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Long-Term Mortality Study of Steelworkers

V. Respiratory Cancer in Coke Plant Workers

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"I take it that no pathological question is ever so thoroughly settled or so deeply buried but that it contains the germs of new questions in pathology, often more complex and more difficult to answer than the original question. Sooner or later these present themselves, not always in a welcome fashion, and not always to the original inquirer, but with a repeated intrusion which at length compels attention." —Butlin

In the fourth paper of this series we reported that mortality from respiratory cancer for men employed in the coke plant is two times the rate observed generally among steelworkers. We also noted that all of the difference in mortality from this disease is due to a three-fold excess for nonwhite workers.[1]

The purpose of the present paper is to delineate further the source of the respiratory cancer excess within the coke plant environment; to clarify, to the extent possible, the apparent differential in mortality for white and nonwhite workers; and to relate the differences observed to other reports of unusual cancer mortality in occupations with related environmental exposures.

By reference to work histories, the study population is further classified according to calendar period and duration of employment within several subdivisions of the coke plant. Race-specific mortality for malignant neoplasm of the respiratory system and for other selected causes is presented for each subdivision. The patterns of mortality observed within these groups are then evaluated by comparison with expected mortality based on total steelworker population experience. Finally, we contrast the cancer experience of the several groups of coke plant workers with that reported for other industrial groups with similar environmental exposures, and consider the implications of differences in site-specific cancer mortality.

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Background

The primary function of the coke plant is the production of metallurgical coke for use at the blast furnace. A secondary function is the recovery of chemical by-products during the transformation (high temperature carbonization) of bituminous coal into coke. Prior to World War I the main source of metallurgical coke in the United States was the beehive coke oven. These ovens were used solely for the production of coke, and the volatile matter produced during carbonization was emitted into the atmosphere. The by-product coke plant, which allows for recovery of tar, oils and chemicals from the volatiles, was introduced around the turn of the century and by 1931 by-product ovens accounted for 80 per cent of coke production. In recent years, except for brief periods during World War II and the Korean War, this figure has been about 95 per cent.[2] All of the coke plant workers included in the present report were employed at by-product plants.

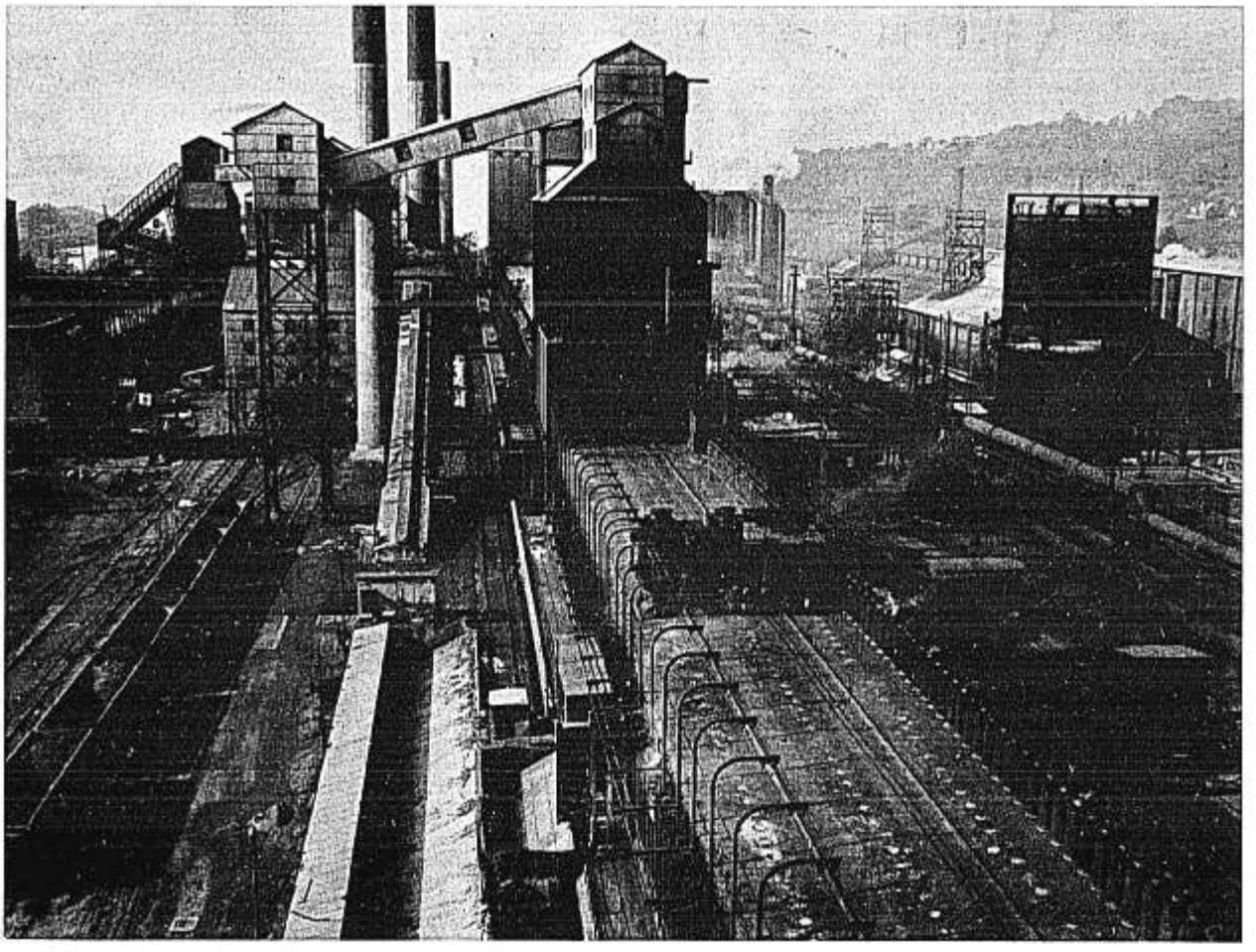
In addition to the production of metallurgical coke, there are several other methods of coal carbonization which are of interest because of common exposure to the volatiles and their by-products, and there are previous reports of unusual cancer experience in workmen employed in these areas. Major among these processes

are the production of household gas in vertical and horizontal retorts and the generation of producer gas for industrial use.

Materials and Methods

One of the most challenging tasks in the long-term study of occupational disease is the classification of the work force according to distinctive environmental exposures. Even when detailed work histories are available, as in the present study, the variable use of many job titles and abbreviations among firms and over long periods of time makes categorization difficult.[3] Classification according to well-defined work areas is also precluded by the use of generic terms which describe neither the specific occupation nor the specific work area, or by the use of titles appropriate to more than one work area. The title "laborer, coke plant," for example, defies categorization in the absence of other information. Fortunately, the great majority of coke plant titles noted in the steelworker histories could be assigned to discrete work areas. It should be noted, however, that the classification scheme used for the present report produces some changes in work area designation from that shown in an earlier report.[4] This results primarily from further clarification of obsolete job titles and a reinvestigation of work histories which included generic titles. These reclassifications produce only slight changes in mortality by work area from that reported earlier.

The modern by-product coke plant is a semicontinuous operation which may be subdivided into three rather distinct work areas in terms of function and potential exposure to environmental hazards. These are: (1) the coal handling area where coal is received by rail or barge and where provision is made for the handling, storage and blending of several types of coal before transfer to the coke ovens; (2) the coke ovens, grouped into one or more batteries, with equipment for charging and discharging the ovens and the quenching of coke; and (3) a by-products plant for recovery of gas and chemical products.[5] Variation in work area definition among plants and our desire to subdivide the coke plant population according to potential areas of exposure required some interpretation of work area designations stated in the occupational history. Because of prior reports of higher lung cancer rates for men engaged primarily in the coal carbonization process,[6,7] our interest centered on men employed at the coke ovens or in the immediate vicinity. Therefore, certain occupational titles indicating employment some distance from the coke ovens were assigned to a nonoven group,



Coke oven battery showing larry car in position for charging an oven. The pushing machine is on the right side of the battery aligned with the larry car. The quencher car is on the left side, adjacent to the coke wharf. (Photo courtesy of American Iron and Steel Institute.)

even though the work area noted in the occupational history was "ovens." The nonoven group included all job titles which designate employment only in the coal handling and by-product areas and general coke plant occupations which would rarely require employment in the immediate vicinity of the coke ovens. Conversely, certain coke plant maintenance titles which require periodic employment at the ovens were included in the coke oven group, although the indicated work area may not have been "ovens." The coke oven group includes all job titles requiring some part of the working day spent at the topside of the ovens or the side of the ovens, including the quenching station, the coke wharf and the coke screening station. In this way each period of employment for the individual coke plant workers was designated as coke oven or non-coke oven.

