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Research and Development

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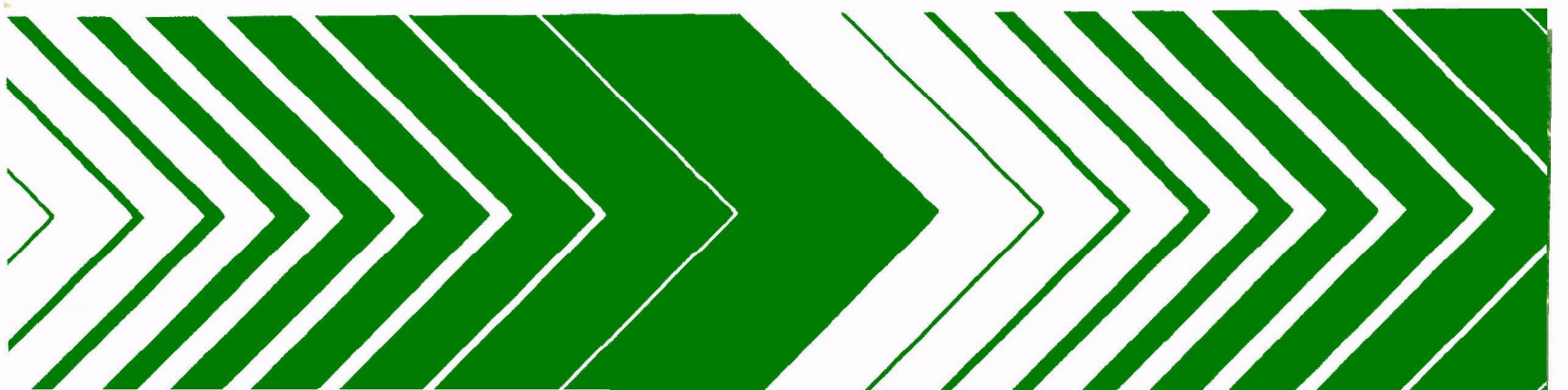
Preliminary Analysis of Cancer Rates in Primary Organic Chemical-Producing Counties



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PRELIMINARY ANALYSIS OF CANCER RATES IN ORGANIC CHEMICAL-PRODUCING COUNTIES

By
Amy J. Cross and G. Bruce Wiersma
Monitoring Systems Research and Development Division
Environmental Monitoring and Support Laboratory
P. O. Box 15027
Las Vegas, Nevada 89114

ENVIRONMENTAL MONITORING AND SUPPORT LABORATORY
OFFICE OF RESEARCH AND DEVELOPMENT
U.S. ENVIRONMENTAL PROTECTION AGENCY
LAS VEGAS, NEVADA 89114

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FOREWORD

Protection of the environment requires effective regulatory actions that are based on sound technical and scientific information. This information must include the quantitative description and linking of pollutant sources, transport mechanisms, interactions, and resulting effects on man and his environment. Because of the complexities involved, assessment of specific pollutants in the environment requires a total systems approach that transcends the media of air, water, and land. The Environmental Monitoring and Support Laboratory-Las Vegas contributes to the formation and enhancement of a sound monitoring data base for exposure assessment through programs designed to:

- develop and optimize systems and strategies for monitoring pollutants and their impact on the environment
- demonstrate new monitoring systems and technologies by applying them to fulfill special monitoring needs of the Agency's operating programs

This study is designed to determine whether there is an association between cancer mortality and the industrial production of environmental carcinogens. Mortality rates in counties containing organic chemical production facilities are compared to rates in control counties. The study aids in the development of statistical techniques for determining the contribution of environmental contaminants to the rise in cancer rates. Research of this type assists in the identification of compounds that need to be regulated. For further information, contact the Monitoring Systems Research and Development Division of the Laboratory.

George B. Morgan
Director
Environmental Monitoring and Support Laboratory
Las Vegas

SUMMARY

This study is designed to determine whether there is an association between cancer mortality and the production of environmental carcinogens. Mortality rates of counties containing organic chemical production facilities are compared to rates of control counties. Twelve different cancer sites in lung, stomach, etc., and eight organic carcinogens were considered. Although a rigorous statistical analysis was not conducted, for most cancer sites mortality rates were found to be higher in counties of organic carcinogen production than in control counties. The study aids in the development of statistical techniques for determining the contribution of environmental contaminants to the rise in cancer rates. Research of this type assists in the identification of compounds that need to be regulated.

INTRODUCTION

The literature indicates an increase in cancer mortalities in chemical-producing counties (CPCs). Mason's report (1975) shows that a high rate of multiple myeloma and liver cancer occurs in vinyl chloride workers. A study by Hoover and Fraumeni (1975) relates a significant risk in male lung cancer with CPCs. Hoover et al. (1975) correlates cancers of the large intestine, rectum, esophagus, and bladder as a complex of urbanization.

An investigation (Kwalick et al., 1976) of New Jersey cancer mortalities cites this state as the most cancer-prone. The author suggests population density, industrialization, and a high concentration of organic chemical producers in the state as possible etiologic factors.

Figure 1 shows the location of primary organic chemical producers in the United States. When compared to Figure 2, which was taken from data in the cancer atlas (Mason et al., 1975), a relationship is suggested between organic chemical production and higher than average lung cancer mortality rates. Comparison of Figure 1 to other maps extracted from the cancer atlas for cancers of the large intestine, rectum, liver, and female breast illustrates similar relationships. Comparison of a population density map to the map for lung cancer (Figure 2) also shows a correlation.

Several problems complicate a statistical analysis of cancer incidence. Since no record of cancer incidence exists for any number of years, mortality data are used as an indication of incidence (Hoover and Fraumeni, 1975; Hoover et al., 1975; Mason, 1975). A certain amount of error is involved in the assumption that mortalities are indicative of incidence, most obvious of which is the fact that medical advances are lowering the mortality rates while the incidence rates rise (Levin et al., 1974). Mortality rates may not reflect the county of exposure since each neoplasm is recorded by county of death, not by county of residence and/or exposure. Differences in medical diagnosis of primary and secondary causes of death also inject error into the data.

Other problems involved in this type of analysis include variability in industrial processes, possible synergistic effects of chemicals, bias toward industries which dominate the employment of counties, and bias toward industries with variability in exposure. Limitations of a statistical study of site-specific cancer mortalities in relation to any other variable include non-industrial correlates: smoking habits, dietary differences, hormonal factors, cancer induction-related diseases, urbanization, meteorology, the latency period of cancers, sex, and age (Hoover and Fraumeni, 1975; Hoover et al., 1975; Mason, 1975; and Levin et al., 1974).

