational Safety and Health Administration to investigate a possible cancer cluster at a paperboard packaging manufacturing plant. The plant produces printed paperboard boxes for use as food containers. A local physician had reported that one worker had been diagnosed with renal cell cancer and another had been diagnosed with bladder cancer while working in the finishing department.

From the [1]National Institute for Occupational Safety and Health, Cincinnati, OH, and [2]Division of Epidemiology, Emory University School of Public Health, Atlanta, GA.

Address correspondence to: Thomas Sinks, Division of Environmental Hazards and Health Effects, National Center for Environmental Health, Centers for Disease Control, 1600 Clifton Road, F-46, Atlanta, GA 30333.

The paperboard packaging plant had been in operation for over 30 years. In 1986, the plant employed approximately 310 workers, 80 of whom worked in the finishing department. The manufacturing process involves four basic operations: (1) rolled paperboard is cut into sheets (sheeting); (2) printing presses are used to print colored product labeling and designs onto paperboard (printing); (3) printed paperboard sheets are cut to carton size, windows are cut into the cartons if required, and the cartons are creased for folding (cut and creasing); (4) cartons are waxed, and cellophane is glued in place over any windows (finishing). The purpose of this investigation was to determine whether an excess of bladder or renal cell cancer had occurred and, if so, to determine whether it was associated with any specific exposure or work-related process.

Subjects and Methods

CANCER MORTALITY

We conducted a retrospective cohort mortality study. Name, date of birth, sex, race, and Social Security number were abstracted from all personnel and death records in the company's possession. We considered workers whose race was unknown (85%) as white and whose sex was unknown (0.2%) as male. The study begin-date was January 1, 1957, the date from which we assumed all personnel records were complete. The

study end-date was June 30, 1988, when data collection began. We excluded 36 workers from the analysis because their date of hire or birth was missing, because they worked at the facility for only 1 day, or because they had not worked at the facility during the study period.

We determined vital status from the Social Security Administration (SSA) and by verifying a current mailing address through the U.S. Postal Service. An initial list of 825 employees was sent to the SSA in July 1988 for vital status determination. SSA reported whether workers were alive as of December 31, 1985 or were known to be dead, or whether vital status could not be determined. SSA discontinued this service shortly after this computer list was sent. To determine the vital status of the remaining 1,261 workers, we cross-referenced the complete list of 2,086 workers with SSA mortality tapes for the years 1937–1990. These tapes provide death information but do not provide any other information.

A subject was considered alive if we could identify a current mailing address as of April 15, 1989. If not, workers were considered alive as of December 31, 1985, if so indicated by the SSA. Workers whose vital status could not be verified by either a current mailing address or the SSA were considered alive as of their last day observed (June 30, 1988, for current workers and the last day of employment for terminated workers).

We requested copies of all death certificates from the respective state vital statistics offices. Underlying cause of death was coded by a qualified nosologist according to the International Classification of Diseases[1] codes in effect at the time of death.

Person-years at-risk of dying were accumulated for each worker starting the first day of employment at the plant or January 1, 1957, whichever came later. We stopped accumulating person-years at-risk at the study end-date (June 30, 1988), the date of death, or the date last observed, whichever came first. We used the NIOSH Life Table Analysis System[2] to distribute person-years at-risk over sex- and race-specific 5-year calendar time periods and 5-year age groups. Expected numbers of cause-specific deaths were calculated by multiplying the age, sex, and calendar time-specific United States mortality rates by the corresponding person-years at-risk. The number of observed cause-specific deaths was divided by the number of expected cause-specific deaths to yield a standardized mortality ratio (SMR). Exact 95% confidence intervals (CI) around the SMR were calculated based on the Poisson distribution.[3]

