CANCER MORTALITY IN AN INDUSTRIAL AREA OF BALTIMORE

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American Public Health Association°
Washington, D.C.

Supported by Contract # 68-01-3859
awarded to the American Public Health Association by
Office of Toxic Substances
U.S. Environmental Protection Agency
401 M Street, SW
Washington, D.C. 20460

Cancer Mortality in an Industrial Area of Baltimore

Arsenic has long been known to be a poison when ingested in large quantities by man, animals or plants. It is known that continued ingestion of high natural levels of arsenic in water or food will produce skin lesions including cancer (Tseng et al., 1968; Braun, 1958). Consumption of arsenic as a therapeutic agent is also known to cause skin lesions (Neubauer, 1947).

The risk of inhalation of arsenic has not been as extensively investigated. Workers exposed to arsenic in the manufacture of pesticides have an increased risk of lung cancer and lymphomas (Ott et al., 1974; Baetjer et al., 1975). Less is known about the chronic health effects in the general population exposed to arsenic in the air. It is known that children around smelters may have high arsenic levels in nails and hair but it is not clear whether these observed indications of absorption of the agent also indicate long-term toxicity. Blot and Fraumeni (1975) have suggested that there may be an association between excessive lung cancer mortality and the existence of non-ferrous smelting industries in several counties in the U.S. It is not known whether some by-product of this industry such as arsenic is associated with these carcinogenic effects.

The purpose of the current study is to determine whether there is an excess mortality from cancer in the population which resides near a chemical plant in the inner city of Baltimore and whether any observed excess can be associated with previous exposure to arsenic. The plant has produced insecticides, herbicides, and other arsenic products from 1897 until early 1976. In 1952, the original plant was torn down and a new one erected with better hygienic conditions for the workers. The plant produced arsenic acid, calcium and lead arsenate, Paris green (a cupric acetoarsenite), and sodium arsenite. In the past, all products were dried and packaged except sodium

arsenite which was shipped as a liquid. Paris green was not produced after the early 1950's and no dry arsenicals after 1973. Other pesticides such as chlorinated hydrocarbons and organophosphates were not produced at this facility but were made into formulations on-site since 1947. There are several other industries which are currently located in the area or have been manufacturing in that vicinity in the past.

METHODS

The census tracts which were selected as having had possible environmental exposure to arsenic from the point source of the pesticide plant were defined empirically as those for which at least 50 percent of their area lay within a 3/4 mile radius of the plant. This distance was chosen so that large tracts which lay across the river and in which the majority of the population did not reside within a one mile radius of the plant would not be included. The four index census tracts which fit these criteria were 2303, 2302, 2404 and 2301. The tract in which the plant was located was 2303.

The comparison group of census tracts consisted of all tracts which matched the index ones on age distribution, race, sex and socioeconomic factors. Index tracts 2303, 2302 and 2404 were similar in these matching characteristics and were compared to the same set of comparison tracts designated as Match I. Index tract 2301 differed in age and race distribution from the others and was compared to a second set of tracts, Match II. Death rates for the index tracts in 1958 through 1962 were compared to tracts selected for matching through information available from the 1960 census. The matching criteria used to select control tracts for comparison of death rates in all other years were derived from the 1970 census. The age distribution differs in the tracts across time intervals, so to compare rates between years, the figures have been age-adjusted.

The initial matching criteria for the 1970 census were:

Age distribution ± 10% for each age

Race ± 15%

Sex ± 5%

Median income ± $1,000

% below poverty level ± 10%

% head of household over 65 years ± 20%

The matching criteria based on the 1960 census were the same except that the variation in median income was reduced to reflect current inflation, and information on the last two characteristics was not available in the earlier decade.

The matching tracts in 1970 and 1960 are shown in figures 1 and 2. A total of 18 Match I control tracts was identified from 1970 census data and 45 tracts from 1960 data. A total of five Match II tracts was found in both census periods. The variation in the numbers of Match I tracts between the two periods is the result of changing racial distribution especially in middle income census tracts over the past ten years. The three index tracts have a predominantly white population and fewer census areas have that racial distribution in the later time period.

The index area was stable with an increasing proportion of individuals living in the same household for five to seven years from the 1960 to the 1970 census. This stability is also reflected in the slight increase in age of the population of the area.