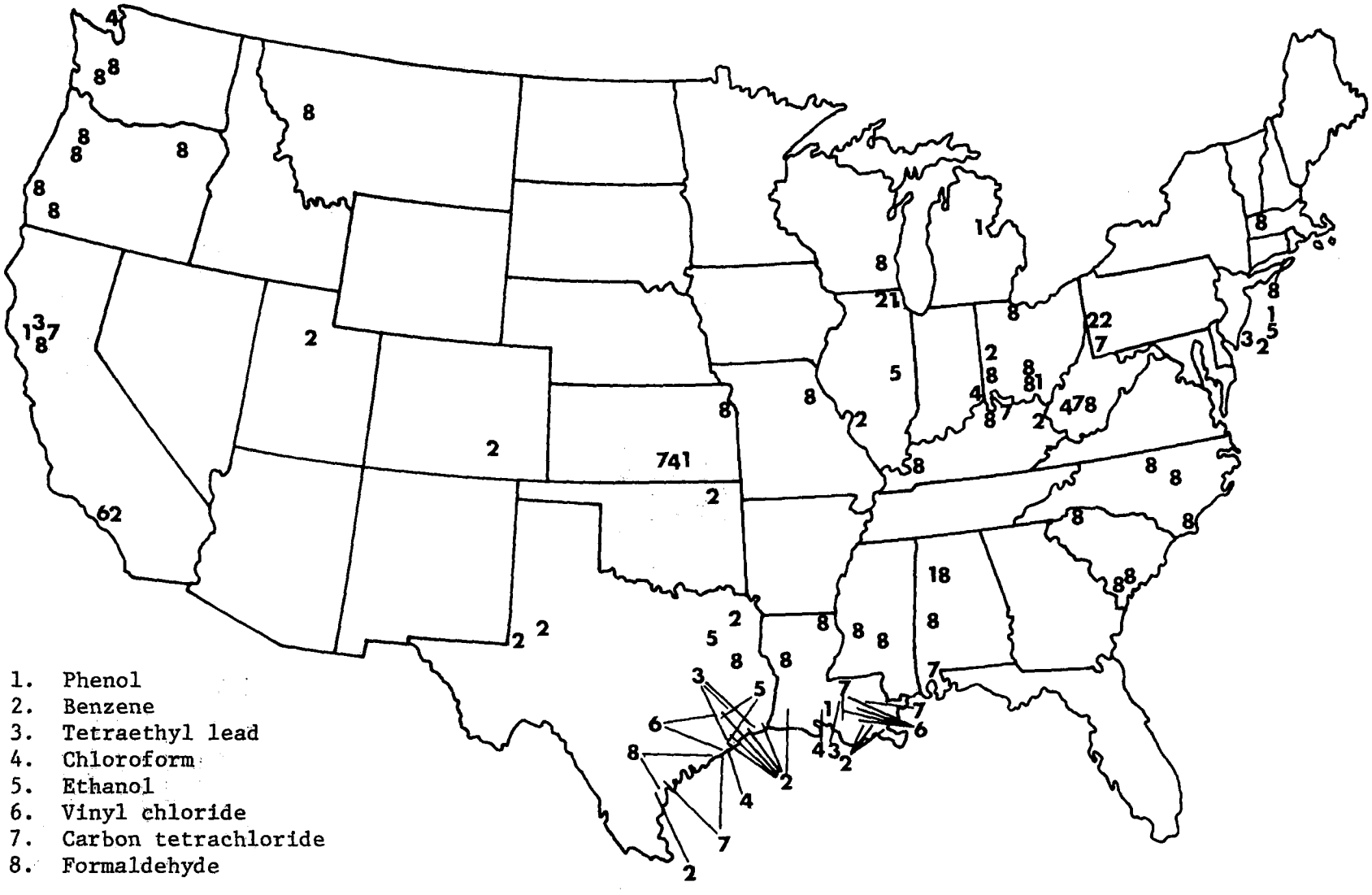


Figure 1. Locations of Chemical Producing Counties

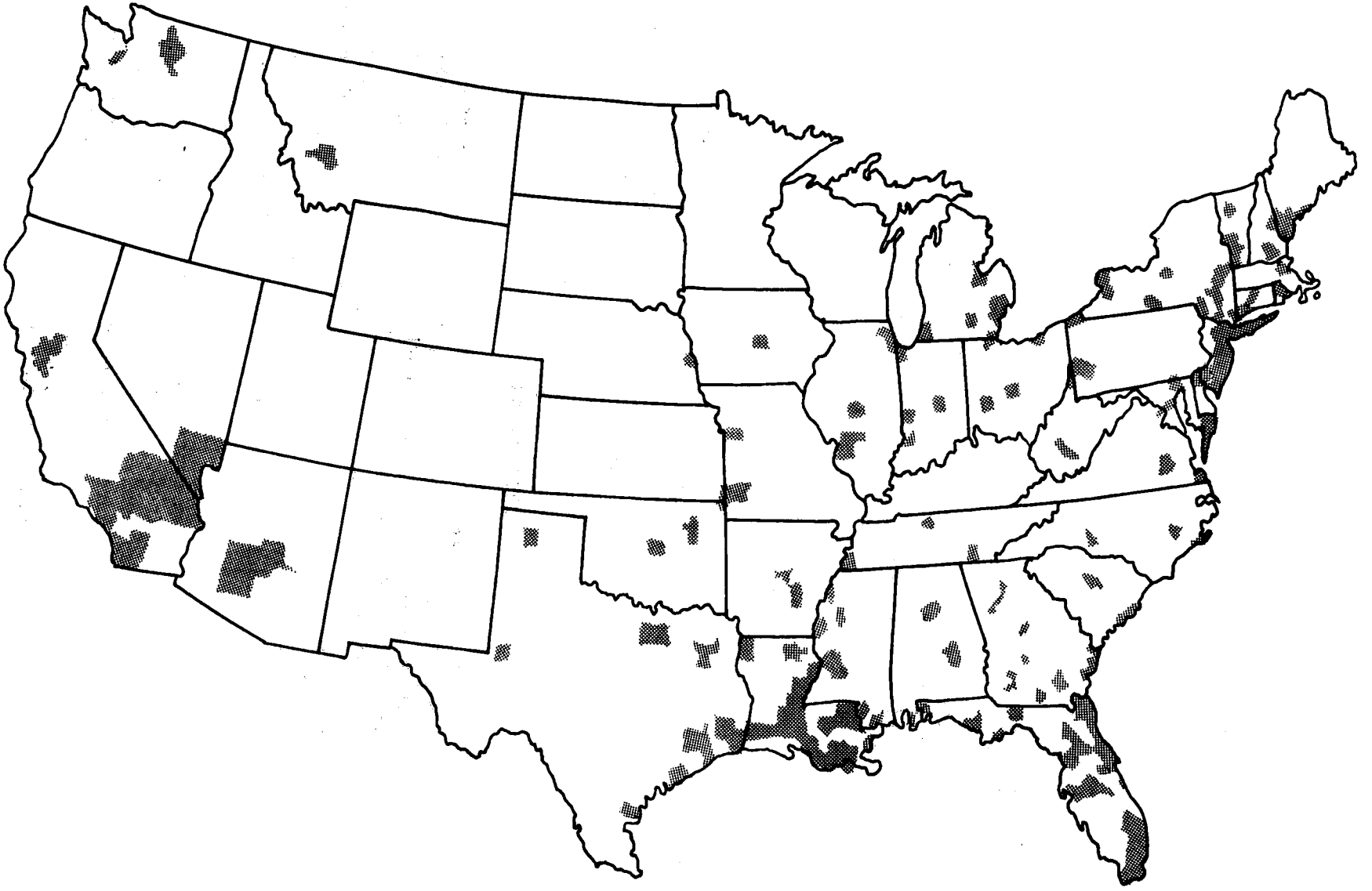


Figure 2. Counties of significantly high trachea, bronchus, and lung cancer mortalities 1950-1969.

In spite of the cautions involved in relating cancer incidence with cancer mortality data, such relationship still provides the most accessible and systematic means of studying the hazards associated with potential carcinogens in the environment. Basic statistics are used descriptively in this study, not in hypothesis testing.