CANCER MORBIDITY

Current and former workers who had developed cancer were identified from three sources. First, we interviewed employees and management officials and examined company medical records. Second, we used the underlying and contributory causes of death noted on death certificates. Third, the master list of all current and former employees was sent to the Georgia Center for Cancer Statistics in May 1989 and matched against three databases. The Atlanta Metropolitan Area Surveillance, Epidemiology, and End Results (Atlanta-SEER) registry collects cancer incidence and mortality data on all residents of the five-county metropolitan area of Atlanta. Over 70,000 cancer cases diagnosed since January 1, 1975, were included in this database. Because the plant is located just outside the Atlanta-SEER catchment area, we also used a second incident cancer database. This was the Atlanta-SEER ineligible file, which included data on an additional 48,000 people who lived outside the five-county Atlanta metropolitan area but who were treated for cancer in metropolitan Atlanta hospitals since January 1, 1975. Finally, the cohort was matched against 180,000 records in a statewide registry, the Georgia State Tumor Registry. This registry has collected diagnostic information on all Georgia residents since January 1, 1980. Inasmuch as the state registry relies on voluntary reporting by hospitals, it is known to be incomplete, including approximately one-half the number of cases expected. Where possible, we accessed medical records to confirm the diagnosis and used these records to establish the date of diagnosis.

Next, a standardized incidence ratio (SIR) was calculated for renal cell and bladder cancers. This calculation was limited to white males in the cohort, who represented 88% of the person-years at-risk. The numbers of expected incident bladder and renal cell cancers were estimated using the age-specific person-years at-risk previously calculated but accumulated through December 31, 1990. Person-years at-risk were multiplied by age-specific cancer incidence rates[4] from the Atlanta-SEER registry for the years 1973–1977. The numbers of observed renal cell and bladder cancer cases were then divided by the estimated expected numbers to calculate the SIRs. We then calculated exact 95% CIs.[3]

CASE-CONTROL STUDY

We conducted a nested case-control study to determine whether work assignment (department) was related to the risk of renal cell cancer. Cases included all workers who developed renal cell cancer after their

date of first employment. We chose eight controls per case at random from a risk set of all employees born within 5 years of the case, the same sex as the case, and having attained the age at which the case was diagnosed (or died if date of diagnosis was unknown) without being diagnosed with renal cell cancer.[5] We also required that the age of first employment at the plant for controls be less than the case's age at diagnosis.

We used the detailed work histories of cases and controls to determine the length of employment in each department. We truncated work histories for controls at the age at which the index case was diagnosed. We tested the association between renal cell cancer and duration of employment in specific departments and in the plant overall. Owing to the small number of cases, we dichotomized duration of employment overall, and within specific departments, into less than 5 years and 5 or more years. We calculated an odds ratio based on the conditional maximum likelihood estimate with an exact 95% CI based on the mid-P definition.[6] To account for the long latent period of renal cell cancer, we reanalyzed the duration of employment variables by discounting duration of employment (lagged duration) just before the age of failure for each case using a 5- and 10-year lagged duration. We added time since first employment, date of hire, and age at hire to a conditional logistic regression model[7] to examine the effect on the parameter estimates for duration of employment. Owing to the small number of cases, the models were used only to assess confounding. The potential confounders did not alter the odds ratios for duration of employment, and these results are not presented.

ENVIRONMENTAL ASSESSMENT

We reviewed Material Safety Data Sheets (MSDS) for materials shown on the chemical inventory for the finishing department and the ink mixing and storage rooms. When the material safety data sheets provided little or no information on chemical contents of the product, we contacted the manufacturer. We reviewed the identified chemicals for carcinogenic potential. Bladder cancer has been associated with occupational exposure to various aromatic amines used in dye production.[8] Because several colored inks are used in the printing process at the study plant, we contacted manufacturers to determine whether the inks contained aromatic amines. Sixteen bulk samples of red, yellow, and orange printing inks were submitted to the NIOSH laboratory for measurement of free, unreacted dichlorobenzidine and o-toluidine content.[9]

Results

CANCER MORTALITY

As of the study end-date, 141 workers (7%) were deceased, 1,705 (83%) were alive, and 204 (10%) had been lost to follow-up (Table 1). A total of 115 (82%) death certificates were obtained from state vital statistics departments. For the final cohort, the median time since first employment was 20.4 years (mean = 19.8, range = 0.2-31.6); the median duration of employment was 2.5 years (mean = 2.9 years, range = 2 days to 32.7 years); the median age at hire was 23.7 years (mean = 26.4 years, range 18-58.7 years); and the median age at the study end-date was 44.6 years (mean = 46.4, range = 19.4-83.1 years). Table 2 shows the distribution of person-years at-risk by duration of employment and time since first employment.

