CANCER MORBIDITY AND MORTALITY AMONG BENZENE WORKERS AT A CHEMICAL PLANT

Benzene is an aromatic hydrocarbon which has been used extensively in industry as a solvent. At room temperature, benzene is a colorless, clear liquid which is highly volatile. Numerous case reports and epidemiologic studies have linked benzene to leukemia in humans when exposures were prolonged and at high levels.[1,2,3]

More recent epidemiologic investigations have studied workers exposed to benzene in chemical plants, where exposures are generally lower. Ott et al[4] reported results from a cohort mortality study of 594 workers employed at Dow. Among persons in this cohort, 2 deaths were due to leukemia, with 1.0 expected. An additional death certificate showed leukemia on part 2, but could not be coded to this cause. A study by Decoufle et al[5] analyzed the mortality experience of 259 men employed at a chemical plant in Baltimore where benzene was used. Three deaths from leukemia occurred, with 0.44 expected. Infante et al[6] and later Rinsky et al[7] published findings from an investigation of persons with benzene exposures at 2 pliofilm plants in Ohio. Again, excess leukemia deaths were seen: 7 observed, 1.25 expected, according to the more complete follow-up by Rinsky. No leukemia deaths were reported by Tsai et al[8] among 484 refinery workers exposed to benzene; 0.78 deaths from leukemia were

expected. A large interindustry cohort mortality study of 4602 benzene workers was recently conducted by Wong[9] for the Chemical Manufacturer's Association. Wong reported 7 leukemia deaths, with 5.96 expected, based on U.S. rates. However, a second comparison group--an internal control population--experienced extraordinarily low leukemia rates (0 deaths, 3.40 expected) and this has complicated the interpretation of the data.

Overall, these epidemiologic studies of chemical workers suggest an increase in the risk of leukemia associated with benzene exposure; however, not all the studies have yielded increased risks, and in those studies with excesses the level of risk varies considerably.

Our study was undertaken to investigate whether benzene exposures experienced by employees at Du Pont's Repauno Plant in New Jersey are associated with increased risks of leukemia. We also set out to explore whether other major causes of death or cancer incidence are related to benzene exposures.

Benzene was first used at the Repauno Plant in 1915 to manufacture nitrobenzene. The chemical was initially handled by batch process in closed vessels. In the mid-fifties, the batch process was upgraded to a continuous process. Other engineering improvements occurred over the years, as documented by plant records.

Environmental monitoring for benzene levels commenced in 1975, with area sampling measurements generally ranging from

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1-3 ppm. The first personal breathing zone samples were taken in 1976. Eight-hour time-weighted averages for the most part were below 2 ppm and in all instances were below 10 ppm. Since 1980, all jobs monitored have shown levels below 1 ppm, except the nitrobenzene operator, whose exposures are less than 5 ppm. Detailed monitoring results and technical information on the chemical processes at the plant were used to identify and evaluate specific production areas where exposures to benzene could have occurred.

A chemist with industrial hygiene training and many years of service at the plant coordinated the job classification and data collection effort. Close contact was maintained between this plant liaison and the principal investigator, who frequently visited the plant and audited all procedures.

After a thorough review of pertinent information, a list was compiled of job assignments with the potential for benzene exposures. This list is displayed in the Appendix, Item 1. Each job was given an exposure ranking; rankings ranged from a low of 1 to a high of 9. These qualitative rankings were useful in making relative comparisons among jobs and in differentiating exposure levels for a specific job during different time periods.

Work history cards indicating job assignments had been kept at the Repauno Plant since the early days of nitrobenzene production. These cards were examined by trained coders, who abstracted data for all persons who had held jobs with potential

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for benzene exposures at any time from start-up of the nitrobenzene operations in 1915 through December 31, 1977. Abstracted information included name, social security number (when available), sex, race, birth year, termination year and reason, and a history of jobs held at the plant. For each job with potential for benzene exposure, the coder recorded the beginning and ending month and year of the assignment, a 2-digit occupation code, and a 1-digit severity code. Jobs held for less than 1 month were not included.

Coded data forms were sent to the Epidemiology Section, and then entered onto a computer tape. Persons who were not on roll as of January 1, 1945, or later, were then excluded. This cut-off date was used to facilitate follow-up by avoiding the problems of tracing the large numbers of persons who left work during World War II, as well as persons who terminated prior to the establishment of the Social Security Administration and hence lacked social security numbers.