CONCLUSIONS

Comparisons of the mean cancer mortality rates of chemical-producing counties, non-chemical-producing counties (non-CPCs), and the nation support reports of increased cancer rates in CPCs. In 284 comparions, 205 CPC mean cancer rates were higher than the non-CPC rates. In the same number of comparisons to U.S. mean cancer mortalities, 113 CPC rates were higher (Table 1).

Specifically, the study supports statistical investigations in which rates of cancers of the lung, large intestine, rectum, esophagus, breast, stomach, corpus uteri, cervix uteri, leukemia-aleukemia, total cancer, and multiple myeloma (vinyl chloride CPCs observed only) were found to be higher in CPCs than in non-CPCs.

The CPCs with most excessive and numerous mean cancer rate differences from the non-CPCs and the nation were those producers of phenol, tetraethyl lead, ethanol, benzene, chloroform, formaldehyde, and carbon tetrachloride.

Population density was shown to play a significant role in the results of this study. The CPCs were found to have a significantly higher population density than the non-CPCs. This also supports a report in which urbanization is correlated to a complex of cancers of the bladder, large intestine, rectum, and esophagus.

RECOMMENDATIONS

Both the literature and this study indicate that cancer mortality rates for the CPCs of phenol, benzene, vinyl chloride, chloroform, tetraethyl lead, ethanol, possibly formaldehyde, and possibly carbon tetrachloride should be included in the data of a more conclusive statistical analysis. However, the widespread use of ethanol in a non-industrial manner may impose the requirement for different or additional statistical treatment. Studies suggest also that the cancer sites to be studied in relation to these CPCs be the large intestine, rectum, liver, lung, female breast, esophagus, stomach, leukemia-aleukemia, corpus uteri, cervix uteri, bladder, total cancer, and possibly multiple myeloma.

This analysis should include a control of non-industrial correlates of carcinogenesis (System Sciences, Inc., 1975). The data could then be analyzed by factor analysis and three-way analysis of variance. Factor analysis would serve to eliminate the high correlation between variables that cause the regression computations to numerically break down. Instead of the paired T-test, three-way analysis of variance would be used to separate the causes of significance because it can handle more than two groups. A test for

TABLE 1. MEAN CANCER RATES OF U.S., NON-CPCs, AND CPCs

				Chloroform		Benzene		Ethanol		Tetraethyl Lead		Phenol	
			NATION	NON-CPC	CPC	NON-CPC	CPC	NON-CPC	CPC	NON-CPC	CPC	NON-CPC	CPC
Total Cancer	♂	W	174.04	160.433	*173.55**	158.326	*178.396**	157.583	*183.317**	171.300	*183.233**	165.818	*181.327**
		NW	184.28	106.98	*138.940**	163.889	*191.077**	141.160	*164.88	157.233	*194.217**	164.036	*192.500**
	♀	W	130.10	116.26	*126.38	113.993	*122.425	113.660	*131.580**	119.983	*127.733	121.155	*129.050
		NW	139.18	135.16	126.6	114.936	*136.007	120.250	*143.300**	129.467	*136.583	101.836	*130.682
Large Intestine	♂	W	16.54	13.1	*14.4	13.446	*14.711	12.767	*16.183	13.4	*14.35	14.218	*15.482
		NW	12.07	7.65	*16.050**	16.489	10.441	12.325	*12.45**	9.767	*11.050	7.027	*14.418**
	♀	W	16.25	15.06	13.52	12.026	*14.607	14.08	*16.32**	14.133	*15.133	14.464	*15.682
		NW	12.69	21.6	8.55	10.254	*12.934**	10.550	*13.125**	9.900	*11.083	10.927	*18.282**
Rectum	♂	W	7.65	4.467	*5.217	4.870	*6.322	5.700	*7.017	5.35	5.25	6.518	6.455
		NW	5.68	1.400	*4.125	7.036	5.721**	2.675	*6.125**	3.95	*4.167	5.673	4.045
	♀	W	4.82	3.600	3.420	4.464	4.159	2.680	*4.260	3.717	*3.800	4.109	4.018
		NW	4.46	.850	*3.875	2.793	*3.941	1.825	*4.925**	5.433	4.467**	2.064	*3.273
Esophagus	♂	W	4.10	2.620	*3.460	3.107	*3.557	3.260	*3.800	3.850	*4.317**	3.836	*4.109**
		NW	9.44	5.600	*9.050	7.961	7.811	6.000	*9.400	6.750	*7.550	6.427	*7.655
	♀	W	1.03	.520	*1.080**	.843	*.977	.816	*.820	2.017	1.183**	.755	*.964
		NW	2.17	.380	*2.850**	1.446	1.757	1.600	1.700	2.017	*2.500**	.964	*1.209
Lungs	♂	W	37.98	37.900	*42.650**	36.544	*45.715**	35.850	*43.400**	40.22	*46.32**	34.918	*42.905**
		NW	36.67	24.750	*33.500**	37.982	*45.802**	34.850	*43.250**	34.417	*41.67**	27.045	*49.709**
	♀	W	6.29	20.233	*23.250**	5.818	*6.629**	6.26	*7.660**	6.933	*7.383**	6.336	*6.650**
		NW	6.27	4.4	*5.7	7.000	*7.573**	5.600	*7.050**	8.117	7.233	4.527	*6.336**
Breast	♀	W	25.51	17.9	*21.4	18.500	*22.400	19.660	*23.380	20.233	*23.250	20.755	*23.614
		NW	22.10	18	14.94	15.779	*20.071	17.500	*24.550**	20.55	*21.233	14.427	*16.464
Stomach	♂	W	15.22	13.960	13.220	13.485	*14.038	10.820	*13.160	16.733	12.850	15.582	*15.741**
		NW	24.03	18.475	*30.825**	23.868	*25.513**	21.225	*25.800**	20.300	*25.883**	39.364	28.136
	♀	W	7.70	6.96	6.66	7.921	7.250	6.54	*6.68	8.300	6.567	7.300	*7.332
		NW	10.69	5.925	*8.475	9.361	*10.279	12.150	10.075	11.767	*11.800**	6.645	*9.500

(continued)

TABLE 1. MEAN CANCER RATES OF U.S., NON-CPCs, AND CPCs

				Chloroform		Benzene		Ethanol		Tetraethyl Lead		Phenol	
			NATION	NON-CPC	CPC	NON-CPC	CPC	NON-CPC	CPC	NON-CPC	CPC	NON-CPC	CPC
Liver	♂	W	5.160	6.083	5.033	5.282	5.086	5.867	5.167**	7.083	5.367**	5.291	5.159
		NW	6.910	5.280	*6.560	8.379	6.720	7.520	6.180	6.983	*7.283**	4.655	*6.636
	♀	W	5.340	6.730	5.500**	5.036	4.659	4.560	*4.920	4.800	4.400	5.400	4.686
		NW	4.590	0.760	*3.280	3.286	*4.793**	7.040	3.980	3.800	*5.483**	2.482	*5.045**
Prostate	♂	W	17.840	16.180	*17.033	16.454	*17.130	18.550	17.950**	15.933	*18.400**	17.036	15.159
		NW	27.390	17.033	*21.960	27.421	23.920	22.640	22.240	22.500	*28.850**	24.718	24.127

				Vinyl Chloride				Formaldehyde		Carbon Tetrachloride	
			NATION	NON-CPC	CPC			NATION	NON-CPC	CPC	NON-CPC	CPC
Multiple Myeloma	♂	W	1.760	1.344	*2.056**	Liver	♂ W	5.160	5.189	*5.423**	5.691	5.336**
		NW	2.700	0.938	*2.400		♂ NW	6.910	7.191	*7.978**	3.750	*7.740**
	♀	W	1.240	1.922	1.378**		♀ W	5.340	4.989	4.918	5.682	5.118
		NW	1.830	0.725	*1.663		♀ NW	4.590	4.266	*4.686**	4.470	3.490
						Prostate	♂ W	17.840	16.649	*17.893**	17.045	16.773
							♂ NW	27.390	23.161	22.586	20.340	*22.860