The scattered distribution of the control tracts has placed them in areas which may also have had different risks. The adjacent tracts contiguous to the index ones may have had minimal exposure to the same agents as in the major area. Southern tracts are in heavy industrial areas as are the central tracts

but the characteristics of the populations and their stability are different. The northern area consists of mainly residential dwellings with little industrial exposure. For tnese reasons the controls were divided into four groups for comparison - adjacent, south, central, and north.

Cancers were identified by examining all certificates of deaths which occurred within the city for the years 1958-62 and 1968-74. The death was selected for study if cancer appeared as a cause listed anywhere on the death certificate, with the exception of the years 1973-74 where only cancers listed as underlying causes were chosen. Deaths of city residents were selected from the total cancer list. This procedure would not include the deaths of city residents which occurred outside the city. In order to determine the extent of these differences we abstracted information on out-of-city deaths of city residents for the three years, 1970-72. The proportional increase in deaths for the index tracts was five percent and for the control tracts 13 to 15 percent. This difference is not large enough to account for the variation in cancer rates observed. Deaths were included only once using either underlying cause or first cancer listed. Adjustments were made in the changing codes in the 7th and 8th revision so that data by site of cancer were compatible for the total period.

The hospital records of a sample of cancer deaths were reviewed to verify the accuracy of death certification of cancers in Baltimore, to identify any possible differences in diagnosis by area in the city, to determine any variation in pathological characteristics of cancers in index and comparison areas and to investigate differences in personal characteristics such as smoking as described in hospital charts. The review specifically focused on unusual cell types of lung cancer and possible arsenic-associated symptoms and diseases

in cancer patients from the index and control areas.

The soil was sampled for the presence of arsenic in the areas near the chemical plant. The original selection of sampling sites was determined both by distance from the plant and by direction from north through south coordinates. We intended to collect about half the samples within the 1/4 mile radius and 40 percent at the next 1/4 mile distance with the remaining samples collected further out on the radii. Control samples would be taken from two parks nearby but a distance greater than 1 mile from the plant. The field survey team had problems adhering to the sampling design since the sources of soil were limited in the area. We attempted to take samples near residences whenever possible as long as there were no obvious problems of tree-cover, water run-off, or redevelopment. For those few samples taken at private housing, the residents were interviewed concerning the use of herbicides or pesticides in the area and the sample was avoided if the soil had been treated. After collection of the original 101 samples taken at 35 sites under these directives and including additional samples in the park, a second set of samples was collected in a north and north-west direction to determine how far distant the high levels could be detected. Special emphasis was placed on sampling from the park which was adjacent to the plant. This park has a central grassed area which had been recently re-sodded. Surrounding the park was a dirt-track which had been undistrubed. Part of this path was adjacent to the fence along the plant boundary and near the areas where railroad cars were filled. Another portion bordered on the water and the last was adjacent to railroad tracks.

Samples were collected at one, two and four inches at each location unless otherwise noted. A core sampler, with a 3/4 inch bore and marked at one inch intervals, was driven into the ground and samples were removed down to the

appropriate depth marked. A one foot circle was marked off around a selected site and a set of samples was collected according to the described technique until the 30 ml. polyethylene sample bottle was filled with soil from the appropriate depth but from different core samples. Initially we had tested four sites using consecutive one inch samples down to a depth of four inches. We found that samples at three inches were usually close to those at four and thus it was elected to take the extreme depth and discard the three-inch level. Samples in control areas were all obtained from two city parks, Riverside or Federal Hill.

All instruments used in collecting samples were free of arsenic. The analysis was done using either conventional flame or flameless atomic absorption spectrophotometry depending on initial level of arsenic.

RESULTS

Mortality by Tracts

The crude rates for cancers at four specific sites, oral, pancreas, lung and prostate as well as for all cancers are presented for males in tables 1 and 2. The first table includes data for the five-year period around the 1960 census and the second table for a seven-year period around the 1970 census. As can be seen the risk for lung cancer and for all cancers is excessive in the period around the 1970 census for tract 2303 compared to any of the control groups. This is not consistently true in the earlier period. The weighted relative risk of lung cancer in white males from index tract 2303 as compared to north controls which had the highest control rate is 2.5 as shown in table 2 with a probability of .0005 as determined by the chi square calculated by the Woolf-Haldane method. The black males in tract 2301 also have a higher rate of lung cancer but this was not true for white males in the same tract. In the 1960

census period, although the lung cancer rate is higher in white males in tract 2303 than the north and south controls, there is very little difference between the rates for all index tracts and for the adjacent and central controls. There are no differences in rates for males in tract 2301 and their control groups.