After the exclusion of employees not on roll on or after 1945, there remained 890 persons. Of these, 863 were males and 27 were females; very few were non-whites. We elected to include only males in the cohort, since the number of females was small.

Follow-Up Procedures

To determine vital status and obtain medical data for the cohort, we used resources within Du Pont as well as those outside the company. Cancer incidence data were retrieved from our

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Cancer Registry, which includes diagnoses and/or deaths among active employees. We used several approaches to establish vital status: 1) a search of the Du Pont Mortality File: this file was begun in 1957 and contains information on deaths which have occurred among all employees and pensioners; 2) Searches by the Social Security Administration (SSA) for persons who terminated for any reason prior to 1957 or who terminated without a pension after that date; 3) Inquiry to the National Death Index to supplement SSA searches. For deceased persons, we requested death certificates from state health departments. Cause of death was coded by trained nosologists in our Medical Division, according to the International Classification of Diseases.

Vital status and employment status of the cohort are presented in detail in Table 1. As of December 31, 1982, 587 men (68.0%) remained alive, 249 (28.9%) had died, and 27 (3.1%) had unknown vital status. About 11% of the cohort remained employed at Du Pont, with 41% pensioned and 42% separated for reasons other than retirement or death.

Analytic Methods

For cancer incidence analyses (diagnoses among persons actively employed at Du Pont), person-years were accumulated from the date of first exposure after January 1, 1956, when the Cancer

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Registry was begun, until the earliest of the following events: date of cancer diagnosis, date of death, date of termination (resignation, discharge, or retirement), or December 31, 1983.

For mortality analyses, person-years began at the date of first exposure after January 1, 1945, and continued until the date of death, the date lost to follow-up, or until December 31, 1982, whichever occurred first. Table 2 presents, by calendar year and age group, the compilations of person-years of observation used in the incidence and mortality analyses. In total, 8692 person-years accrued for incidence analyses and 21,482 for mortality analyses.

Expected numbers were computed using the OCMAP program developed by Marsh and Preininger[10]. Observed and expected numbers have been statistically evaluated using probability tables for the Poisson distribution (two-tailed).

Cancer incidence rates obtained for the Du Pont Company's male wage roll population were used in the incidence calculations. Mortality rates for wage roll males in Du Pont and for U.S. white males were used as referents for mortality analyses.

For comparison purposes, Du Pont rates are preferable to those derived from the general population because the general population includes persons hospitalized or otherwise too sick to be employed and thereby generally yields expected numbers which are higher than those determined using a healthier employed population as the referent. The magnitude of an excess risk,

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then, may be underestimated when expected numbers are based on general population rate because of this "healthy worker effect." Furthermore, ascertainment of cases is consistent within Du Pont, whereas methods employed elsewhere might differ.

Results - Cancer Incidence

In total, 30 cases of cancer were diagnosed from 1956-1983 among employees in the cohort, compared to 21.5 expected according to company rates (Table 3). This overall excess results from higher than expected cancer experience for certain specific malignancies: lung (8 observed, 4.2 expected), bladder (4 observed, 1.0 expected), and leukemia (3 observed, 0.6 expected). The differences between observed and expected numbers for bladder cancer and leukemia reached statistical significance.

To examine dose-response patterns, we defined an index of cumulative exposure as the sum across all jobs of the cross-product of duration and level of exposure. Cumulative exposure was categorized as less than 1, 1-10, and more than 10. These break points were selected to split the person-years into roughly 3 equal groups. Three time periods, less than 15 years, 15-20 years, and more than 20 years, were selected to examine latency. For these analyses, all past exposures, including those prior to 1945, were considered in determining the categories into which the person-years fell, even though person-years before 1945 were not used in the actual calculations, as explained earlier.

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In Table 4, it can be seen that the lung cancer excess in the cohort occurred primarily in the 20+ latency group, and was greatest in the 10+ category of cumulative exposure (4 observed, 1.2 expected). This excess was not statistically significant.

Examining the margins of Table 5, no clear pattern of latency or dose-response emerged for the 4 bladder cancer cases; however, 2 of the 4 cases occurred more than 20 years after first exposure among the group whose cumulative exposure category was 10+.

Table 6 presents the distribution of the leukemia cases. There were no cases of this type of cancer in the 20+ latency period or in the 10+ cumulative exposure category. More specific information for the 3 leukemias is shown below:

Type	Duration of Exposure (Yrs)	Cumulative Exposure Index	Latency (Yrs)
Chronic Myeloid	1.1	2.2	16.2
Acute Lymphatic	0.3	0.3	10.2
Lymphatic, Unspecified	2.0	3.7	15.0

These men spent relatively short periods in benzene areas and worked in jobs that were considered to have little potential for exposure.