The mortality experience from 1953 to 1961 of men employed in each of the two areas and certain subdivisions is contrasted with the experience predicted by the age-, race- and nativity-specific rates for the total population of steelworkers,[1] and significant differences are determined by a summary χ-square test.[8]

The Population At Risk

The number of men employed in coke oven and nonoven areas in 1953 and in prior years is shown in Table I. Of the 58,828 steelworkers employed in 1953, 2,552 worked in the coke plant. However, an additional 978 steelworkers employed in other work areas in 1953 had been employed at the coke plant in some prior year. We have noted previously the effect of excluding part of the population at risk by restricting the study group to those employed at a single point in time.[1] In the present case, limiting the study to men employed in 1953 would exclude 28 per cent of men with prior coke plant exposure and 35 per cent of men with prior coke oven exposure.

Differing distributions by race within the two work areas of the coke plant are seen for 1953 and for prior years. Eighty-nine per cent of nonwhite workers were employed at the coke ovens in 1953 compared to only 32 per cent of white workers. A marked difference in these proportions is still evident after inclusion of men employed in the coke plant only in prior years (nonwhite, 91 per cent; white, 42 per cent). It is also interesting to note that a greater proportion of white coke oven workers would be excluded by limiting work area classification to a single year. Forty-eight per cent of white workers with prior coke oven exposure were not employed at the coke oven in 1953, whereas only 24 per cent of nonwhite coke oven workers were in this category.

Mortality of Men Employed in 1953

Observed and expected deaths by area of employment in 1953 are shown in Table II. All causes mortality for coke oven and nonoven workers is seen to be only slightly higher than that predicted by total steelworker experience, with both groups showing the same relative risk (SMR = 104). On the other hand, both groups show significant deviation from expectation for certain causes of death. The excess mortality from respiratory neoplasms for coke plant workers is seen to be limited to men employed at the coke ovens with 20 deaths observed, compared to 7.5 expected. In contrast, the nonoven workers, whose mortality from respiratory neoplasms is about as expected, show a significant excess of deaths from nonmalignant diseases of the respiratory system (8 observed versus 3.7 expected).

The mortality patterns by race for coke oven and nonoven workers employed in 1953 present a mixed picture. A three-fold excess for nonwhite workers accounts for almost all of the difference in respiratory neoplasm mortality for coke oven workers (17 observed versus 5.7 expected). However, the experience of nonwhite workers employed in the nonoven area suggests a similar risk (4 observed versus 1.1 expected). White workers employed in the nonoven area show a deficit of deaths from respiratory neoplasms, while the experience of white coke oven workers is consistent with an excess, but based on insufficient numbers to draw conclusions (3 observed versus 1.9 expected). Both white and nonwhite workers employed in the nonoven area in 1953 show

TABLE I

DISTRIBUTION OF COKE PLANT WORKERS BY WORK AREA, RACE AND PERIOD OF EMPLOYMENT

	Employed in 1953				
	Coke Plant	Coke Oven		Nonoven	
		Number	Per Cent	Number	Per Cent
Total	2,552	1,327	52.0	1,225	48.0
White	1,645	520	31.6	1,125	68.4
Nonwhite	907	807	89.0	100	11.0

	Employed in 1953 or Prior Years				
	Coke Plant	Coke Oven		Nonoven	
		Number	Per Cent	Number	Per Cent
Total	3,530	2,048	58.0	1,482	42.0
White	2,369	993	41.9	1,376	58.1
Nonwhite	1,161	1,055	90.9	106	9.1

TABLE II

OBSERVED AND EXPECTED DEATHS AND STANDARDIZED MORTALITY RATIOS FOR SELECTED CAUSES BY RACE, 1953 TO 1961; MEN EMPLOYED IN COKE PLANT AND SUBDIVISIONS IN 1953

Cause of Death	Coke Plant			Coke Oven			Nonoven		
	Observed Deaths	Expected Deaths	SMR*	Observed Deaths	Expected Deaths	SMR*	Observed Deaths	Expected Deaths	SMR*
					TOTAL				
All causes	206	198.9	104	100	96.6	104	106	102.3	104
Malignant neoplasms—respiratory system	25	14.3	175†	20	7.5	267†	5	6.8	74
Malignant neoplasms—digestive organs and peritoneum	16	14.8	108	4	6.5	62	12	8.3	145
Other malignant neoplasms	18	14.8	122	10	7.6	132	8	7.0	114
Vascular lesions affecting central nervous system (CNS)	19	16.2	117	7	7.9	89	12	8.3	145
Heart disease	64	73.0	88	22	31.7	69	42	41.3	102
Diseases of respiratory system	12	7.7	156	4	4.0	—	8	3.7	216‡
All other causes	52	58.1	90	33	31.3	105	19	26.8	71
					WHITE				
All Causes	114	121.1	94	25	31.4	80	89	89.7	99
Malignant neoplasms– respiratory system	4	7.5	53	3	1.9	—	1	5.7	18
Malignant neoplasms—digestive organs and peritoneum	11	9.8	112	1	2.4	—	10	7.4	135
Other malignant neoplasms	7	8.0	88	2	2.1	—	5	6.0	83
Vascular lesions affecting CNS	13	9.4	138	1	2.3	—	12	7.2	167
Heart disease	47	50.2	94	9	12.8	70	38	37.4	102
Diseases of respiratory system	6	4.1	146	0	1.0	—	6	3.1	194
All other causes	26	31.9	82	9	8.9	101	17	23.0	74
					NONWHITE				
All causes	92	77.8	118	75	65.2	115	17	12.6	135
Malignant neoplasms—respiratory system	21	6.8	309†	17	5.7	298†	4	1.1	—
Malignant neoplasms—digestive organs and peritoneum	5	4.9	102	3	4.1	—	2	0.8	
Other malignant neoplasms	11	6.7	164	8	5.6	143	3	1.2	—
Vascular lesions affecting CNS	6	6.8	88	6	5.6	107	0	1.1	—
Heart disease	17	22.8	75	13	18.9	69	4	3.9	—
Diseases of respiratory system	6	3.6	167	4	3.0	—	2	0.6	—
All other causes	26	26.2	99	24	22.4	107	2	3.8	—

*SMR = $\frac{\text{Observed deaths}}{\text{Expected deaths}}$ x 100. Significance based on summary χ-square with 1 degree of freedom.[8]

†1% level

‡5% level

—less than five deaths

TABLE III

OBSERVED AND EXPECTED DEATHS AND STANDARDIZED MORTALITY RATIOS FOR SELECTED CAUSES BY RACE, 1953-1961; MEN EMPLOYED IN COKE PLANT AND SUBDIVISIONS IN 1953 AND PRIOR YEARS