If we examine the comparable crude mortality rates for females in tables 3 and 4, we can find no excess risk of cancer at any site for census area 2303. In fact, the overall cancer rate appears somewhat low especially in the 1958-62 period. The mortality from breast cancer is slightly high in the early period and there are no deaths from cervical cancers. The lack of an observed increase in lung cancer in women in 2303 might be the result of a small population size. This will be discussed later in the report.

Adjusted Rates

The age distribution of the index tract changed with time and these differences were reflected in similar changes in the control group. In order to have appropriate comparisons the mortality rates for each cancer site and for all cancers have been adjusted using the method of standardized mortality ratios. The average annual Baltimore City mortality rates were calculated from all deaths in the 1968-74 period and these values were used as standards to adjust the mortality in each time period. As seen in table 5, the mortality ratios for white males in the tract 2303 were high for cancers of the lung, pancreas, stomach, prostate, oral cavity and all sites. The numbers of deaths except for lung and all sites were small but the pancreas cancer rate was still significantly higher than that for the city. White females in 2303 had an unremarkable overall cancer rate with excesses noted only for oral and rectal cancers of which only the latter ratio is significantly greater than unity.

In figure 3, we examine the lung cancer mortality in two or three year time intervals. Using rates adjusted by the direct method to the 1970

Baltimore City population as a standard, we find that the death rate for this cancer has always been higher in males from tract 2303 than from most controls but that it has been rising rapidly. The "all cancer" rates have also shown higher values than among controls. A preliminary look at the lung cancer rates for 1950-51 indicated that the adjusted rates for that period were high for tract 2303 with a rate of 253 per 100,000 population as compared to rates ranging from 35.8 to 87.4 in other index tracts and controls.

Employees of an industry may live in close proximity and could have accounted for an increased mortality in the census tract due to occupational exposure. With the cooperation of the company and the investigators studying the employees we reviewed lists of all employees to match with known deaths. Four employees were found among the cancer deaths in tract 2303 but removing these individuals did not change the significance of the rates.

The geographic distribution of cases was plotted on spot maps as shown in figures 4 and 5 for the two census periods. For both periods, lung cancer appears to be concentrated in an area about eight blocks wide and nine to twelve blocks long lying to the north and east of the plant. The area encompasses all of tract 2303 and parts of 2302 and 2301. If one takes all of tracts 2303, 2301 and 2302 which lie within a 3/4 mile radius of the plant and calculates the proportion of lung cancers to all cancers in this area compared to the remaining census areas on the figure 4, the proportion is 44.4 percent near the plant and 16.8 percent in the outer areas. For figure 5, the proportions show similar differences for 1973-74 as in the previous three years. For the area within the defined census tract and 3/4 miles of the plant, lung cancer represents 47.8 percent of the total cancers whereas in other areas it is only 33.3 percent. The northerly direction of this lung cancer excess is

not compatible with the strong wind directions in that area. These winds arise in the northwest and west and should have carried contaminants to the east and southeast of the plant. The particles may have been moved by gentler winds and deposited nearby.

Soil Sampling

The highest arsenic levels are shown in figure 6. In most cases these levels occurred at 2 inches suggesting higher contamination in the past. Occasionally, as in the area adjacent to the plant, the levels were highest at one inch. In general, arsenic levels were highest where lung cancer mortality was also highest. The mean arsenic level from 20 sample sites in tract 2303 was 63 ppm of arsenic. Even the omission of samples from the park adjacent to the plant only reduced the mean arsenic level to 38 ppm. Tracts 2301 and 2404 had means of 6 ppm and 2302 a mean of 4 ppm based on only 2 to 4 sample sites. All sites in the park had high levels except for an area which has been turned over and resodded and in which low arsenic levels were present. The one inch levels near the fence were as high as 695 and 226 ppm whereas at the opposite side of the park the values were only 29 to 97 ppm at one inch but as high as 46 to 161 ppm at two inches deep. From the soil levels of the original samples, the data indicated high levels within a 3/8 mile radius of the plant. It was also apparent that higher levels were found in a northerly direction along the railroad lines.