Overall mortality for the cohort was similar to that expected (SMR = 1.0, 95% CI = 0.9-1.2), as was mortality from diseases of the heart (SMR = 0.9, 95% CI = 0.7-1.3), accidents (SMR = 1.0, 95% CI = 0.7-1.5), and violence (SMR 1.2, 95% CI = 0.6-1.9) (Table 3). The SMR for all cancers was less than expected (SMR = 0.6, 95% CI = 0.3-1.0). One bladder cancer death (SMR = 2.6, 95% CI = 0.1-14.5) and one renal cell cancer death (SMR = 1.4, 95% CI = 0.0-7.8) were included in the life table analysis. Although the risks of mortality were greater than expected for these cancers, they were based on only one death each.

The worker who died of renal cell cancer began employment 24 years before his death and left employment 5 years before he died. The worker who died of bladder cancer did so 11 years after beginning employ-

TABLE 1. Cohort Description and Vital Status

A. Cohort Status Breakdown			
Race/Sex	Total Cohort	Rejected	Final Cohort
White males	1,792	27	1,765
White females	228	9	219
Black males	63	0	63
Black females	3	0	3
Total	2,086	36	2,050
B. Vital Status			
Vital Status	In	Rejected	Analyzed
Alive	1,732	27	1,705
Dead	144*	3	141
Unknown	210	6	204†
Total	2,086	44	2,050

* Twenty-two additional deaths occurred after the study end-date and were considered alive in the analysis.
† Person-years at-work for these workers were censored at the date of last follow-up.

TABLE 2. Person-Years At-Risk by Latency and Duration of Employment

Years since First Employment	Duration of Employment						
	<6 Months	6 Months to 5 Years	5–10 Years	10–15 Years	15–20 Years	20+ Years	Total
<5	4,656	4,642					9,298
5–10	3,999	2,321	2,443				8,763
10–15	3,759	2,091	495	1,701			8,046
15–20	2,842	1,692	430	108	1,291		6,364
20–25	1,061	822	286	82	83	738	3,071
25+	282	253	122	49	47	447	1,201
Total	16,598	11,822	3,776	1,940	1,422	1,184	36,744

ment at the facility and working at the plant for 6 years.

Twenty-two deaths occurred after June 30, 1988, and were not included in the life table analysis. The underlying cause of death for two of these deaths was renal cell cancer. One of the two more recent renal cell cancer deaths occurred 19 years since first employment, and this worker had been employed at the facility for 19 years. The third renal cell cancer death occurred 22 years since first employment, but this worker had only been employed at the facility for 1 month.

Mortality from other and unspecified causes of death was greater than expected, owing to the relatively large number of deceased workers for whom no death certificate was obtained. As a result, the estimated cause-specific mortality rates presented underestimate the true rates for some causes. Death certificates could not be obtained for three deaths that occurred after the study end-date.

CANCER MORBIDITY

We identified a total of three workers with bladder cancer and six workers with renal cell cancer (Table 4). Interviews with employees and management identified four cases of renal cancer and two cases of bladder cancer. Pathology reports in the medical records of these workers confirmed the diagnosis in each case. Sixteen workers with cancer were identified by the Georgia Center for Cancer Statistics, but none of these had been diagnosed with cancer of the urinary tract. The SSA records identified three renal cancer deaths (one of which was known to the company) and one bladder cancer. The six observed cases of renal cell cancer exceeded the 1.6 cases expected (SIR = 3.7, 95% CI = 1.4–8.1). No increased risk of bladder cancer

TABLE 3. Observed and Expected Deaths, SMR, and 95% CI

Underlying Cause of Death	Observed Deaths	Expected Deaths	SMR	95% CI
All causes	141	135.0	1.0	0.9–1.2
All cancers	16	27.8	0.6	0.3–0.9
Site-specific cancers				
Digestive organs	5	6.1	0.8	0.3–1.9
Respiratory system	5	9.4	0.5	0.2–1.2
Renal cell	1	0.7	1.4	0.0–7.7
Bladder	1	0.4	2.6	0.1–14.7
Hematopoietic	1	3.4	0.3	0.0–1.6
Other and unspecified	2	4.7	0.4	0.1–1.5
Nonmalignant causes of death				
Heart diseases	35	37.0	0.9	0.7–1.3
Respiratory diseases	8	5.2	1.5	0.7–3.0
Accidents	24	23.3	1.0	0.7–1.5
Violence	15	12.9	1.2	0.6–1.9
Other causes*	27	4.1		

* Deaths from other causes include 23 deaths for which a death certificate was not obtained. Twenty-two deaths occurred after the study end-date and are not considered in this analysis.