(continued)

TABLE 1. MEAN CANCER RATES OF U.S., NON-CPCs, AND CPCs

				Chloroform		Benzene		Ethanol		Tetraethyl Lead		Phenol	
			NATION	NON-CPC	CPC	NON-CPC	CPC	NON-CPC	CPC	NON-CPC	CPC	NON-CPC	CPC
Cervix Uteri	♀	MN	8.81	8.880	* 9.220 **	8.686	* 8.727	10.200	10.020 **	8.250	* 9.600 **	9.173	8.532
		M	5.77	3.250	* 6.700 **	3.900	* 5.188	9.050	6.625 **	4.317	* 5.400	4.727	* 4.900
Corpus Uteri	♀	MN	5.74	5.240	* 5.560	5.279	* 5.611	5.240	* 6.420 **	5.033	* 6.100 **	5.573	* 6.114 **
		M	3.88	2.750	2.025	2.650	* 4.591 **	4.600	4.100 **	6.100	4.317 **	3.682	* 3.973 **
Leukemia-Aleukemia	♂	MN	6.13	6.240	6.180 **	6.107	5.513	5.700	* 6.880 **	6.400	5.617	6.745	6.282 **
		M	11.3	5.075	* 7.975	10.886	10.470	10.925	10.225	8.150	* 11.530 **	6.127	* 11.064
	♀	MN	7.79	9.720	* 10.340 **	7.446	* 7.850 **	7.640	* 7.860 **	8.683	7.833 **	7.618	* 8.545 **
		M	18.92	12.125	* 13.250	21.032	17.173	11.650	* 17.375	14.517	* 14.850	13.018	* 13.418

(continued)

TABLE 1. MEAN CANCER RATES OF U.S., NON-CPCs, AND CPCs (cont'd)

			NATION	Formaldehyde NON-CPC	Formaldehyde CPC	Carbon Tetrachloride NON-CPC	Carbon Tetrachloride CPC
Total Cancer	♂	W	174.04	156.24	* 169.72	158.47	* 177.21**
		NW	184.28	160.39	* 174.46	139.50	* 173.54
	♀	W	130.10	119.56	* 123.35	113.99	* 122.44
		NW	139.18	133.28	129.47	117.72	* 141.98**
Large Intestine	♂	W	16.54	13.38	* 14.29	11.34	* 14.31
		NW	12.07	10.61	* 11.75	11.59	11.36
	♀	W	16.25	13.88	* 14.46	12.72	* 14.76
		NW	12.69	14.55	12.16	6.87	* 8.44
Rectum	♂	W	7.65	5.77	* 6.36	3.91	* 5.44
		NW	5.68	3.79	* 6.28**	2.35	* 4.10
	♀	W	4.82	3.87	* 3.95	3.25	* 3.52
		NW	4.46	5.57	4.16	2.40	* 3.24
Esophagus	♂	W	4.10	3.43	* 4.22**	2.70	* 3.67
		NW	9.44	6.63	* 8.67	6.55	* 6.63
	♀	W	1.03	1.08	* 1.46**	1.78	1.05**
		NW	2.17	1.31	* 1.70	0.42	* 2.41**
Lungs	♂	W	37.98	34.48	* 39.38**	38.70	* 45.58**
		NW	36.67	31.76	* 37.20**	31.41	* 38.65**
	♀	W	6.29	6.07	5.70	6.17	* 6.78**
		NW	6.27	5.39	5.28	7.43	6.93**
Breast	♀	W	25.51	22.30	* 23.38	18.11	* 21.78
		NW	22.10	22.43	19.58	27.92	16.78
Stomach	♂	W	15.22	12.61	* 13.49	14.50	14.45
		NW	24.03	21.28	* 22.11	16.64	* 25.41**
	♀	W	7.70	6.86	* 6.97	7.37	6.86
		NW	10.69	7.96	* 9.71	9.71	9.61
Leukemia-Aleukemia	♂	W	8.81	5.93	5.78	5.75	* 5.76
		NW	5.77	4.96	* 5.88**	3.48	* 6.52**
	♀	W	5.74	8.66	* 9.00**	7.36	* 8.82**
		NW	3.88	3.34	* 4.06**	2.58	* 3.28
Cervix Uteri	♀	W	6.13	8.89	8.56**	9.93	9.43**
		NW	11.30	15.85	* 17.83**	17.46	16.42**
Corpus Uteri	♀	W	7.79	5.68	* 5.85	5.21	* 5.70
		NW	18.92	13.25	* 10.03	7.75	* 10.94

(continued)

normality of raw data would be requisite to all testing.

The findings from these analyses would be utilized in narrowing the field of choices of organic chemicals and their related cancer sites for pathways studies.

METHODS AND MATERIALS

The purposes of this study are to: 1) assist in the selection of candidate organic chemicals which are carcinogens or suspected carcinogens suitable for pathways studies; 2) determine if a relationship exists between cancer incidence and the organic chemical industry; and 3) aid in development of a statistical method of relating disease incidence to industry.

The 1977 Registry of Toxic Effects of Chemical Substances (Fairchild et al., 1977) was used to identify potentially carcinogenic and carcinogenic chemicals in the U.S. Information on 400 organic industrial chemicals and their 610 production locations was obtained from the Organic Chemical Producers' Data Base Program (Garner and Dzierlenga, 1976). This program includes data, when available, for each chemical on toxicity, production volumes, costs, emission factors, cross-indexed chemical tree, and Wiswesser Line Notation. Each production plant is classified according to original feedstock source, product slate, and whether it is refinery associated. Plant-specific information on each chemical includes production capacities, and production routes (Wilkins, 1976).

The carcinogenic chemicals selected were those having more than three production locations and whose production capacity was available. The list of chemicals includes benzene, phenol, carbon tetrachloride, formaldehyde, chloroform, tetraethyl lead, ethanol, and vinyl chloride. The CPCs selected contained producers of one or more of the selected chemicals.

Following a review of the literature, cancer sites chosen for representative mortality rates were: lung, rectum, stomach, prostate, large intestine, liver, female breast, esophagus, corpus uteri, cervix uteri, leukemia-aleukemia, aleukemia, total cancer, and multiple myeloma (for vinyl chloride CPCs only). Age-adjusted mortality rates/100,000 individuals are available for the years 1950-1969 by sex and race for each CPC (Mason et al., 1975). The analysis includes counties not classified as a CPC of any of the listed chemicals, increasing the data spread and minimizing bias. A random number table facilitated the selection of a county adjacent to or near such CPC, designated a non-CPC. Any CPC which does not contain a nonwhite population was excluded from calculations of the nonwhite cancer rates. The nearness of CPCs to non-CPCs lessens variation due to differences in lifestyles, meteorological variation, and urbanization. This may allow for exposure of the non-CPCs to the chemical of the analysis.

A paired T-test of cancer rates by organic chemical and linear regression analysis of such numerous production volumes and cancer rates are statistically invalid, by simultaneous inference. A large number of tests performed makes possible simultaneous statistical inferences (R. R. Kinnison, personal

communication). Mortality means were calculated of each site-specific cancer for all CPCs and non-CPCs.