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Results - Mortality

Deaths from 1945 through 1982 among benzene workers numbered 249, with 229.8 expected based on Du Pont rates (SMR = 108) and 274.4 expected based on U.S. rates for white males (SMR = 91).

Observed and expected numbers are presented in Tables 7 and 8. Based on Du Pont Company rates, this cohort experienced a significant excess of diseases of the circulatory system (146 observed, 122.1 expected), due largely to excesses of ischemic heart disease. By the U.S. comparison, this cause of death was not excessive.

Cancer deaths numbered fewer than expected by either comparison (49 observed; 51.4 expected, Du Pont; 54.6 expected, U.S.). No specific type of cancer was significantly in excess. Lung cancer, which was nonsignificantly elevated in the incidence analyses, was underrepresented as a cause of death (14 observed, 16.6 expected). Bladder cancer deaths were slightly in excess (3 observed, 2.0 expected). Leukemia mortality was as predicted (2 observed, 2.0 expected).

Dose-response and latency analyses of deaths from all malignant neoplasms failed to reveal any patterns of increasing risk as the amount of exposure or time since first exposure increased. In reviewing trends for bladder cancer deaths, it can be noted that all 3 occurred 20+ years after exposure: 2 of these were in the 10+ cumulative exposure category, with 0.8 expected;

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the other one was in the middle (1-10) category, with 0.5 expected. No trends were seen for lung cancer deaths, which were in deficit, or for the 2 leukemia deaths.

Discussion

To evaluate the findings of our study, there are a number of limitations to be considered. First, analyses of cancer incidence stemmed from data from Du Pont's Cancer Registry, and were therefore confined to diagnoses occurring during years of employment. We were unable to ascertain incident cases among persons after they were separated from Du Pont. Nonetheless, our analyses did provide cancer morbidity data, which is lacking in so many studies of workers. Furthermore, cancer cases which developed among persons after they left employment and which resulted in death could be discovered through our mortality follow-up.

Second, our cohort, while larger than most described in the benzene literature, was limited to 863 men. As such, the power of our study to detect a cancer excess was only moderate: with $\alpha = .05$, $\beta = .20$, for all cancers, a relative risk for cancer of about 2.0 could be detected in incidence analyses and 1.7 in mortality analyses. Detectable risks for lung cancer were 3.0 and 2.1, respectively. For types of cancer which occur more rarely, the power to detect an excess was clearly less.

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Another limitation results from the cohort inclusion criteria we selected. For ease of tracing and to ensure successful follow-up, we restricted the cohort to persons on roll on or after January 1, 1945. In fact, follow-up was highly successful, with only 3.1 percent untraced. However, a very large number of persons with early exposures to benzene had to be eliminated from the study to meet the 1945 inclusion criterion. If persons who terminated prior to 1945 had a different cancer experience from those who remained, our study would not fully reflect the risks of all employees exposed to benzene at the Repauno Plant. It should be noted, however, that our cohort did include many persons whose exposures occurred decades ago, allowing for a long period of follow-up.

Lastly, our study shares the difficulties which most retrospective investigations of workers have with respect to measuring exposures. We were unable to isolate a cohort of workers exposed purely to benzene. A variety of other agents were present in the production areas at the Repauno Plant. In particular, nitrobenzene and aniline exposures were common, and to a lesser extent, acetone. Any associations which emerged from our data analyses could have been confounded by these or other chemicals. Additionally, our measurement of benzene exposure was incomplete, as personal sampling data were not available before 1976, and earlier area monitoring results were sparse. Instead, we elected to systematically rank jobs based on an evaluation of

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the available monitoring data and informed judgments as to levels in the past. Such a system, with its limitations, nonetheless provides a sound basis to differentiate jobs from one another and thus is useful in examining dose-response trends. To the extent that our linear ranking of jobs provided an imperfect measure of an individual's exposure, our dose-response analyses must be interpreted with caution.