Hospital Validation

The deaths of the total 14 years of study were included in the sample and stratified by control and index census tracts. The sample for hospital record review included all deaths for residents in the index tracts. Deaths for control tracts were stratified by age, race, and sex and three time periods,

1958-62, 1966-67 and 1968-74. Four control deaths were selected randomly from each stratum for each index tract death within the same stratum. For the following analyses no attempt was made to expand the sample to the original population size.

The hospital abstract form included information on the following variables:

1. Cancer diagnosis
2. Final diagnosis other than cancer
3. Source of information for cancer diagnosis
4. Arsenic-associated symptoms
5. Personal characteristics as smoking and occupation
6. Description of pathological specimens; operative or autopsy findings

Verification of the identification of the correct individual on the hospital record was done by name, birthdate, residence, and date of death.

Records were reviewed in eleven of the hospitals in Baltimore City. The remaining five non-cooperating hospitals were small and did not limit substantially the number of records reviewed.

All possible medical conditions found on record review were listed and coded by the same nosologist who coded all the death certificates. The cell types were classified, in general, according to the Manual of Tumor Nomenclature and Coding. Since this coding scheme does not appropriately classify the cells of several tumors, especially those of non-solid origin, a revision of the scheme was made to include these cancers if we felt that their frequency was sufficient to warrant specific classification.

The causes of death were grouped into two time periods which represented the use of the 7th and 8th ICDA codes and grouped into causes as listed on the

certificate by the first two digits of the code. These causes were then compared to the first four medical conditions or diagnoses as noted on the hospital records.

The problem of validation of death certificate information was reviewed further by a physician who examined the data on the abstract forms. As indicated in table 6, there was complete agreement in diagnoses to four digits in the ICDA code in only 75.0 percent of the cancer deaths. If classification to three digits only is used we will correctly verify 80.7 percent of the cases listed on the certificate. In 1.8 percent of cases metastatic lesions were identified on the death certificate as underlying and in another 5.5 percent multiple cancers were listed on the certificate and the primary site varied from that listed on the hospital record. There was no cancer diagnosis listed anywhere on the hospital record for 2.7 percent of deaths. A further examination of the method of diagnosis of cases was attempted in order to demonstrate whether differences in the methods might have changed the accuracy of death certification. Data from autopsy and histological examination of tissue were used for the diagnosis of 82 percent of the cases with complete agreement in records and 88.9 percent of cases where the agreement was less than perfect. Therefore, the consistency of cancer diagnosis on hospital record and death certificate is not related to the method by which the cancer was identified.

The method of cancer diagnosis differed only slightly in the larger hospitals with 69.7 to 91.4 percent of cases diagnosed by autopsy or histology. An examination of the differences in diagnosis by census tract has not been completed, but it is unlikely that there will be variations in results since we have included all hospitals used by individuals from the index area in the above evaluation.

An examination of the hospital records for possible arsenic-associated symptoms included gastrointestinal signs, skin lesions, Mee's lines on nails, neurological or neuromuscular symptoms, cardiovascular disease, stroke and asthma. Only respiratory symptoms were slightly higher in the index tract but since there were so many records in which there was no comment about these symptoms, it is difficult to interpret the small variation. We also sought information on diseases for which arsenic might have been used as a treatment, such as syphilis, trypanosomiasis and amebiasis and the results indicated no higher frequency of these conditions among residents of index tracts.

Both smoking and drinking histories were abstracted from hospital records. Drinking habits were rarely recorded and smoking histories were also frequently missing. Table 7 indicates the smoking characteristics of lung cancer deaths in index and control tracts as determined from the hospital records. For 46.6 percent of the patients, the smoking histories are unknown. Despite that fact, we attempted to compare the smoking levels in index versus the control tracts. The percent of smokers is slightly higher in the index tracts but the difference is not impressive. If one includes only those charts with a recorded history, almost all cases are positive for smoking in both index and control tracts.