TABLE 4. Characteristics of Workers with Renal Cell or Bladder Cancer

| Cases* | Sex | Dates of | | | | Pathology |
		Birth	Employment	Diagnosis	Death	
Renal cell cancer						
A	M	07/31	09/62–06/88	01/89	NA†	Renal carcinoma (clear cell)
B	M	03/31	10/69–06/88	12/88	02/90	Renal carcinoma (hypernephroma)
C	M	08/40	02/62–06/88	11/87	NA	Renal carcinoma (adenocarcinoma)
D	M	10/33	04/60–09/85	02/85	NA	Renal carcinoma (clear cell)
E	M	01/13	01/57–02/76	NA	02/81	Renal carcinoma‡
F	M	07/39	02/66–03/66	NA	07/88	Renal carcinoma‡
Bladder cancer						
AA	M	03/36	01/57–06/88	03/79	NA	Transitional cell
BB	M	08/35	06/61–06/88	07/82	NA	Transitional cell
CC	M	09/13	02/57–04/63	NA	05/68	Primary bladder‡

* Cases A, B, C, D, AA, and BB were identified using company records and employee interviews. Cases B, E, F, and CC were identified through the Social Security Administration. No cases were identified through a search of the Georgia Center for Cancer Statistics records.
† NA, not available or not applicable.
‡ No pathology data obtained.

was observed (2.8 cases expected, SIR = 1.1, 95% CI = 0.2–3.1).

NESTED CASE-CONTROL STUDY
The nested case-control analysis included six persons with renal cell cancer and 48 controls. The association between renal cell cancer risk and duration of employment was not limited to any single department or work process (Table 5). Employment in the plant for 5 or more years was associated with renal cell cancer. Three of the cases and three controls had worked in the finishing department. Employment in the finishing department for 5 or more years was associated with an increased risk of renal cell cancer (OR = 16.6, 95% CI = 1.7–453.1). These odds ratios did not change when employment during the most recent 5 or 10 years was not included in the calculation. One case had worked in maintenance, another case had worked in the office, and a third case had worked as a press operator for more than 5 years. Another case had worked in cut and crease, but for less than 5 years.

ENVIRONMENTAL EXPOSURE ASSESSMENT
Three potential carcinogens were identified in a review of the company material safety data sheets: methylene chloride,[10] formaldehyde,[10] and trichloroethylene.[11] These carcinogens are generally not considered as etiologic agents in human renal or urinary tract cancers. Nevertheless, occupational exposure to formaldehyde in the paper drying areas of the pulp and paper mill industry was associated with urinary tract cancer in one study.[12]

We contacted the suppliers of the printing inks and determined that several red, yellow, and orange pigments used in the paperboard printing process were manufactured from 3,3′-dichlorobenzidine (DCB) and from various substituted derivatives of toluidine. Laboratory analysis of 16 bulk pigment samples, however,

TABLE 5. Association between Renal Cell Cancer and Duration of Employment (5 or More Years) in a Paperboard Printing Manufacturing Plant

| Employment (Department or Work Process) for 5 or More Years | Renal Cell Cancer | | | | OR (95% CI)* |
| | Cases | | Controls | | |
	Yes	No	Yes	No	
All departments	5	1	9	39	∞ (3.0–∞)
Cut and crease	0	6	1	47	0.0 (0.0–152.0)
Finishing	3	3	3	45	16.6 (1.7–453.1)
Maintenance	1	5	2	46	5.3 (0.1–223.4)
Office	1	5	0	48	∞ (0.4–∞)
Press operators	1	5	0	48	∞ (0.4–∞)

* Odds ratios are based on conditional maximum likelihood estimates with 95% exact mid-P confidence intervals.

was inconclusive. The free amines of interest could not be detected in any of the samples. A second analysis to determine the total benzidine, 3,3'-dichlorobenzidine, and toluidine congener content was also unsuccessful, apparently due to difficulties in separating the pigments from the extracting solvents. Thus, it was not possible to confirm whether these compounds were present in the printing inks we sampled.

Three workers with renal cell cancer worked in the finishing department, where exposures included wax and its additives, various adhesives, and paper dust. Printed boxes enter the finishing department, where they may be coated with wax, cellophane windows may be added, and/or various glues may be applied. We could not determine whether finishing department workers were exposed to inks from recently printed cartons.