Having considered the limitations of the study, one can now critically examine its findings. Our analyses of cancer incidence among men who worked in jobs with potential for benzene exposure revealed statistically significant excesses of bladder cancer and leukemia. These excesses were based on small numbers of diagnosed cases: 4 bladder cancers and 3 leukemias. Further subdivisions of the cohort into categories of cumulative exposure and latency yielded many empty cells and marginal subtotals which are based on one or two cases. No clear patterns of dose-response were evident in these data. Persons with the highest (10+) cumulative exposure and those with the longest (20+) latency period were not more likely to develop these malignancies that those with less exposure or shorter latency. This lack of clear trend weighs against causality, but it must be acknowledged that the data were very sparse.

Another criterion for judging causality is consistency of findings across studies. As reviewed earlier, benzene's leukemogenic properties at high levels of exposure have been established, and several epidemiologic studies have now reported

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excess risks among chemical workers. In these other cohorts, deaths due to leukemia were few, as in this study. Our finding of a five-fold excess in cancer incidence is reasonably consistent with risks seen in these earlier studies.

Our mortality statistics were not elevated. Two leukemia cases counted in our incidence analyses did not appear in the leukemia mortality statistics because their deaths could not be coded to this cause according to information provided on the death certificate. In each instance, the certificate indicated that death was due to another type of malignancy. Had these deaths been due to leukemia, our mortality statistics would have yielded a two-fold increase in risk.

Specificity of target organ and cell type can be suggestive of causal associations. Many previous researchers have examined histologic type in evaluating their findings of excess leukemias. Infante noted that all 7 leukemia deaths in his study were of the myelogenous or monocytic type. Ott's study included 1 leukemia of unknown type, one acute myelogenous leukemia, and 1 myeloblastic leukemia. Decoufle reported 1 case each of chronic lymphocytic, acute monocytic and acute myelomonocytic leukemia. Wong's 7 leukemia deaths included 2 each of chronic lymphatic and chronic myeloid types and 1 each of acute lymphatic, lymphatic (unspecified) and acute (unspecified) leukemia.

The 3 leukemia cases in our cohort were chronic myeloid, acute lymphatic, and lymphatic (unspecified). An

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additional death due to acute lymphatic leukemia is included in our mortality analysis. Thus, no unique histologic type of leukemia is responsible for the excess seen in our cohort. A mixture of leukemia types appears to characterize several of the cohort studies reported in the literature, and is in marked contrast to the predominance of acute myelogenous leukemias which have been causally linked to high levels of benzene.[11]

A review of the literature for associations between benzene and bladder cancer failed to disclose studies offering support for this hypothesis. No bladder cancers were among the 9 cancer deaths in Decoufle's study nor among the 4 cancer deaths in Tsai's report. Deaths from bladder cancer were not reported in the publications by Infante, Rinsky, or Ott. Wong's study found 4 bladder cancer deaths, with 3.91 expected. Hence, our excess of bladder cancer stands alone in the published reports. Considering the absence of a dose-response trend, the most likely explanation of our finding is that it represents a chance occurrence. Since our study analyzed numerous types of cancer incidence and many causes of death, it would be surprising not to see a few which are statistically in excess as a result of the multiple comparisons which were made.

Summary

This retrospective cohort study of 863 men with potential for exposure to benzene at Du Pont's Repauno Plant revealed a

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statistically significant excess of bladder cancer and leukemia cases. Mortality data did not show significant excesses of deaths due to these or other types of cancer. The leukemia excess was based on only 3 cases. The relative risk seen in this cohort was similar in magnitude to risks reported in other studies of chemical workers. On the other hand, the short duration and low intensity of exposure to benzene which occurred among cases weigh against a causal relationship. Our reported excess of bladder cancer incidence was also based on small numbers and did not show a dose-response gradient. Increased risks from bladder cancer have not been reported in other studies of benzene workers, and our finding may well be a consequence of multiple comparison testing. Continued follow-up of this cohort and additional analyses from other studies should help clarify the meaning of our results.

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TABLE 1

Vital Status as of 12/31/82

Alive	587	(68.0%)
Deceased	249	(28.9%)
Unknown	27	(3.1%)
TOTAL	863	(100%)

Employment Status as of 12/31/82

Currently Employed	92	(10.7%)
Terminated	366	(42.4%)
Retired	354	(41.0%)
Died while Employed	51	(5.9%)
TOTAL	863	(100%)