The original hypothesis was that if arsenic had caused the lung cancers, the cell type of lesions from the index tract might differ compared to other areas with an expected predominance of small cell or oat cell tumors in the exposed tracts. The data in table 8 would indicate that the cell types differ very little from index to control tracts.

DISCUSSION

An excess mortality from lung cancer has been demonstrated among men living in a highly industrialized area of South Baltimore over a period from 1966 through 1974. The death rate is significantly higher than in control tracts in the later years.

The area surrounding the pesticide plant has high levels of arsenic in the soil which corresponds generally to the same areas where a high proportion of lung cancers to other cancers has occurred. There was no attempt to correlate directly arsenic levels to residences of lung cancer deaths.

The review of hospital records did not indicate that the excess of lung cancer deaths had occurred because of variations in diagnostic practices, cell types or other factors. The information on other risk factors was poorly ascertained from hospital records.

There are some definite questions which arise in regard to the data. Why did the excess risk appear primarily in the late 60's and early 70's when the plant had existed and produced arsenical products since the early 1900's? The discrepancy could indicate that the plant did not account for the excess but some other local industry or occupational group accounts for the excess. It is also possible that the men in the area had a higher frequency of smoking and smoked a higher dose of cigarettes than did populations in the rest of the city. It is possible that selective mobility of younger, healthier males has left the area with a high risk among the remaining group.

The sudden rise in lung cancer might be related to the destruction of the old plant in 1952. Such an undertaking could have spread dust diffusely throughout the community. Under these circumstances we must ask why the concentration of lung cancer in the area does not coincide with the assumed wind spread of

particles. It is necessary to further examine the mortality in the 1950 period to determine whether an excess existed at any time before the destruction of the old plant. It would be interesting to see if the appearance of the excess risk of lung cancer in the community coincided with that found in the workers within the pesticide plant. If we presume that arsenic may not be causing the excess then it would be necessary to examine the mortality experience of workers in other industries in the area, especially the natural gas plant, to see if they have an excess lung cancer mortality. In almost all cases, workers within an industry should have higher exposure and a greater risk of disease than the general public. It is necessary to investigate whether the increase in lung cancer can be related to a change in production or methods of operation of any of the businesses. For example, differences in handling arsenic, changes in formulation of pesticides or the conversion of the gas plant from carburetted water gas to oil gas production could have created variations in level of type of pollution.

The fact that the excess lung cancer mortality has occurred only in men raises the question as to whether another environmental factor, differences in smoking characteristics, or occupation has caused the increased death rate in tract 2303. It is possible that smoking plus an environmental pollutant are required to produce the excess of cancer. The rates in older women then could be lower because they did not smoke and the possible synergistic effect of cigarettes and the environmental factor were not observed. Many of these questions might be determined by a community survey.

Further sampling for arsenic should be done to determine at what distance the levels actually returned to background. It was first thought that the high levels along railroad lines might indicate a relationship to previous coal use. However, further investigation showed that the arsenic content of coal

in local use did not reach levels as high as those measured along the tracks. Rail transport of materials from the plant may have been related to the high levels. Further investigation of this possibility is needed. It appeared that use of herbicides did not explain the arsenic levels in the rail beds.

In summary, men living in close proximity to a chemical plant which produced arsenicals have a higher risk of lung cancer than comparable individuals in other areas of the city. The distribution of arsenic in the soil near the plant and along the railroad line is higher than in control areas.

Table 1

Average Annual Crude Death Rate per 100,000 for Index and Control Census Tracts

Deaths 1958-62

Males

Match I

	2303		2302		2404		Adjacent		South		Central		North	
Cause	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate
Oral	0	-	2	21.1	1	9.5	5	19.3	10	8.3	20	7.6	11	8.9
Pancreas	1	18.5	0	-	2	19.0	3	11.6	8	6.6	31	11.8	18	14.6
Lung	4	74.1	12	126.8	8	75.9	23	88.8	79	65.4	193	73.6	60	48.8
Prostate	2	37.1	2	21.1	3	28.4	3	11.6	19	15.7	43	16.4	23	18.7
All Cancer	11	203.9	26	274.8	32	303.5	56	216.1	244	202.1	615	234.6	293	238.2