Cause of Death	Coke Plant			Coke Oven			Nonoven		
	Observed Deaths	Expected Deaths	SMR	Observed Deaths	Expected Deaths	SMR	Observed Deaths	Expected Deaths	SMR
					TOTAL				
All causes	320	300.5	106	184	173.1	106	136	127.4	107
Malignant neoplasms—respiratory system	37	21.8	170†	33	13.3	248†	4	8.5	47
Malignant neoplasms—digestive organs and peritoneum	27	21.5	126	9	11.8	76	17	9.7	175‡
Other malignant neoplasms	22	23.7	93	16	14.1	113	6	9.6	62
Vascular lesions affecting CNS	28	24.3	115	15	14.2	106	13	10.1	129
Heart disease	111	113.2	98	49	61.5	80	62	51.7	120
Diseases of respiratory system	14	11.3	124	7	6.8	103	7	4.5	156
All other causes	81	84.7	96	55	51.4	107	27	33.3	81
					WHITE				
All causes	199	193.8	103	80	80.2	100	119	113.6	105
Malignant neoplasms—respiratory system	11	12.2	90	8	5.0	160	3	7.3	41
Malignant neoplasms—digestive organs and peritoneum	21	14.9	141	6	6.1	98	14	8.8	159
Other malignant neoplasms	10	14.0	71	6	5.8	103	4	8.2	49
Vascular lesions affecting CNS	17	15.3	111	6	6.4	94	11	8.9	124
Heart disease	87	81.5	107	29	32.9	86	58	47.6	122
Diseases of respiratory system	7	6.5	108	2	2.5	—	5	3.9	128
All other causes	46	49.3	93	23	20.5	112	24	28.9	83
					NONWHITE				
All causes	121	106.7	113	104	92.9	112	17	13.8	123
Malignant neoplasms—respiratory system	26	9.6	271†	25	8.4	298†	1	1.2	—
Malignant neoplasms—digestive organs and peritoneum	6	6.6	91	3	5.7	53	3	0.9	—
Other malignant neoplasms	12	9.7	124	10	8.3	120	2	1.4	—
Vascular lesions affecting CNS	11	9.0	122	9	7.8	115	2	1.2	—
Heart disease	24	31.6	76	20	27.6	72	4	4.1	—
Diseases of respiratory system	7	4.8	146	5	4.2	119	2	0.6	—
All other causes	35	35.3	99	32	31.0	103	3	4.4	—

† 1% level
‡ 5% level
— less than five deaths

TABLE IV

OBSERVED AND EXPECTED DEATHS AND STANDARDIZED MORTALITY RATIOS FOR MALIGNANT NEOPLASMS OF DIGESTIVE ORGANS AND PERITONEUM BY LENGTH OF EMPLOYMENT AND SITE 1953-1961; MEN EMPLOYED ONLY IN NONOVEN AREA IN 1953 AND PRIOR YEARS

	Total			Five or More Years of Employment			Less than Five Years of Employment		
	Observed	Expected	SMR	Observed	Expected	SMR	Observed	Expected	SMR
All malignant neoplasms of digestive system	17	9.7	175‡	12	6.7	180	5	3.0	167
Stomach	4	3.0	–	3	2.1	–	1	0.9	–
Intestines except rectum	7	2.8	249‡	6	1.9	316†	1	0.9	–
Rectum	1	1.1	–	0	0.8	–	1	0.4	–
Pancreas	3	1.3	–	2	0.9	–	1	0.4	–
Other digestive organs	2	1.5	–	1	1.0	–	1	0.4	–

† 1% level
‡ 5% level
– less than five deaths

excess mortality from diseases of the respiratory system, but neither difference is significant.

Mortality of Men Ever Employed in the Coke Plant

To further clarify the differences in mortality between coke oven and nonoven workers, we next consider the experience of men employed in these areas in 1953 or in prior years. For this purpose, the population of men employed at the coke plant during that period is subdivided into those who had ever worked at the coke ovens and those who had never worked at the coke ovens. The mortality experience of these two groups (designated coke oven and nonoven) is depicted in Table III. Three hundred twenty deaths from all causes were observed among men employed in the coke plant in 1953 or prior years, an increase of 55 per cent over the number observed among men employed in 1953. The inclusion of men employed prior to 1953 has also allocated an additional 84 deaths to the coke oven area, an increase of 84 per cent.

An excess of mortality from all causes is noted for men who had ever been employed at the coke plant, the difference being somewhat greater than observed for men employed in 1953. This increase, which is observed for both oven and nonoven workers, indicates a higher mortality for men employed in prior years. A significant excess of respiratory neoplasms is again noted for coke oven workers, with observed deaths being almost 2 1/2 times the number expected. It is interesting to note that 13 of the 33 respiratory neoplasm deaths in this group were among men employed at the coke ovens prior to 1953, but not employed in that area in 1953. A comparison of the observed and expected deaths in Tables II and III shows that coke oven workers employed only prior to 1953 experienced more than twice the number of expected deaths from respiratory neoplasms.

Examination of the differences by race again shows that respiratory neoplasm mortality for nonwhite coke oven workers is significantly different from expectation, with an almost three-fold excess (25 versus 8.4). Of the 25 deaths, 23 were attributed to malignant neoplasm of the lung. These data also indicate that white coke oven workers may be more liable to respiratory neoplasms with eight deaths observed compared to five expected, but the difference is not significant. All of the eight respiratory neoplasm deaths among white coke oven workers were attributed to neoplasm of the lung. Further examination of Table III shows that coke oven worker mortality from diseases other than malignant neoplasm of the lung is little different from expectation.

Subdivision of the population by areas of prior employment also demonstrates that nonoven workers are not at excess risk for respiratory neoplasms. Of the 25 respiratory neoplasm deaths in nonwhite coke oven workers, eight (32 per cent) were among men employed at the coke oven prior to 1953 only. The apparent excess for nonwhites employed in the nonoven area in 1953 is, therefore, explained by allocation of former coke oven workers to the nonoven area. There is no indication in these data that either white or nonwhite coke plant workers employed only in the nonoven area experienced other than the expected level of respiratory neoplasm deaths.

Further examination of the mortality pattern for men employed only in the nonoven areas shows that the excess for diseases of the respiratory system, while still apparent, is no longer significant (7 versus 4.5). On the other hand, a significant excess of neoplasms of the digestive organs is now observed for nonoven workers (17 versus 9.7). Although neither of the differences by race is significant, they suggest that both white (14 versus 8.8) and nonwhite workers (3 versus 0.9) may be more liable to death from this cause. The breakdown of nonoven worker deaths from malignant neoplasms of digestive organs is displayed in Table IV. Here it is seen that the excess is observed for both long- and short-term employees and is not limited to a single site. The greatest differences are noted for malignant neoplasm of the pancreas and malignant neoplasm of intestine excluding rectum, the latter difference being significant for men

employed five or more years (6 versus 1.9). Because population figures are not immediately available for the subdivisions of the nonoven area, the possible relationship to employment in more specific work areas cannot be considered at present. However, examination of work histories of the decedents does not suggest that the differences are associated with employment in any single area. Of the three men dying from cancer of the pancreas, one had been employed in the by-products area and two had been employed in the coal handling area. Of the six deaths from cancer of the intestine observed for men with more than five years of employment, four had worked in coal handling, and two had worked in by-products. Although the proportion of foreign-born among nonoven workers is little different than for all steelworkers (17 per cent versus 15 per cent), it is also interesting to note that three of these six men were foreign-born (two Hungarians and one Italian) and two were of foreign parentage (Austrian and German). Higher digestive cancer rates have been reported for immigrants from several of these countries by Haenszel.[9]

Although these findings suggest that men employed in the nonoven area may be more liable to death from digestive cancers, the observation of comparable risks for long- and short-term workers, the nonspecificity by work area, the concentration of foreign-born, and the paucity of numbers suggest caution in attributing these differences to exposure in the coke plant environment. Records of steelworker deaths for 1961 to 1966 are now being collected, and when the information becomes available this question will be re-examined.