TABLE 2

Person-Years At Risk

CANCER INCIDENCE	1955-59	1960-64	1965-69	1970-74	1975-83	TOTAL
<20	5.1	5.9	0.6	0.0	0.0	11.6
20-24	33.3	183.8	62.0	1.4	1.9	282.5
25-29	149.0	224.2	245.9	24.6	12.5	656.2
30-34	222.5	283.0	247.2	153.6	19.0	925.3
35-39	212.7	300.7	288.9	188.3	87.9	1078.5
40-44	225.7	274.6	292.3	270.1	192.6	1255.3
45-49	190.0	271.4	280.1	272.2	285.3	1299.0
50-54	194.1	222.0	267.5	289.6	355.7	1328.9
55-59	160.9	225.6	204.1	242.6	315.0	1148.2
60-64	108.8	146.9	146.4	112.8	148.6	663.5
65-69	6.1	15.6	12.3	3.4	5.8	43.1
TOTAL	1508.1	2153.7	2047.3	1559.80	1424.3	8692.1

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TABLE 2 (CONT.)

Person-Years At Risk

MORTALITY

	1945-49	1950-54	1955-59	1960-64	1965-69	1970-74	1975-83	TOTAL
<20	0.0	0.0	5.1	5.9	1.2	0.0	0.0	12.1
20-24	57.2	143.3	46.0	195.5	93.2	17.5	3.4	556.1
25-29	119.8	321.3	352.4	267.0	318.2	122.6	29.5	1530.8
30-34	228.5	255.8	471.4	454.0	324.0	367.7	141.4	2242.8
35-39	256.1	285.3	385.4	523.9	483.0	393.4	472.6	2799.8
40-44	258.7	290.3	349.7	406.8	528.2	509.8	649.2	2992.7
45-49	203.7	269.6	301.9	363.1	420.3	529.0	699.6	2787.3
50-54	188.4	216.6	274.4	285.6	354.3	435.4	841.1	2595.7
55-59	108.1	180.6	223.5	266.6	269.6	365.1	696.3	2109.8
60-64	70.1	105.0	173.6	204.3	249.3	260.4	582.6	1645.3
65-69	13.1	62.3	88.2	128.9	181.9	224.6	407.7	1106.8
70-74	0.0	10.0	48.8	57.1	106.9	152.4	281.0	656.2
75-79	0.0	0.0	7.6	27.0	41.0	76.2	171.3	323.0
80+	0.0	0.0	0.0	2.7	5.6	24.5	90.7	123.5
TOTAL	1503.8	2140.2	2728.0	3188.4	3376.7	3478.6	5066.3	21482.0

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TABLE 3

Observed and Expected Cancer Incidence 1956-1983

Based on Du Pont Company Rates

	Observed	Expected
All Sites	30	21.5
Buccal Cavity/Pharynx	0	0.8
Esophagus	0	0.3
Stomach	1	0.8
Intestine	1	1.9
Rectum	1	1.1
Pancreas	2	0.5
Larynx	2	0.7
Lung, Brochus, Trachea	8	4.2
Malignant Melanoma	1	0.7
Skin	2	2.5
Breast	1	0.1
Prostate	1	1.1
Bladder	4*	1.0
Kidney	0	0.6
Brain	2	0.6
Leukemia	3*	0.6
Other lymphopoietic	0	1.3
Unknown	1	0.4

*P < .05

TABLE 4

Observed and Expected Lung Cancer Cases, 1956-1983

By Latency And Cumulative Exposure

Based on Du Pont Company Rates

		Cumulative Exposure			
Latency		< 1	1-10	10+	TOTAL
<15	OBS	1	0	0	1
	EXP	0.3	0.4	0.2	0.8
15-20	OBS	1	0	0	1
	EXP	0.2	0.2	0.2	0.6
20+	OBS	0	2	4	6
	EXP	0.6	0.9	1.2	2.7
TOTAL	OBS	2	2	4	8
	EXP	1.1	1.6	1.5	4.2

TABLE 5

Observed and Expected Bladder Cancer Cases, 1956-1983

By Latency And Cumulative Exposure

Based on Du Pont Company Rates

		Cumulative Exposure			
Latency		< 1	1-10	10+	TOTAL
<15	OBS	0	1	0	1
	EXP	0.1	0.1	0.0	0.2
15-20	OBS	0	0	0	0
	EXP	0.0	0.0	0.0	0.1
20+	OBS	1	0	2	3*
	EXP	0.2	0.2	0.3	0.6
TOTAL	OB	1	1	2	4*
	EXP	0.3	0.3	0.4	1.0