Match II

	2301				Adjacent				Central				North			
	White		Nonwhite		White		Nonwhite		White		Nonwhite		White		Nonwhite	
Cause	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate
Oral	0	-	0	-	1	21.8	0	-	0	-	0	-	3	18.4	0	-
Pancreas	0	-	1	16.8	0	-	1	22.9	1	17.7	1	18.9	2	12.2	0	-
Lung	3	75.2	2	33.5	2	43.6	1	22.9	5	88.7	6	113.6	12	73.4	8	39.3
Prostate	1	25.1	2	33.5	1	21.8	1	22.9	0	-	0	-	5	30.6	3	14.7
All Cancer	10	250.6	16	268.2	8	174.3	12	274.9	14	248.2	17	322.0	43	263.2	39	191.6

Table 2

Average Annual Crude Death Rate per 100,000 for Index and Control Census Tracts

Deaths 1968-74

Males

Match I

Census Tracts

	2303		2302		2404		Adjacent		South		Central		North	
Cause	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate
Oral	2	33.9	0	-	0	-	4	12.7	7	6.8	6	9.0	5	11.7
Pancreas	2	33.9	0	-	1	8.0	3	9.6	9	8.8	9	13.4	3	7.0
Lung	18	305.0	12	103.3	12	96.1	24	76.4	111	108.3	60	89.6	48	112.4
Prostate	1	16.9	1	8.6	2	16.0	4	12.7	15	14.6	25	37.3	7	16.4
All Cancer	33	559.2	26	223.8	24	192.2	86	273.9	254	247.9	206	307.7	110	257.7

Match II

	2301				Adjacent				Central				North			
	White		Black		White		Black		White		Black		White		Black	
Cause	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate
Oral	0	-	1	16.9	2	20.5	2	20.3	1	17.5	0	-	0	-	1	6.9
Pancreas	0	-	3	50.7	0	-	3	30.5	2	35.1	0	-	0	-	1	6.9
Lung	7	113.3	13	219.5	13	133.1	16	162.6	4	70.1	5	102.8	15	124.5	6	41.3
Prostate	1	16.2	3	50.7	4	41.0	4	40.6	1	17.5	2	41.1	3	24.9	3	20.7
All Cancer	17	275.0	27	455.9	34	348.2	42	426.7	17	298.0	13	267.2	41	340.3	28	192.9

Table 3

Average Annual Crude Death Rate per 100,000 for Index and Control Census Tracts

Deaths 1958-62

Females

Match I

	2303		2302		2404		Adjacent		South		Central		North	
Cause	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate
Oral	0	-	0	-	0	-	0	-	2	1.6	2	0.7	4	2.9
Pancreas	0	-	2	19.9	0	-	0	-	8	6.4	23	8.4	12	8.8
Lung	0	-	0	-	0	-	6	22.6	8	6.4	18	6.5	15	11.0
Breast	3	57.5	2	19.9	3	28.7	8	30.2	28	22.5	81	29.4	51	37.5
Cervic	0	-	1	10.0	1	9.6	3	11.3	15	12.0	34	12.4	12	8.8
All Cancer	6	115.1	17	169.2	13	124.4	13	162.3	186	149.3	460	167.2	263	193.3

Match II

	2301				Adjacent				Central				North			
	White		Nonwhite		White		Nonwhite		White		Nonwhite		White		Nonwhite	
Cause	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate
Oral	0	-	0	-	1	27.4	0	-	0	-	0	-	0	-	0	-
Pancreas	0	-	0	-	1	27.4	1	27.3	0	-	0	-	1	5.8	0	-
Lung	0	-	1	15.8	0	-	0	-	1	19.7	0	-	1	5.8	0	-
Breast	3	71.4	2	31.5	3	82.2	1	27.3	2	39.5	1	17.1	8	46.6	4	18.3
Cervix	1	23.8	1	15.8	1	27.4	1	27.3	2	39.5	1	17.1	5	29.1	3	13.8
All Cancer	6	142.9	10	157.6	10	274.0	8	218.6	9	177.7	6	102.6	41	238.9	21	96.3