These calculations and national cancer mortality means (Mason and McKay, 1974) were compared in order to determine whether a difference exists between cancer rates in the CPCs and non-CPCs chosen. The national means were used in conjunction with non-CPC values as a control.

RESULTS AND DISCUSSION

In 284 comparisons between mean cancer mortalities of CPCs and non-CPCs, 205 CPC means are higher, as shown in Table 1 and illustrated in Figures 3 through 19. Especially higher means occur for nonwhites in tetraethyl lead CPCs, all males in benzene CPCs, all females in chloroform CPCs, and nonwhite males in phenol CPCs (Figure 3).

A high rate of a cancer for a sex in both races suggests occupational exposures to cancer-causing agents encountered in the dissimilar environments (until recently) of the traditional male/female roles. This possibly explains the difference between mortality rate means in phenol CPCs for stomach cancer (Figure 4), tetraethyl lead CPCs for liver cancer (Figure 5), chloroform CPCs for leukemia-aleukemia (Figure 15), lung cancer in all of the CPCs (Figures 3, 7, and 8), and esophageal cancer in benzene CPCs (Figure 16).

A high rate of cancer for both sexes of only one race suggests exposures to cancer-causing agents encountered in different ethnic lifestyles because of dietary differences, alcohol and tobacco intake, differences in blue collar and white collar occupational exposures, living conditions, etc. Therefore, influences on non-industrial correlates are suggested by excess mortality rates of a cancer of either racial group. This is illustrated by stomach cancer rates in tetraethyl lead and chloroform CPCs (Figure 4), cervix uteri cancer rates in benzene CPCs (Figure 9), corpus uteri cancer rates in tetraethyl lead CPCs (Figure 5), rectal cancer rates in ethanol CPCs (Figure 6), rates of cancer of the large intestine in phenol CPCs (Figure 10), total cancer mortalities in tetraethyl lead, phenol, and ethanol CPCs (Figure 18), and liver cancer mortalities in phenol, and ethanol CPCs (Figure 18), and liver cancer mortalities in benzene, chloroform, formaldehyde, and phenol CPCs (Figures 13 and 19).

Similar comparisons between CPC cancer rates and national mean cancer rates are given in Table 1. The association of tetraethyl lead production and mortality is most obvious for nonwhites, noted in esophageal cancer (Figure 11), stomach cancer (Figure 4), cancer of the large intestine (Figure 10), and lung cancer (Figure 3). Of the 284 comparisons, 113 mean CPC rates are higher than the national means (Table 1). Corpus and cervix uteri cancer rates are higher than the national means in every CPC studied except for cancer of the corpus uteri in benzene CPCs (Table 1). In addition to tetraethyl lead CPCs (Figures 5 and 17), ethanol CPCs show an excess prostate cancer and female breast cancer mortality rate for both racial groups over national means (Figures 17 and 12). Leukemia-aleukemia rates are higher for all but nonwhite females in tetraethyl lead CPCs (Figure 14).

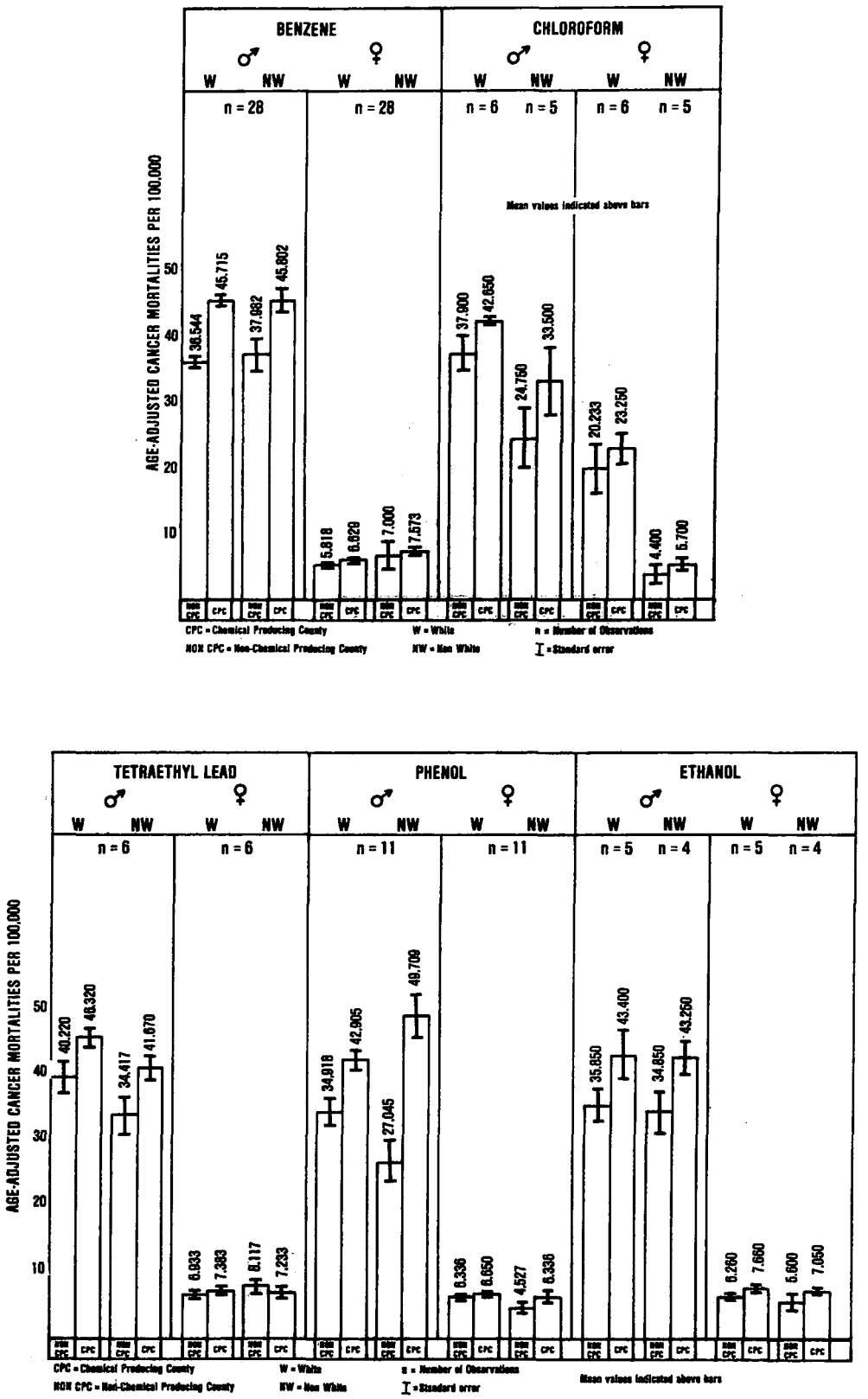


Figure 3. Mean age-adjusted lung cancer mortalities in CPCs and non-CPCs.