Subdivision of the Coke Oven Population

To further delineate the relationship between lung cancer mortality and prior employment at the coke ovens, we next consider length of employment and differential mortality within the several work divisions of the coke ovens. A brief description of the coke oven operation will clarify the further subdivision of the study population. Before charging of a coke oven, the doors on the ends are closed to effect complete sealing. The oven is then charged with 16 to 20 tons of coal through ports at the top, and the ports are sealed. When coking is completed (16 to 20 hours), the doors at the ends of the oven are opened and a pusher ram shoves the entire charge of coke into a railway car. After the car has been moved to a water quenching station, the coke is taken to a wharf for further cooling and later screening.[5] To provide efficient operation the coke ovens are grouped into batteries of 10 to 100 ovens in a parallel configuration. In terms of exposure to coke oven effluents, the work force can be divided into those who work on the top of the batteries, and who have considerable exposure, and men who work at the side of the ovens and have less exposure. A further subdivision can be made in terms of time spent at the topside of the ovens. There are three occupations which require full-time work on top of the coke batteries. These are the larry car operator who charges the ovens, the lidman who opens and seals the ports and the standpipe man who maintains an offtake pipe through which the volatiles pass. Several other occupations require only occasional periods of work at the topside of the battery. Included here are the foreman, the heater and occasional maintenance workers such as pipefitters. The remainder of the coke oven work force are employed at the side of the oven, which includes the quenching station, the coke wharf and the screening station.

As shown in Table V, the excess mortality in coke oven workers is associated primarily with employment at the full-time topside occupations. The total mortality experience of men employed only at the side of the oven differs little from expectation, and in contrast with the previously noted differential by race, both white and nonwhite workers show similar patterns of relative risk. Moreover, the differential in lung cancer risk for men employed only at the side of the ovens is the reverse of that previously noted, with nonwhite workers showing no excess (5 versus 5.4) while white workers experienced more lung cancer deaths than expected (5 versus 2.7). Mortality for men employed in partial topside jobs also shows little deviation from that predicted and no excess of lung cancer is noted.

Deaths among men employed at the full-time topside jobs account for all of the coke oven worker excess in total mortality and almost all of the excess mortality from cancer of the lung. Mortality from all causes for these men is 68 per cent greater than expected, the same relative risk being indicated for both white and nonwhite workers. The observed deaths from malignant neoplasm of the lung for full-time topside workers are seven times expectation (19 versus 2.6), the risk for nonwhite workers being eight times greater than expected (18 versus 2.2). The limitation of small numbers precludes a conclusion concerning the lung cancer risk for the white workers (1 versus 0.5). It should be noted, however, that the differing lung cancer response for white and nonwhite coke plant workers is now seen to be due in large part to the differing distribution by work area (19.1 per cent of nonwhites were employed at full-time topside jobs compared to 3.4 per cent of whites) and the unusually high lung cancer rate for full-time topside workers. The lung cancer experience of coke plant workers never employed at full-time topside jobs is comparable to that observed for other steelworkers, and the relative risks for white (10 versus 11.2) and nonwhite workers (5 versus 6.5) do not suggest a differential response by race. Nonwhite workers employed at full-time topside jobs also experience more than the expected number of deaths from other malignant neoplasms (7 versus 4.3), but the difference is not significant.

The findings in Table V also serve to illustrate how occupationally related disease might be masked by limiting study to broad occupational groups. Since the full-time topside workers constitute only 15 per cent of coke oven workers and only 9 per cent of coke plant workers, the seven-fold lung cancer risk for topside workers is reflected as a considerably lower relative risk for the broader work groups (coke oven, 2.5; coke plant, 1.7). If the lung cancer rate for full-time topside workers

TABLE V

NUMBER EMPLOYED, OBSERVED AND EXPECTED DEATHS AND STANDARDIZED MORTALITY RATIOS FOR SELECTED CAUSES BY RACE, 1953-1961; MEN EMPLOYED IN SUBDIVISIONS OF THE COKE OVEN IN 1953 AND PRIOR YEARS

Work Area and Race	Number Employed	All Causes			Malignant Neoplasms of Lung			Other Malignant Neoplasms			Heart Disease			All Other Causes		
		Observed Deaths	Expected Deaths	SMR	Observed Deaths	Expected Deaths	SMR	Observed Deaths	Expected Deaths	SMR	Observed Deaths	Expected Deaths	SMR	Observed Deaths	Expected Deaths	SMR
Total coke oven	2048	184	173.1	106	31	12.3	252†	27[3]	26.9	100	49	61.5	80	77	72.4	106
White	993	80	80.2	100	8	4.7	170	12	12.2	98	29	33.9	86	31	29.4	105
Nonwhite	1055	104	92.9	112	23	7.6	303†	15[3]	14.8	101	20	27.6	72	46	43.0	107
Side oven[1]	1431	106	114.6	92	10	8.0	125	13[3]	17.5	74	34	40.0	85	49	49.1	100
White	606	45	47.2	95	5	2.7	185	6	7.3	82	18	19.9	90	16	17.4	92
Nonwhite	825	61	67.3	91	5	5.4	93	7[3]	10.3	68	16	20.1	80	33	31.6	104
Partial topside[2]	315	24	26.3	91	2	1.7	—	6	4.0	150	5	10.9	46	11	9.7	113
White	307	22	25.1	88	2	1.6	—	5	3.8	132	5	10.5	48	10	9.2	109
Nonwhite	8	2	1.2	—	0	0.1	—	1	0.2	—	0	0.3	—	1	0.5	—
Full topside	302	54	32.2	168†	19	2.6	731†	8	5.4	148	10	10.6	94	17	13.6	125
White	80	13	7.8	167	1	0.5	—	1	1.2	—	6	3.5	171	5	2.8	179
Nonwhite	222	41	24.4	168†	18	2.2	818†	7	4.3	163	4	7.2	56	12	10.8	111

† 1% level
‡ 5% level
— less than five deaths

[1] Never partial or full topside
[2] Never full topside
[3] Includes two respiratory cancer deaths other than lung

TABLE VI

NUMBER EMPLOYED, OBSERVED AND EXPECTED DEATHS AND STANDARDIZED MORTALITY RATIOS FOR SELECTED CAUSES, 1953-1961 BY LENGTH OF EMPLOYMENT AS OF JANUARY 1, 1953, AND RACE; MEN EMPLOYED AT COKE OVENS IN 1953 AND PRIOR YEARS

Length of Coke Oven Employment and Race	Number Employed	All Causes			Malignant Neoplasms of Lung			Other Malignant Neoplasms			Heart Diseases			All Other Causes		
		Observed Deaths	Expected Deaths	SMR	Observed Deaths	Expected Deaths	SMR	Observed Deaths	Expected Deaths	SMR	Observed Deaths	Expected Deaths	SMR	Observed Deaths	Expected Deaths	SMR
Less than five years	1,144	67	72.8	92	4	4.7	—	7	10.5	67	24	25.7	93	32	31.8	101
White	593	41	39.1	105	3	2.2	—	5	5.9	85	15	16.1	93	18	14.9	121
Nonwhite	551	26	33.8	77	1	2.6	—	2	4.6	—	9	9.6	94	14	17.0	82
Five or more years	904	117	100.3	117	27	7.6	355†	20	16.4	122	25	35.7	70	45	40.5	111
White	400	39	41.1	95	5	2.6	192	7	6.3	111	14	17.8	79	13	14.5	90
Nonwhite	504	78	59.2	132†	22	5.1	431†	13	10.1	129	11	18.0	61	32	26.0	123

† 1% level
‡ 5% level
— less than five deaths

had been only double the rate for other steelworkers, we would have failed to note a significant excess for coke oven workers, while a five-fold risk would have been insufficient to demonstrate a significant excess for coke plant workers. A similar diluting effect may result from inclusion in the study group of coke oven workers with too few years of observation to allow for the appearance of latent effects. The extent to which mortality estimates may vary in relationship to the length of the observation period was not determined in the present study since the study population was observed for only nine years. It should be noted, however, that the present estimates of lung cancer mortality may be conservative since the average latent period for the occupational lung cancers ranges from 15 to 25 years.[10] If disease response is related to degree of exposure, inclusion of short-term employees also would result in an underestimate of disease rate. We next consider, therefore, the relationship between cause-specific mortality and length of employment at the coke ovens.