Discussion

This study represents the first report of excess renal cell cancer among paperboard printing manufacturing workers. As a group, this population experienced an excess risk of renal cell cancer. Five of the six renal cell cancers were not identified in the original cluster. The excess risk persisted when the index case was excluded from the analysis (SIR = 3.1, 95% CI = 1.1–6.8). The excess risk was associated with overall duration of employment but was not limited to any single department or work process.

Renal cell, bladder, and other urologic cancers accounted for over 70,000 new cases and over 20,000 deaths in the United States in 1989.[13] The annual age-adjusted renal cell cancer rate for the United States was 8.3 cases per 100,000 persons in 1987, and this rate had been steadily increasing by approximately 2% per year since 1973.[14] The annual age-adjusted renal cell cancer incidence rate in men is almost double the rate in women (11.6 vs 5.6 per 100,000 persons in 1987). Incident rates for renal cell cancer do not vary much between white and black men (11.7 vs 12.9 cases per 100,000 persons in 1987).

Little is known about the etiology of renal cell cancer. In the last 10 years, however, there has been growing evidence indicating occupational causes of renal cell cancer. Employment in petroleum refining and gasoline distribution,[15,16] production dry cleaning,[17,18] coke oven work,[19] leather work,[20] and the printing industry[21] has been associated with renal cell cancer. Recently, architects have been reported to be at excess risk of renal cell cancer.[22]

It is not clear why paperboard printing workers should be at excess risk of renal cell cancer. Whether this finding is spurious or related to an unidentified causal exposure is not clear. We could not account for exposures outside the work place, such as cigarette smoking, or during employment elsewhere. Certain pigments containing bladder carcinogens had been used at this plant. Both 3,3'-dichlorobenzidine and toluidine are suspected of causing cancer of the transitional cell epithelium that lines the bladder and the renal pelvis.[23,24] These chemicals are not thought to increase the risk of cancer of the renal parenchyma, the presumed site of all renal cell cancers identified in this study. Furthermore, we could not determine which workers were actually exposed to these chemicals.

The mortality analysis had several limitations. Because of changes in the availability of information normally supplied by the SSA, we could not determine the vital status of approximately 10% of the cohort. To account for this, we limited the person-years at-risk contribution from those lost to follow-up to the date last observed. We have no reason to believe that the lack of follow-up information is associated with disease outcome. A second limitation was the fact that the site-specific mortality rates were underestimated because the death certificates for 17% of those known to have died could not be obtained. Future follow-up of this cohort should help us to obtain these death certificates. The completeness of the cohort was not verified and could have resulted in an underestimation of person-years at-risk and our missing additional cancer deaths.

Cancer morbidity information could not be thoroughly assessed, and the SIR that we estimated should be considered as a rough estimate of the true renal cell cancer risk. For several reasons, the number of renal cell and bladder cancers experienced by this cohort may have been higher than we observed. First, Atlanta-SEER collects cancer incidence data from all hospitals within the metropolitan Atlanta region and does not include the nearby county where the facility was located. Second, the Georgia State Tumor Registry is considered incomplete and has only collected tumor incidence data since 1980. Third, migration of cohort members from the Atlanta-SEER catchment area would prevent identification of cancers among these persons. Finally, workers diagnosed with cancer before 1975 could only be identified through interviews with employees and management or death certificates. At the same time, owing to the small but steady increase in renal cell cancer incidence, the expected number of renal cell cancers that we calculated may have been slightly underestimated for person-years accumulated

since 1980. The expected number of renal cell cancers we calculated using the Atlanta-SEER rates (1.61) was almost identical to the expected number based on the overall SEER rates combined (1.59), and it is unlikely that the renal cell cancer rates reported by the Atlanta-SEER registry are seriously biased.

Another limitation was the potential for selection bias owing to our reliance on the reports of management and workers to identify persons with cancer. Thus, long-term workers who developed cancer may have been more easily identified than short-term workers who developed cancer. This possibility for bias may have resulted in a spurious association between duration of employment and renal cell cancer. The finding that the association between renal cell cancer and duration of employment was not limited to a single department supports the hypothesis that such a bias may have occurred. Such a bias, however, would not have affected the overall renal cell cancer excess experienced by this cohort.

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