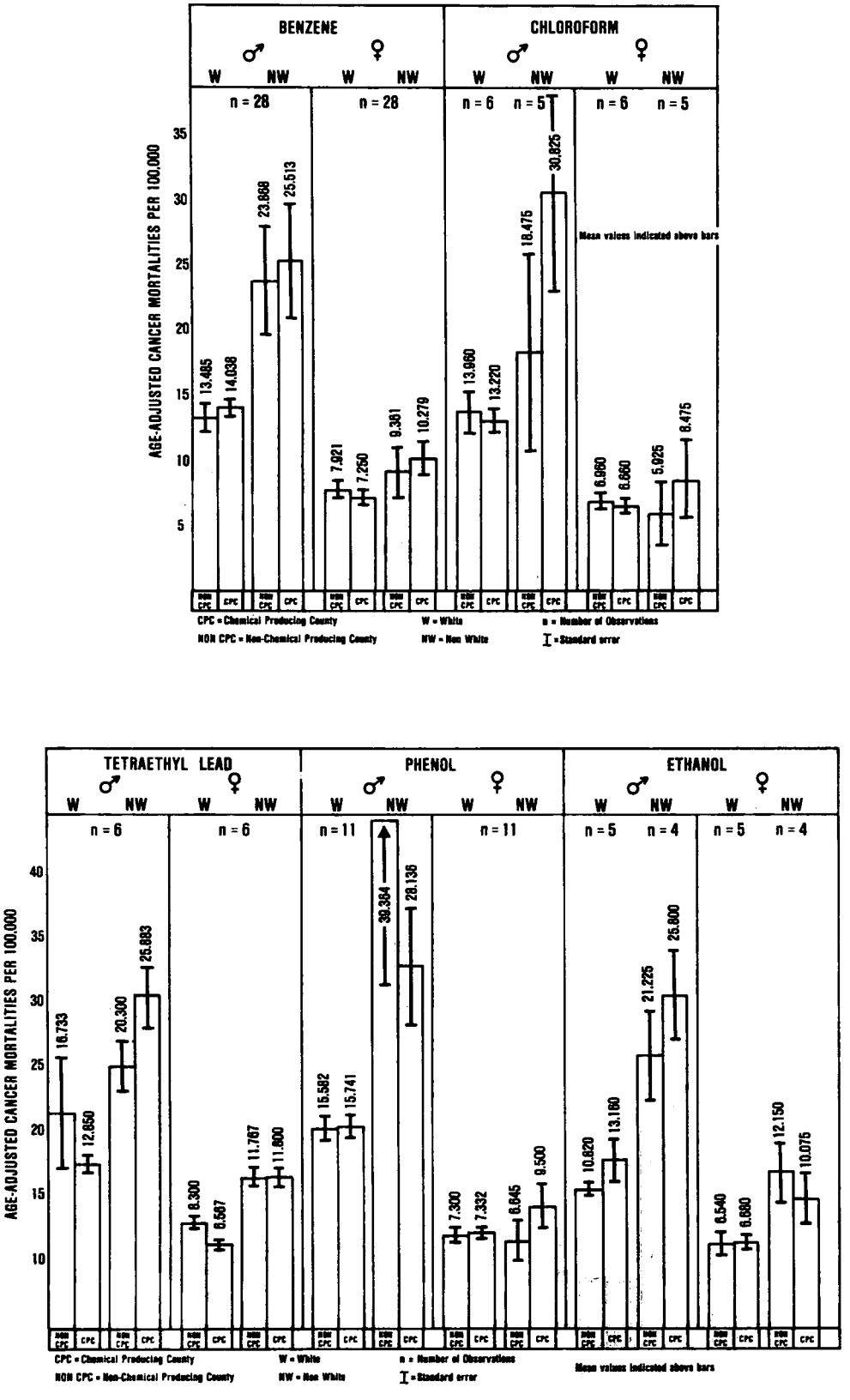


Figure 4. Mean age-adjusted stomach cancer mortalities in CPCs and non-CPCs.

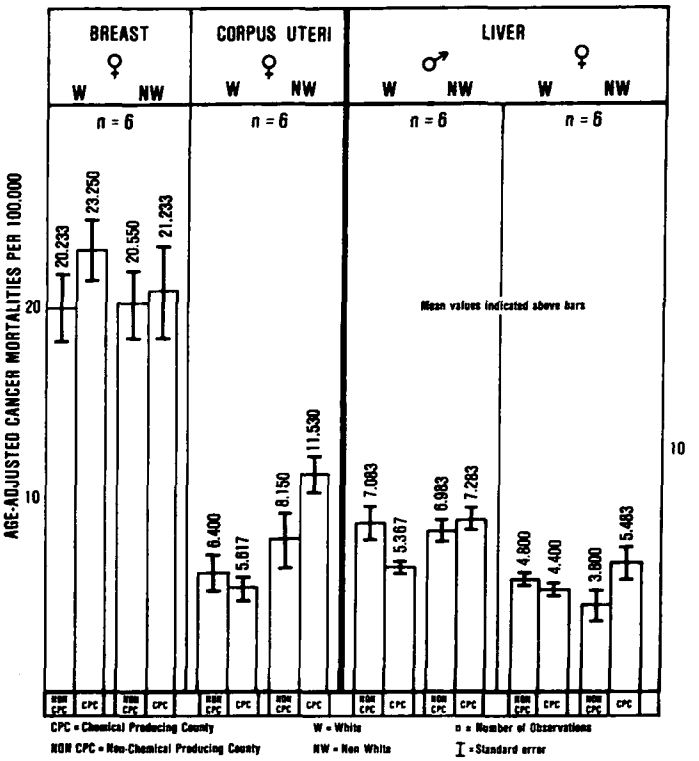


Figure 5. Mean age-adjusted cancer mortalities in tetraethyl lead CPCs and non-CPCs.