Observed and expected deaths by length of employment as of January 1, 1953, are shown in Table VI. Here it is seen that men employed less than five years at the coke ovens showed little deviation from expected mortality. Additionally, there was no indication in this group of excess mortality from malignant neoplasm of the lung or of other sites. A further breakdown of those employed less than five years by work area within the coke oven (figures not shown)also showed no differences of consequence in cause-specific mortality. Among the 209 men employed at partial or full-time topside jobs, 14 deaths were noted, compared to 13.3 deaths expected, and no lung cancer deaths were observed in this group. It is thus seen that the unusual mortality experience of coke oven workers is a reflection of a higher risk for the 44 per cent of the population employed five or more years in that area. Men employed at the coke ovens for five or more years showed total mortality 17 per cent in excess of expectation and a lung cancer mortality 3 1/2 times that predicted (27 versus 7.6). The excess in total mortality was limited to the nonwhite workers with observed deaths 32 per cent greater than expectation. On the other hand, while the greater part of the lung cancer excess was accounted for by a four-fold risk for nonwhite workers (22 versus 5.1), a greater lung cancer risk also was seen for white workers employed five or more years at the coke ovens (5 versus 2.6).

Examination of mortality within the coke oven subdivisions for men employed five or more years further clarifies the differences already noted. As seen in Table VII, total mortality for men employed only at the side of the oven or with less than five years employment at full-time topside jobs differed little from expectation, whereas the number of deaths for men employed five or more years at full-time topside jobs was double the expected number (35 versus 17.4). Almost all of the latter difference was accounted for by a 10-fold risk of lung cancer for full-time topside workers (15 versus 1.5). A greater than expected lung cancer risk, of considerably less magnitude, is also noted for the other groups with five or more years employment at the coke ovens. Among men with less than five years of full-time topside employment, six lung cancer deaths were observed, compared to 2.1 deaths expected. The relative contribution of topside and non-topside exposure to this difference cannot be determined, but the much greater risk indicated for the former group suggests that a large part of the difference is associated with employment at the full-time topside occupations. However, the possibility that part of this difference may be associated with long-term employment at the side of the ovens also should be considered. Although the differences are not statistically significant, it should be noted that the slight excess of lung cancer observed for men employed only at the side of the ovens is limited to men with five or more years employment.

The figures in Table VII also indicate that excess mortality from nonpulmonary neoplasms may be associated with topside employment. A significant difference between observed and expected deaths is seen for men employed five or more years at the coke oven who had less than five years of full-time topside employment (9 versus 4.3). This difference cannot be accounted for solely by employment at the full-time occupations since six of the deaths from nonpulmonary malignancies were seen in men with five or more years partial topside employment who had never been employed at the full-time occupations, whereas 2.7 deaths would have been expected. Review of observed and expected deaths for more detailed sites, as seen in Table VIII, shows that this difference is not limited to a single anatomical site. No difference in mortality from nonpulmonary malignancies was noted for men employed five or more years only at the side of the ovens.

Nonwhite workers employed five or more years at full-time topside jobs also show a significant excess of deaths from "all other causes" (11 versus 6.1). Most of this difference is accounted for by deaths from vascular lesions of the central nervous system (4 versus 1.2), and tuberculosis (2 versus 0.6).

Although the differences are not significant, one further point of interest concerning coke oven worker mortality is worth noting because of similar observations for gas retort workers[6] and workers in coal gas and coke.[11] Mortality from heart disease for coke oven workers is only 80 per cent of expectation, the deficit being more marked for men with five or more years experience, and is apparent for both white and nonwhite workers. No deficit in heart disease is seen for the non-oven workers. It does not appear that this difference is due solely to competing causes of death since the heart disease deficit for coke oven workers is least apparent for the group with the highest lung cancer risk. We have previously noted that differences in heart disease mortality within the steelworker population may be related to selection for health.[1]

Discussion

It has long been recognized that some agent produced during the combustion or distillation of bituminous coal

TABLE VII

NUMBER EMPLOYED, OBSERVED AND EXPECTED DEATHS AND STANDARDIZED MORTALITY RATIOS FOR SELECTED CAUSES, 1953-1961; MEN EMPLOYED FIVE OR MORE YEARS AT COKE OVENS AND SUBDIVISIONS AS OF JANUARY 1, 1953

Work Area and Race	Number Employed	All Causes			Malignant Neoplasms of Lung			Other Malignant Neoplasms			Heart Diseases			All Other Causes		
		Observed Deaths	Expected Deaths	SMR	Observed Deaths	Expected Deaths	SMR	Observed Deaths	Expected Deaths	SMR	Observed Deaths	Expected Deaths	SMR	Observed Deaths	Expected Deaths	SMR
Side oven only	496	53	55.1	96	6	4.1	146	7	8.9	79	15	19.4	77	25	22.7	110
White	171	16	18.7	86	2	1.1	–	2	2.9	–	6	8.1	74	6	6.6	91
Nonwhite	325	37	36.4	102	4	3.0	–	5	6.0	83	9	11.3	80	19	16.1	118
Side and topside (less than 5 years full topside)	276	29	27.9	104	6	2.1	286†	9	4.3	209‡	5	10.8	46	9	10.7	84
White	202	19	19.4	98	2	1.3	–	5	2.9	172	5	8.4	60	7	6.9	101
Nonwhite	74	10	8.4	119	4	0.8	–	4	1.4	–	0	2.4	–	2	3.8	–
Full-time topside	132	35	17.4	201†	15	1.5	1000†	4	3.2	–	5	5.6	89	11	7.1	155
White	27	4	3.0	–	1	0.2	–	0	0.5	–	3	1.3	–	0	1.0	–
Nonwhite	105	31	14.4	215†	14	1.3	1077†	4	2.8	–	2	4.3	–	11	6.1	180‡

† 1% level
‡ 5% level
– less than five deaths

TABLE VIII

OBSERVED AND EXPECTED DEATHS AND STANDARDIZED MORTALITY RATIOS FOR MALIGNANT NEOPLASMS BY SITE, 1953-1961; MEN EMPLOYED FIVE OR MORE YEARS IN THE COKE PLANT AND SUBDIVISIONS AS OF JANUARY 1, 1953

Cause of Death	Five Years Coke Plant			Five Years Coke Plant Never Oven			Five Years Coke Oven			Five Years Side Oven Never Topside			Five Years Coke Oven, Less than Five Years Full Topside			Five Years Full-Time Topside		
	Obs.	Exp.	SMR	Obs.	Exp.	SMR	Obs.	Exp.	SMR	Obs.	Exp.	SMR	Obs.	Exp.	SMR	Obs.	Exp.	SMR
Malignant neoplasms, all sites	68	44.8	152†	19	18.4	103	47	24.0	196†	13	12.9	101	15	6.4	234†	19	4.7	404+
Bronchus, lung and trachea	29	13.6	213†	1	5.3	19	27	7.6	355†	6	4.1	146	6	2.1	286†	15	1.5	1000†
Other respiratory system	2	1.0	-	1	0.3	–	1	0.7	–	1	0.4	–	0	0.1	–	0	0.2	–
Nonrespiratory system	37	30.2	123	17	12.8	133	19	15.7	121	6	8.5	71	9	4.2	214‡	4	3.1	–
Buccal and pharynx	1	1.2	–	0	0.6	–	1	0.5	–	0	0.2	–	1	0.2	–	0	0.1	–
Stomach	4	4.9	–	3	2.1	–	1	2.5	–	0	1.4	–	1	0.7	–	0	0.5	–
Intestines, excluding rectum	9	4.0	225‡	6	1.9	316†	3	1.8	–	2	1.0	–	1	0.5	–	0	0.3	–
Rectum	1	1.6	–	0	0.8	–	1	0.8	–	1	0.4	–	0	0.3	–	0	0.1	–
Pancreas	3	2.2	–	2	0.9	–	1	1.3	–	0	0.7	–	1	0.4	–	0	0.2	–
Other digestive organs	2	2.0	–	1	1.0	–	1	0.8	–	0	0.4	–	1	0.3	–	0	0.1	–
Prostate	6	3.3	182	1	1.1	–	4	2.1	–	3	1.2	–	0	0.4	–	1	0.5	–
Other genital organs	0	0.2	–	0	0.1	–	0	0.1	–	0	0.0	–	0	0.0	–	0	0.0	–
Kidney and other urinary organs	3	1.9	–	1	0.8	–	2	1.1	–	0	0.5	–	2	0.2	–	0	0.4	–
Brain and central nervous system	1	1.1	–	0	0.4	–	1	0.7	–	0	0.4	–	0	0.2	–	1	0.1	–
Other and unspecified sites	3	4.8	–	1	2.0	–	2	2.6	–	0	1.5	–	0	0.7	–	2	0.4	–
Leukemia	1	1.5	–	1	0.6	–	0	0.9	–	0	0.4	–	0	0.3	–	0	0.2	–
Lymphoma	3	1.5	–	1	0.6	–	2	0.8	–	0	0.4	–	2	0.2	–	0	0.2	–