Table 4

Average Annual Crude Death Rate per 100,000 for Index and Control Census Tracts

Deaths 1968-74

Females

Match I

	2303		2302		2404		Adjacent		South		Central		North	
Cause	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate
Oral	2	33.3	0	-	0	-	4	12.1	3	2.7	3	4.1	2	4.3
Pancreas	0	-	2	16.8	2	15.0	1	3.0	11	9.9	10	13.6	1	2.2
Lung	1	16.6	3	25.2	1	7.5	7	21.1	16	14.4	18	24.5	9	19.5
Breast	1	16.6	1	8.4	2	15.0	8	24.1	25	22.5	29	39.5	9	19.5
Cervix	0	-	3	25.2	0	-	3	9.1	6	5.4	5	6.8	0	-
All Cancer	9	149.9	14	117.8	17	127.3	56	169.0	165	148.7	175	238.2	78	169.2

Match II

	2301				Adjacent				Central				North			
	White		Nonwhite		White		Nonwhite		White		Nonwhite		White		Nonwhite	
Cause	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate
Oral	0	-	0	-	0	-	1	9.7	0	-	0	-	0	-	0	-
Pancreas	1	16.4	0	-	1	10.9	2	19.3	1	16.7	0	-	3	23.0	0	-
Lung	2	32.8	1	15.4	2	21.9	7	67.6	1	16.7	1	18.1	3	23.0	0	-
Breast	5	82.0	3	46.2	6	65.7	4	38.6	2	33.4	0	-	8	61.4	3	18.1
Cervix	0	-	2	30.8	3	32.8	1	9.7	0	-	1	18.1	2	15.4	1	6.0
All Cancer	15	246.0	18	277.1	26	284.6	25	241.5	14	233.6	5	90.5	34	261.1	16	96.3

Table 5

Time-Adjusted SMR's Based on Average Annual Baltimore City Rates*

Deaths 1958 - 1962 and 1966 - 1974

Match I White Males

Cause	2303 obs	2303 SMR	2302 obs	2302 SMR	2401 obs	2401 SMR	Adjacent Controls obs	Adjacent Controls SMR	South Controls obs	South Controls SMR	Central Controls obs	Central Controls SMR	North Controls obs	North Controls SMR
Oral	3	2.45	2	0.88	1	0.43	9	1.43	18	0.88	29	0.91	16	0.89
Stomach	2	1.60	3	1.18	4	1.59	10	1.44	23	0.98	60	1.40	17	0.70
Colon	3	1.22	9	1.74	6	1.21	9	0.62	43	0.94	67	0.91	38	0.90
Rectum	1	1.09	3	1.62	4	2.19	6	1.18	14	0.85	34	1.25	15	0.97
Pancreas	5	4.15	0	-	3	1.31	7	1.09	21	1.01	43	1.27	21	1.10
Lung	25	2.74	30	1.72	23	1.31	57	1.19	206	1.35	282	1.21	118	0.93
Prostate	3	1.59	3	0.68	5	1.26	7	0.56	36	0.93	74	1.15	31	0.79
Bladder	0	-	3	1.38	2	0.97	5	0.85	20	1.03	37	1.11	28	1.45
Lymphomas	2	0.88	4	0.89	2	0.44	10	0.80	26	0.62	65	1.02	37	1.06
All Cancer	54	1.94	67	1.21	63	1.15	168	1.10	543	1.10	891	1.13	423	0.96

Match I White Females

Cause	2303 obs	2303 SMR	2302 obs	2302 SMR	2404 obs	2404 SMR	Adjacent Controls obs	Adjacent Controls SMR	South Controls obs	South Controls SMR	Central Controls obs	Central Controls SMR	North Controls obs	North Controls SMR
Oral	2	6.31	0	-	1	1.40	4	2.06	5	0.84	5	0.57	6	1.14
Stomach	1	1.32	3	1.74	0	-	5	0.96	14	0.89	37	1.30	18	1.01
Colon	1	0.45	7	1.36	5	0.94	13	0.83	41	0.89	81	1.01	40	0.82
Rectum	4	6.12	0	-	2	1.32	3	0.69	14	1.08	33	1.57	13	1.03
Pancreas	0	-	4	2.32	2	1.13	1	0.19	20	1.34	38	1.59	13	0.90
Lung	1	0.71	4	1.34	1	0.31	15	1.70	26	0.99	37	1.05	23	1.14
Breast	5	1.29	5	0.61	6	0.68	17	0.70	58	0.75	119	0.94	61	0.82
Cervix	0	-	5	2.30	3	1.27	6	0.97	25	1.13	43	1.14	13	0.61
Bladder	0	-	0	-	0	-	0	-	10	1.33	15	1.15	6	0.74
Lymphomas	1	0.65	4	1.19	3	0.86	6	0.61	36	1.22	52	1.05	24	0.83
All Cancer	18	0.96	41	1.00	39	0.91	109	0.90	390	1.05	682	1.10	350	0.96