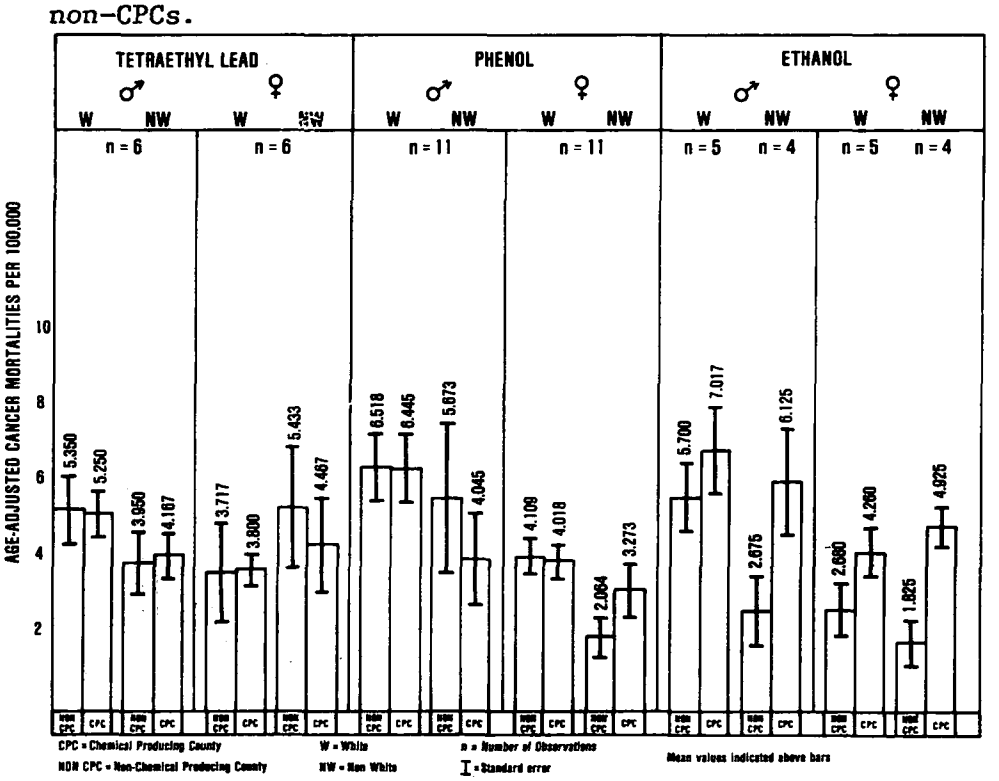
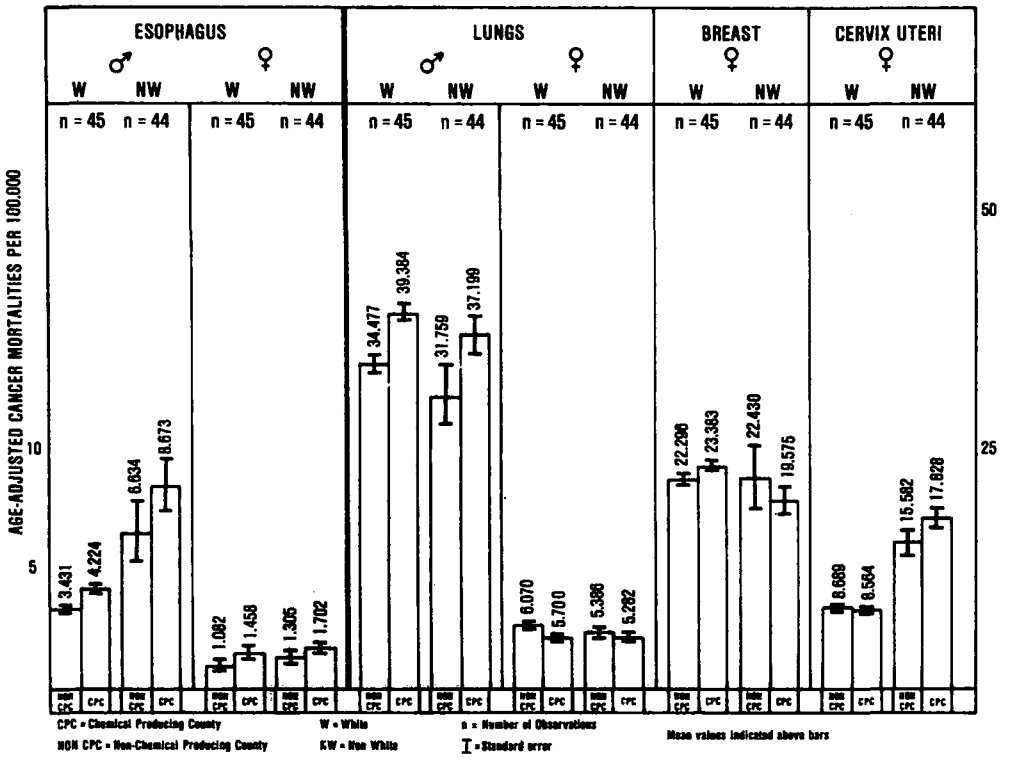


Figure 6. Mean age-adjusted rectal cancer mortalities in CPCs and non-CPCs.



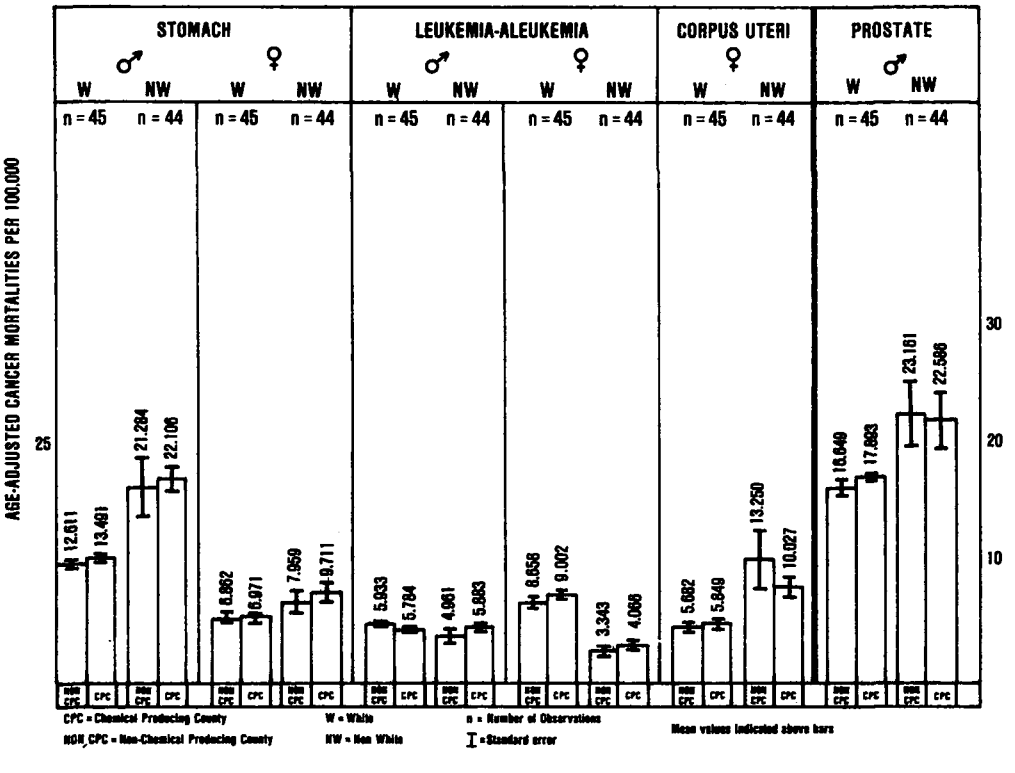


Figure 7. Mean age-adjusted cancer mortalities in formaldehyde CPCs and non-CPCs. (continued)

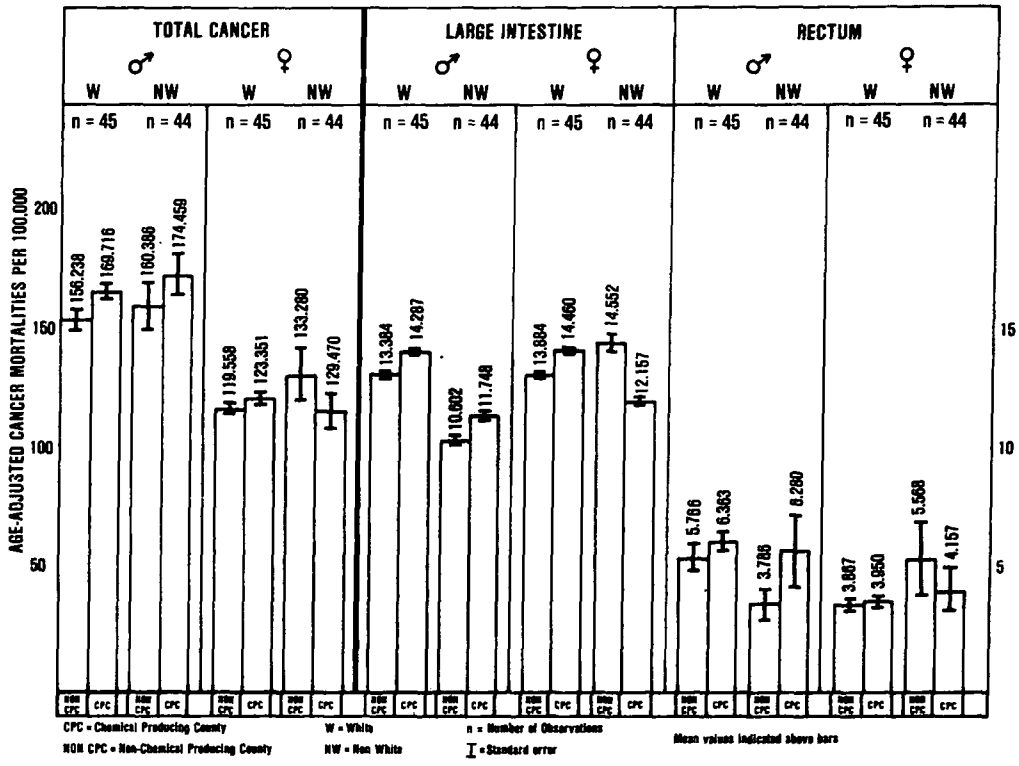


Figure 7. Mean age-adjusted cancer mortalities in formaldehyde CPCs and non-CPCs.