† 1% level
‡ 5% level
– less than five deaths

is carcinogenic for the skin of man, and since the turn of the century it has been demonstrated that a variety of industrial populations exposed to coal tar products have a special liability for cancer of the skin.[12] More recent studies of men employed in some of these areas indicate that exposure to coal tar products also may result in increased liability for cancer of other organ systems.

The history of the "coal tar" cancers begins with the observation on scrotal cancers in London chimney sweeps by Percivall Pott in 1775.[13] He noted that this disease was peculiar to persons employed as chimney sweeps and originated from what was described in the trade as a soot-wart. Pott attributed these cancers to the lodgment of soot in the folds of the scrotum. Little was added to these observations until 1892, when Butlin undertook an extensive review of the question.[14] At that time the incidence of "chimney sweeps" cancer was thought to be declining due to the introduction of mechanical sweeping equipment, but a review of official statistics showed an eight-fold excess of cancer deaths among men employed at that trade. Butlin concluded that scrotal cancer remained a serious problem for British sweeps but was an extremely rare disease elsewhere. He attributed this difference to the British practice of burning bituminous coal (rather than wood or nonbituminous coal) in open grates, and the poor personal hygiene of the British chimney sweeps.

Volkmann, in 1873, was the first to observe that "chimney sweeps cancer" was not limited to persons of that trade.[15] He reported three cases of scrotal cancer in men handling tar and paraffin recovered through the carbonization of lignite, a "young coal." Volkmann noted that this extremely rare disease "agreed to the last detail with the so-called chimney sweep cancer of the British." The first cases of scrotal cancer associated with exposure to by-products of bituminous coal carbonization were reported three years later by Manouvriez.[16] He had observed seven cases of scrotal cancer and three cases of facial epithelioma in French briquette workers exposed to coke oven tar and pitch. Other reports of unusual skin cancer experience among coal carbonizers and handlers of various by-products soon appeared. Ball noted a high skin cancer rate in Irish tar workers[17] and Butlin, in his survey of "chimney sweeps cancers," reported skin cancers in gas workers and in men handling anthracene and pitch.[14] Although scrotal cancers in men not employed as chimney sweeps had been observed in the United States as early as 1837, we have no record of specific occupations.[18] The first American cases associated with exposure to coal tar products were carbon workers reported by Leuke in 1907.[19] In the same year, the British included "scrotal epithelioma occurring in chimney sweeps and epitheliomatous cancer or ulceration of the skin occurring in the handling or use of pitch, tar, and tarry compounds," in the Workman's Compensation Schedule, and these diseases later became notifiable under the Factories Act.[12]

Experimental studies on cancer induction further demonstrated the carcinogenicity of the coal tars and eventually led to the isolation of the first pure chemical carcinogen. In 1915, Yamagiwa and Ichikawa showed that coal tar was carcinogenic for the skin of the rabbit[20] and in 1922, almost 150 years after Pott's observation, Passey induced cancer with an ether extract of chimney soot.[21] Following many years of research on the constituents of the coal tar distillates, 3,4-benzpyrene, a potent carcinogen, was isolated.[22]

The extent of the skin cancer problem among coal tar workers and the variation in incidence within occupations are reported in a comprehensive review by Henry.[12] Unfortunately, comparisons of rates must be limited to the fatal cases of scrotal cancer, since notified cases of skin cancer relate to current occupation. In contrast with a general population rate of 4.2 per million, Henry reported the following minimal estimates for persons exposed to coal tar products:

Chimney sweeps	754.7
Patent fuel workers	504.2
Tar distillery workers (excluding those at gas works)	212.9
Skilled makers of coal gas and coke	123.9
All coke oven workers	21.1
All gas workers	19.9
Producer gas men	10.9
Dyers and dyers laborers	7.9

While each of these groups shows higher than expected mortality from scrotal cancer, there is considerable variation in response by level of coal tar processing. Thus, the rate for tar distillery workers is 10 times that observed for men employed at coal carbonization. On the other hand, scrotal cancer mortality for patent fuel workers is more than double the rate for tar distillery workers. These men are exposed to pitch, the residue following distillation of the coal tar. These differences suggest the possibility that the carcinogenic agent may become more concentrated with successive distillation. Two other points of interest in these data are the differences between the two classes of gas workers and coke oven workers and the indicated excess for dyers. The mortality rate shown for skilled makers of coal gas and coke is considerably higher than for other workers in these areas. This group would presumably include the men engaged in the operation of the ovens or the retorts and thus would include the topside workers. While the excess mortality from scrotal cancer for dyers is not as striking (Henry shows a range on the estimated mortality of 7.9 to 9.2), it is of interest because of the recognized bladder cancer hazard for persons employed in the production of coal tar dyes.[23]

Among the 4,716 steelworker deaths reported in this series, eight skin cancer deaths were noted. However, none of these men had ever been employed in any area of the coke plant. Henry, on the other hand, noted 84 cases of epitheliomatous ulceration or cancer of the skin (40 scrotal) for British coke oven workers for the period 1920 to 1943, and 11 fatal scrotal cancers among men with prior coke oven employment. Because of the

relatively low rate for this disease and the high survival rate for skin cancer, it is impossible to determine whether the absence of skin cancer deaths in our coke plant workers is a reflection of differential response, sampling variation or other factors such as differences in medical care. Skin cancers have been observed in American coke oven workers, but the literature is too sparse for estimating the level of risk. Heller has reported a much higher skin cancer rate for tar distillery workers handling gas works tar than for those handling coke oven tar.[24] The limited data available for comparison does suggest a differential scrotal cancer response for British and American workers. The British mortality rates per million for males 25 and over in 1921 and 1931 were 6.5 and 6.3, respectively,[25] whereas the rate for the U.S. Death Registration States in 1926 was only 1.2.[26] It has also been noted that 7 to 8 per cent of fatal skin cancers in England originate on the scrotum as compared to 1.2 per cent for the United States.[27]

Butlin had suggested in 1892 that the coal tars might be responsible for cancer of internal organs,[14] and many investigators during the early part of the 20th century speculated that the increasing rate of lung cancer might be attributed to the increased use of tar and tar products.[28,29] However, prior to 1936, the evidence linking lung cancer to coal tar exposures was limited to single case reports and the observation by Kennaway that a high proportion of noncutaneous cancers in chimney sweeps were situated in the respiratory tract and the alimentary canal above the stomach.[30] As Brockbank pointed out in 1932, no adequate study of occupational lung cancer had been made, and occupations cited in case reports were generally too vague for meaningful analysis. He noted four primary lung cancers in gas workers among 898 cases cited in the literature to 1932.[31]