* Average annual Baltimore City rates (based on deaths in 1958-1962) were applied to the 1960 match population and were weighted for five years. Average annual Baltimore City rates (based on deaths in 1968-1972) were applied to the 1970 match population and were weighted for nine years.

Table 6

Level of Agreement between Death Certificate Cancer Cause and Hospital Diagnosis

	Number	%	
Complete Agreement (4 digits in ICDA code)	555	75.0	80.7
Agreement to 3 digits	42	5.7	
Agreement to 2 digits	31	4.2	
Metastasis entered on D.C. as underlying	13	1.8	
Multiple cancers on D.C. Primary site not stated	41	5.5	
Other	18	2.4	
No cancer at autopsy or biopsy	20	2.7	
No records available	20	2.7	
Total	740		

Table 7

Smoking History in Lung Cancers from Index and Control Tracts by Sex

	Smoking		Non-Smoking		Unknown		Total
	No.	%	No.	%	No.	%	No.
Index Tracts							
Male	30	59	2	4	19	37	51
Female	6	67	3	33	0	0	9
Control Tracts							
Male	63	52	0	0	58	48	121
Female	5	22	0	0	18	78	23

Table 8

Cell Types of Lung Cancer by Census Tract

	Oat	Squamous	Adenocarcinoma	Epidermoid	Other	UK	Total
Tract 2303							
Male	2	8	-	1	1	4	16
Female	-	1	1	-	-	-	2
Other Index							
Male	3	18	2	1	1	8	33
Female	-	3	3	-	1	-	7
Control							
Male	12	57	16	4	3	18	110
Female	4	4	9	-	4	1	22

Figure 1. Map of Baltimore City showing location of 1970 index and control census tracts.

Figure 2. Map of Baltimore City showing location of 1960 index and control census tracts.

Figure 3. Age- and time-adjusted rates per 100,000 for tract 2303 and North and Adjacent Controls. White males. All cancer and lung cancers. (semi-logarithmic scale)

Figure 4. Spot map showing cancer deaths for 1970-72 by residence at death, excluding chemical plant employees.

Figure 5. Spot map showing cancer deaths for 1973-74 by residence at death.

Figure 6. Arsenic level in soil - ppm. Highest value at each site. Summer 1976 and Spring 1977

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DR. HUNT: I think we do have about five minutes for questions. We can proceed on that basis. Are there any questions for Dr. Matanoski?

DR. O'CONOR: It is a very impressive study. I have two questions. One has to do with the characteristics or definition of the population in the indexed census tracts and how that compares with the control census tract. The other has to do with similar industries to the one that is now apparently defunct in your area. What kind of controls or regulations are in effect now for plants to benefit from the kind of experience which you have described?

DR. MATANOSKI: The control tracts were actually matched for the several characteristics to the index tract, namely age, race, sex, socioeconomic status and percent at poverty level. So they were similar in these characteristics with very small deviation. One thing I did not emphasize is that the data that you have seen was subsequently examined with employees excluded, and that did not change the observed difference, the epidemic still persisted. We had a list of all employees provided through the industry which allowed us to accomplish this task.

Your second question related to what my advice would be for control of this situation. The problem in this plant was apparently from dust. At least when we observed the operation externally, it had a very high dust level and the material was circulating very close to the ground. The plant did not have high stacks. Thus, the material was not moved very far away. We had not anticipated this distribution of arsenic. We expected the material perhaps to be carried by wind currents away from the plant and further out into the population. Instead, the arsenic remained in the very community close to the plant. If one could manage local dust problems, this type of spread could be avoided.

NIOSH

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The papers included in these Proceedings were printed as they were submitted to this office.

Appropriate portions of the discussions, working groups and plenary session were sent to the participants for editing. The style of editing varied, as could be expected. To the extent possible, we have attempted to arrive at a consistent format.

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