*P < .05

TABLE 6

Observed and Expected Leukemia Cases, 1956-1983

By Latency And Cumulative Exposure

Based on Du Pont Company Rates

		Cumulative Exposure			
Latency		< 1	1-10	10+	TOTAL
<15	OBS	1	0	0	1
	EXP	0.1	0.1	0.0	0.2
15-20	OBS	0	2**	0	2**
	EXP	0.0	0.0	0.0	0.1
20+	OBS	0	0	0	0
	EXP	0.1	0.1	0.1	0.3
TOTAL	OBS	1	2	0	3*
	EXP	0.2	0.2	0.2	0.6

*P < .05

**P < .01

TABLE 7

Observed and Expected Deaths, 1945-1982

Based on Du Pont Company Rates

	Observed	Expected	SMR
All Causes of Death	249	229.8	108
Malignant Neoplasms	49	51.4	95
Digestive	13	15.1	86
Lung	14	16.6	84
Prostate	3	4.2	--
Bladder	3	2.0	--
Brain	3	1.3	--
Leukemia	2	2.0	--
Other Lymphopoietic	3	2.4	--
Disease of Circulatory System	146	122.1	120*
Ischemic Heart Disease	105	93.2	113
Cerebrovascular Diseases	21	19.9	105
Respiratory Diseases	10	12.4	80
Digestive System Diseases	8	7.9	102
External Causes	21	14.3	147

*P < .05

TABLE 8

Observed and Expected Deaths, 1946-1982

Based on Rates for U.S. White Males

	Observed	Expected	SMR
All Causes of Death	249	274.4	91
Malignant Neoplasms	49	54.6	90
Digestive	13	15.4	84
Lung	14	17.4	81
Prostate	3	3.7	--
Bladder	3	1.7	--
Brain	3	1.6	--
Leukemia	2	2.2	--
Other Lymphopoietic	3	3.2	--
Disease of Circulatory System	146	145.7	100
Ischemic Heart Disease	105	103.9	101
Cerebrovascular Diseases	21	19.6	107
Respiratory Diseases	10	16.5	61
Digestive System Diseases	8	13.2	61
External Causes	21	24.2	87

* $P < .05$

Appendix, Item 1

Job Assignment	Occupation Code	Severity Code
Nitrobenzene Operator, Before 1925	1 1	9
Nitrobenzene Assistant, Before 1925	1 2	9
Nitrobenzene Operator, 1926-1955	1 1	8
Nitrobenzene Assistant, 1926-1955	1 2	7
Nitrobenzene Operator, 1956-1962	1 1	6
Nitrobenzene Assistant, 1956-1962	1 2	6
Nitrobenzene Operator, 1963-	1 1	5
Nitrobenzene Assistant, 1963-	1 2	5
Aniline Operator, Before 1925	2 1	8
Aniline Assistant, Before 1925	2 2	8
Aniline Operator, 1925-1942	2 1	7
Aniline Assistant, 1925-1942	2 2	7
Aniline Operator, 1943-1958	2 1	5
Aniline Assistant, 1943-1958	2 2	5
Aniline Operator, 1959-1962	2 1	4
Aniline Assistant, 1959-1962	2 2	4
Aniline Operator, 1963-	2 1	3
Aniline Assistant, 1963-	2 2	3
Aniline - Iron Shed Operator or	2 3	2
Reducer House Operator	2 3	2
Diphenylamine Operator,	2 4	3
Diphenylamine Assistant,	2 5	3
Diphenylamine Foreman,	2 6	3
Janitor, Plant Lab	2 9	4
Yardman	1 4	7
RDX	1 3	7
Miscellaneous Foreman	0 0	1
Miscellaneous Operator,	0 1	1
Miscellaneous Assistant (Helper)	0 2	1
Hexite Operator, Before 1963	0 3	1
Hexite Assistant, Before 1963	0 4	1
Hexite Operator, 1963-	0 3	4
Hexite Assistant, 1963-	0 4	4

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Qualitative Severity Code (continued)

Job Assignment	Occupation Code	Severity Code
Hexite Operator - Diamond Recovery 1972-	0 3	0
Hexite Assistant- Diamond Recovery 1972-	0 4	0
PETN	0 5	1
Tetryl	0 6	1
Unicel ND, DNPT	0 7	1
Cap (Caprolactam)	0 8	1
Nitrosan (BL-353)	0 9	1
Mechanic, Hexite Area	1 5	6
NAC Operator - NB Waste Acid	1 8	2
NAC Assistant	1 9	2
Lab Chemist, Before 1955	1 6	6
Lab Technician, Before 1955	1 7	6
Lab Chemist, After 1955	1 6	4
Lab Technician, After 1955	1 7	4

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