The first report of unusual lung cancer experience for men engaged in coal carbonization concerned Japanese producer gas workers.[32] Producer gas is a fuel generated to meet power needs of the plant in which it is produced. Included under this term are a great variety of fuels which differ according to the carbonizing process, the product used (coal, coke, etc.) and the additives employed (water, oil, etc.). The operation described by the Japanese involved the destructive distillation of bituminous coal to generate fuel for open hearth furnaces.[33] Of malignant neoplasms observed in men working at these gas generators, 80 per cent were lung cancers (12 out of 15 cases).[32] In 1936, lung cancer was a relatively rare disease in Japan and accounted for only 3.1 per cent of all malignant neoplasms.[34] The extremely high lung cancer rate for gas generator workers was even more striking in contrast with the experience of other employees at the same steel plant. Not a single lung cancer was noted among the 46 malignant neoplasms observed for other employees. Although a direct estimate of the relative lung cancer risk cannot be made (the population by work area is not given), the proportionate number of lung cancer to total cancer deaths suggests a 26-fold excess over general population mortality and a considerably greater excess in comparison with other Japanese steelworkers. A recent report on men transferred from this facility, when it was closed in 1953, shows that lung cancer for the period 1954 to 1960 was 33 times the rate observed for other steelworkers.[7]

In the same year that the Japanese reported on lung cancer in gas generator workers, Kennaway and Kennaway noted an excess for British gas producermen.[35] Furthermore, their survey of death certificates for England and Wales, 1921 to 1932, showed that other coal carbonization and by-product workers might have experienced higher than expected lung cancer mortality. In this and a subsequent report for 1921 to 1938, the Kennaways reported excess lung cancer mortality for gas producermen, chimney sweeps and several categories of gas works employees.[36] The excess indicated for "gas stokers and coke-oven chargers" was approximately three-fold. More recent reports for England and Wales also show higher than expected lung cancer mortality for men employed in the production of coal gas and coke but the differences are not as striking, the indicated excess being only 32 per cent.[11] Estimates of disease risk based on death certificate ascertainment of occupation, although informative, are subject to substantial biases and can only be taken as suggestive of occupational differences.[3]

Population-based studies of three classes of coal carbonization workers have confirmed the Kennaway findings. The experience of the Japanese generator workers has already been described. Doll, in a study of gas works pensioners (gas retort workers) in 1952, observed an 81 per cent excess of lung cancer deaths in comparison with the general population.[37] More recently, he has confirmed a high risk for British gas workers and has noted differential rates by type of retort and by level of exposure.[6]

Although it had been suggested prior to the present report that coke oven workers might be more liable to lung cancer, the evidence appeared to be contradictory. No lung cancer deaths were reported for Japanese steelworkers not employed at the gas generators, although this group included coke oven workers[32]; the British report of a three-fold excess for "gas stokers and coke-oven chargers" was in marked contrast to a deficit of lung cancer for "tar distillery workers and coke-oven workers" (52 per cent of expectation).[36] Reid and Buck compared the mortality experience of British coke plant workers with that predicted by a "large industrial organization."[38] They observed 14 lung cancer deaths for nonretirees with prior coke oven experience compared to 10 deaths expected. Proportionate mortality comparisons for retirees (population data were not available) did not suggest any difference for lung cancer. These authors concluded that "the mortality in the coking industry from cancer in general and cancer of the lung in particular is not as excessive as had been feared, and may in fact be negligible." Christian, on the other hand, reported a 24-fold excess of lung cancer for men working in coke (not otherwise specified) at a large public utility.[39] The apparent discrepancies between these findings may be explained in part by differences in the

definition of the study populations and the comparison groups, and by the fact that the extremely high risk is limited to a small proportion of the coke oven population. In the British coke plant study, the number of lung cancer deaths observed may have been deficient, since only men dying while "on the books" of the coking plants during the period 1949 to 1954 were included. The authors indicate that men are removed from the books after prolonged absence from work. It should also be noted that the population at risk and the distribution by age and area of prior employment was based on estimates which excluded retirees and those who had left employment. On the other hand, the excessively high relative risk reported by Christian is not adjusted for age and sex, and the average population for the years 1946 to 1961 is taken as the population at risk. The extremely high lung cancer risks reported for other occupational groups which might be expected to have normal risk (for example, 8 observed versus 2.7 expected for field representatives) suggests that the relative risk for "coke" workers was overestimated.

Several authors have suggested that workers handling by-products may be more liable to lung cancer, but the evidence is scanty and contradictory. Kennaway and Kennaway reported a 64 per cent excess of lung cancer for "paviours, street masons, concreters and asphalters" and noted three lung cancer deaths in patent fuel laborers compared to 0.5 expected.[36] At the same time, they reported a deficit of lung cancer for "tar distillery workers and coke-oven workers." Hueper refers to lung cancers observed in a group of tar distillery workers but given no indication of the level of risk.[40] A recent study by Selikoff and Hammond showed no unusual lung cancer experience for roofers working with pitch[41] and the lung cancer mortality of nonoven workers included in the present study was somewhat lower than expected.

Although evidence of a bladder cancer excess for coal tar workers has appeared only in recent years, it has long been recognized that certain synthetic dyes produced from coal tars contain a potent bladder carcinogen. The first cases were reported to the German Surgical Society in 1895 by Rehn.[42] Following many years of research, during which a great number of cases were identified among synthetic dye workers, it was demonstrated that the specific agent responsible for the bladder cancer was β-naphthylamine.[23,43] Since the manufacture of synthetic dyes was far removed from the carbonization and distillation process (β-naphthylamine exposure occurs during the manufacture of intermediate dye compounds using benzene, toluene, xylene, naphthalene and anthracene which are recovered from coal tar), the possibility of a bladder cancer problem among men engaged in the initial processes apparently was ignored. The first evidence of a bladder cancer excess for "coal tar" workers was presented by Henry et al. in 1931.[44] In their review of bladder cancer deaths for 1921 to 1928, they reported a greater than expected mortality for nine occupational groups exposed to coal gas, tar and pitch. Furthermore, of the 15 occupational groups exhibiting an excess of 50 per cent or more, five were among the "coal tar" occupations. Their finding of a higher rate for gas retort workers has since been verified by Doll et al.[6] Bruusgaard, who noted a high frequency of cancers in Swedish gas workers (41 out of 125 autopsies), also reported five cancers of the bladder.[45] An indication that these tumors may result from the same source as those of the dye workers comes from Battye's observation of β-naphthylamine in gas retort effluents.[46] This author also reported five bladder cancers in a population of 467 retort house workers compared to no response among 546 men employed outside of the retort houses.

It is difficult to assess the extent of the bladder cancer problem among "coal tar" workers because of the lack of population data. The findings of Henry et al.,[44] which are admittedly limited, suggest a greater level of risk for by-product workers (28 versus 15.8) than for men engaged in the carbonizing process (45 bladder cancer deaths were observed in gas stokers and coke oven chargers compared to 33.7 expected). Unfortunately, this group cannot be separated to obtain an estimate for the coke oven chargers (larrymen). For all coke oven workers they report six bladder cancer deaths compared to 4.9 expected, an insufficient difference to conclude that coke oven workers have an excess. No bladder cancer deaths were observed among the coke plant workers in our series. The three deaths from cancer of the urinary system shown in Table VIII were of the kidney, and no excess mortality is indicated, the expected number being 2.7. The bladder cancer findings for British coke oven workers is also negative.[38] We would have to conclude, then, that there is currently no evidence of unusual bladder cancer experience among coke oven workers. However, recognizing that this is a comparatively rare disease with a long latent period and that β-naphthylamine has been isolated from coking pitch,[47] further follow-up of the coke oven workers is advisable.

Evidence of an excess risk for cancer of several other sites also has been reported for certain populations exposed to coal tars. This comes primarily from review of occupations noted on death certificates and isolated reports of rare tumors. It should be pointed out that these findings have not been confirmed by other investigators and are subject to certain biases.[3] They are presented to complete the picture of cancer in "coal tar" workers and to allow for contrast with the pattern of nonpulmonary cancers shown in Table VIII. Kennaway and Kennaway reported a two-fold excess of cancer of the larynx for gas stokers and coke oven chargers using death certificates for England and Wales, 1921 to 1938.[35] Interestingly, five coal tar occupations (including other coke oven workers) showed a deficit in mortality from this cause. Jenkins reported a single case of nasal sinus cancer in a gas works laborer,[48] and Bruusgaard reported two cancers of this site in his series.[45] The Registrar General's Report from death certificates for 1949 to 1953 shows a 44 per cent excess of stomach cancer among makers of coal gas and coke.[11] We have noted that coke plant workers not employed at the coke ovens may have experienced some excess from digestive cancer, but the difference was in large part due to cancer of the intestine.

The Registrar General's Report also shows a significant excess of pancreatic cancer in these workers with 21 deaths observed compared to 12 expected. This observation is of particular interest because of the report by Mancuso and El-Attar of an excess of pancreatic cancer for men exposed to β-naphthylamine and benzidene.[49] We have noted three deaths from cancer of the pancreas in nonoven workers compared to 1.3 expected. An excess of leukemia among skilled workers in coal gas and coke also is reported by the Registrar General. This observation is of interest because exposure to benzene, a suspected leukemogen,[50] could occur in the by-products area.[5] Our data do not suggest any excess for this disease.

Our understanding of cancer in man, its etiology and its eventual prevention depends upon the identification of common factors which differentiate groups within the population according to degree and specificity of response. A number of common factors related to disease response become evident when we consider the cancer experience of men employed in the production and processing of coal tar products. It has been observed, for example, that all of the "coal tar" occupations are at increased risk of developing cancer at one or more sites. Such a broad response over so many diverse groups suggests a common causative factor in each of the working environments which is not present at the same level in the general population environment. Furthermore, the appearance of a greater risk for more than one site in several of these occupations suggests that a common agent may be responsible for cancer at more than one anatomical site. Although such a hypothesis is in sharp contrast to the frequently observed site specificity of occupational cancers,[51] it is consistent with experimental findings[52] and the observations on cigarette smoking and cancer.[53]

On the other hand, the possibility of exposure to multiple carcinogens must be considered since a number of carcinogenic agents have been identified in these environments.[54,55] With the exception of the bladder tumors in gas retort workers, which may be attributed to exposure to β-naphthylamine, no single carcinogen present in coal tar has been uniquely associated with the high cancer risk of "coal tar" workers. We have, however, from experimental findings and from the observations on these populations, a number of clues concerning the characteristics and the distribution of the causative factor or factors within the working population. We have noted, for example, that (1) it is available in greater concentration in certain well-defined areas of the working environments; (2) it is probably present in different physical states within the several working environments making access to different organ systems variable; and (3) in the case of skin cancer, the response increases with successive distillations and varies with the source of the coal tar.

A most important relationship which should provide leads for identifying site-specific carcinogens is the relationship between cancer response and the temperature of carbonization or distillation. As early as 1925 Kennaway had observed that coal tars produced at higher temperatures were far more carcinogenic than those produced at the lower temperatures.[56] He reported that the cancer producing agent was formed in very small amounts below 450 C, increased rapidly between 450 and 560 C, and continued to increase at a slower rate over the range of 560 to 1,250 C. As shown in Table IX, there also appears to be a positive relationship between temperatures attained during carbonization and the lung cancer response for men employed in the several areas. The figure shown for coke oven workers is for men with five or more years experience to provide contrast with the British gas workers who had worked at least five years at the retorts. Although the relative excess for Japanese gas generator workers appears to be well beyond that observed for other carbonization workers, available data are insufficient for an exact determination of the lung cancer excess. The percentage excess indicated during the period 1954 to 1960 for men reported as gas generator workers in 1953 was 3,200 per cent (6 deaths observed versus 0.18 expected). This may be an underestimate of the response among men initially included in this group, since a decrease in lung cancer rate was noted following changes in equipment.[33] On the other hand, while it is not stated explicitly, an early description of the work area for the lung cancer cases suggests that many of these men may have been working on top of the gas generator so that exposure to effluents would have been greater. On the basis of the experience for topside coke oven workers, a more conservative estimate of the excess for all gas generator workers would be one-fourth of that reported. We therefore show as a crude estimate 800 per cent. We have already commented on several possible sources of discrepancy between the findings for British coke oven workers[18] and findings from the present study. With regard to the figures shown in Table IX, two other factors should be mentioned which might account for a smaller reported lung cancer excess for British workers. First, the study population may have included many men with limited service. In the present study the amount of excess for all coke oven workers was 152 per cent (Table V) compared to 225 per cent for men employed five or more years (Table VI). Second, American coke ovens are generally operated at higher temperatures than the British coke ovens.[57]

TABLE IX

TEMPERATURE RANGE OF CARBONIZING CHAMBERS AND EXCESS OF LUNG CANCER REPORTED

Carbonizing Chamber	Temperature Range §	Per Cent Excess of Lung Cancer Reported
Vertical retorts	400 - 500 C	27%[6]
Horizontal retorts	900 - 1100 C	83%[6]
Coke ovens	1200 - 1400 C	255% (Table VI)
Japanese gas generators	≥ 1500 C	800% (see text)

§ References for temperature range: [5,33,52,57-59]

The observation of a relationship, albeit crude, between lung cancer response and temperature of carbonization provides another lead in the search for causative factors. It is apparent that more detailed study of these populations, with particular emphasis on environmental measurements, should contribute greatly to our understanding of the human cancer problem.

Summary

In this, the fifth of a series of papers concerning the mortality of steelworkers, we examine the variation in mortality of coke plant workers according to calendar period and length of employment at several work stations within the coke plant. Differences in the composition of the study population and the distribution of deaths by cause for men employed in 1953 and in prior years are considered and differences in patterns of mortality are noted. The cause-specific mortality experience of men employed in each of the coke plant areas is contrasted with the mortality predicted by the age-, race- and nativity-specific rates for the total steelworker population. Site-specific patterns of cancer mortality also are contrasted with those for other industrial groups engaged in the carbonization of bituminous coal or in handling of coal tar by-products.

The findings of particular interest are:

1. The excess of respiratory cancer previously reported for coke plant workers is limited to men employed at the coke ovens, the relative mortality for this disease being 2 1/2 times that predicted.

2. The greatest part of this excess is accounted for by an almost five-fold risk of lung cancer in men working on the tops of the coke ovens.

3. A 10-fold risk of lung cancer is observed for men employed five or more years at full-time topside jobs. Fifteen lung cancer deaths were observed among the 132 men in this group compared to 1.5 deaths expected.

4. The apparent differential in respiratory cancer rates for white and nonwhite coke plant workers, reported in the fourth paper of this series, is accounted for by differing distributions by work area and the unusually high lung cancer risk for topside workers. Lung cancer mortality for white and nonwhite coke plant workers employed at work stations other than the top of the coke oven is comparable.

5. A deficit of deaths from heart disease, previously reported for similar occupational groups, is also seen for coke oven workers.

6. Coke plant workers employed only in the nonoven area may be at excess risk of digestive cancer.

A review of the literature on cancer mortality of men employed in the "coal tar" industries shows that all of these occupations evidence excess cancer of one or more sites. The lung cancer excess for coke oven workers also is observed for other groups engaged at coal carbonization, and it appears that the lung cancer response is positively correlated with the temperature of carbonization.

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Erratum

In the Health Education Newsline item entitled "Emphysema Explained to Employees" in the July, 1970, issue, three lines of type were dropped between pages 272 and 273. The missing three lines should read as follows:

> Ultimately, as these conditions continue, normal breathing, which should be automatic, easy, and unrestricted, is replaced by wheezing and chronic cough, at