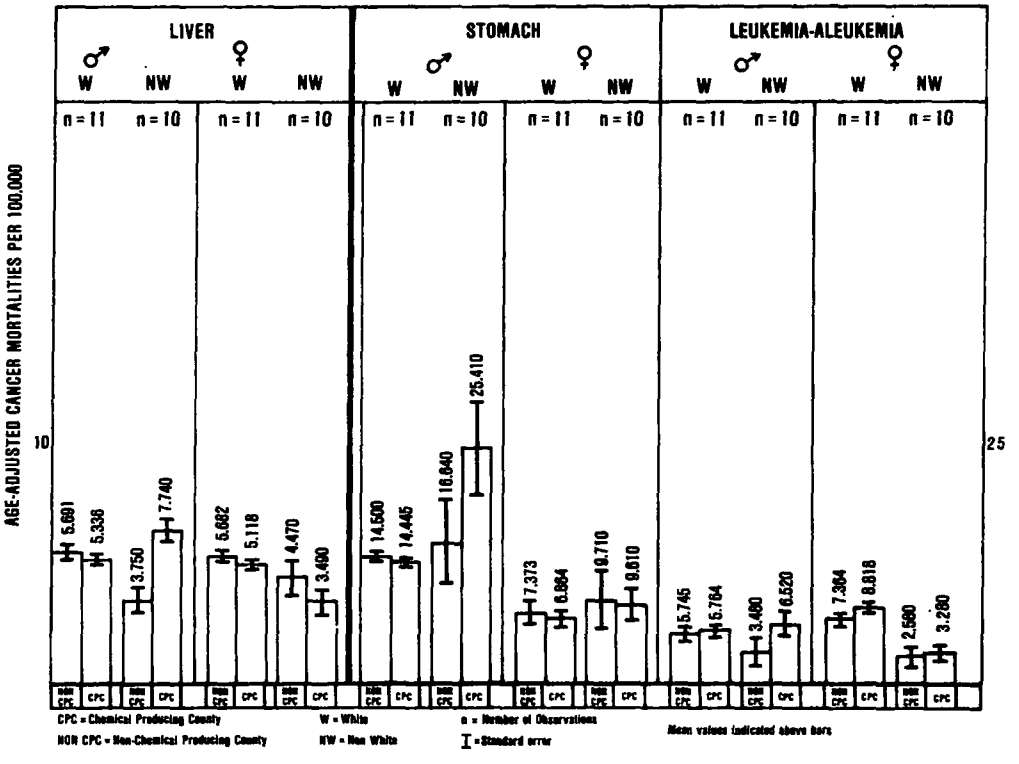


Figure 8. Mean age-adjusted cancer mortalities in carbon tetrachloride CPCs and non-CPCs. (continued)

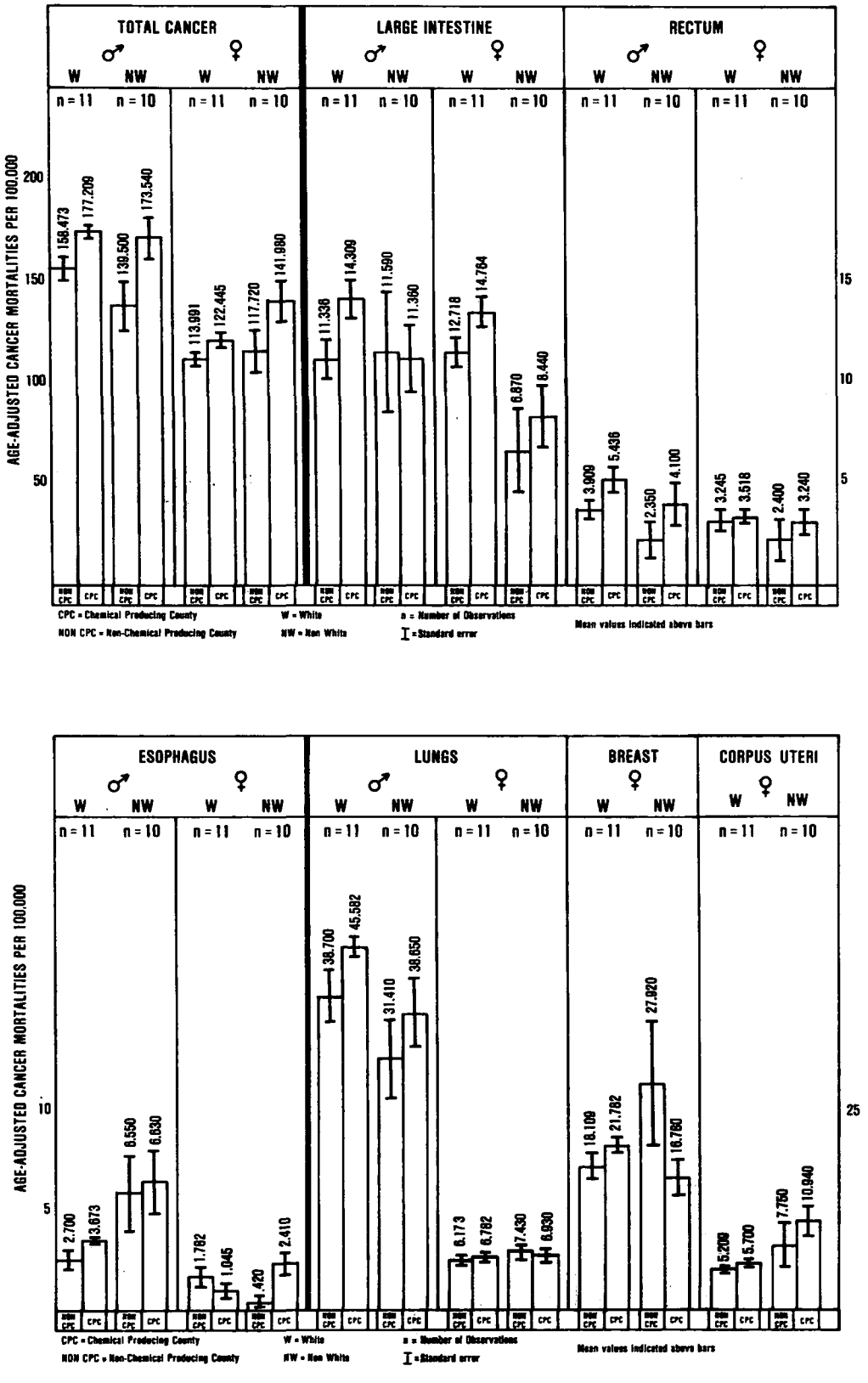


Figure 8. Mean age-adjusted cancer mortalities in carbon tetrachloride CPCs and non-CPCs.

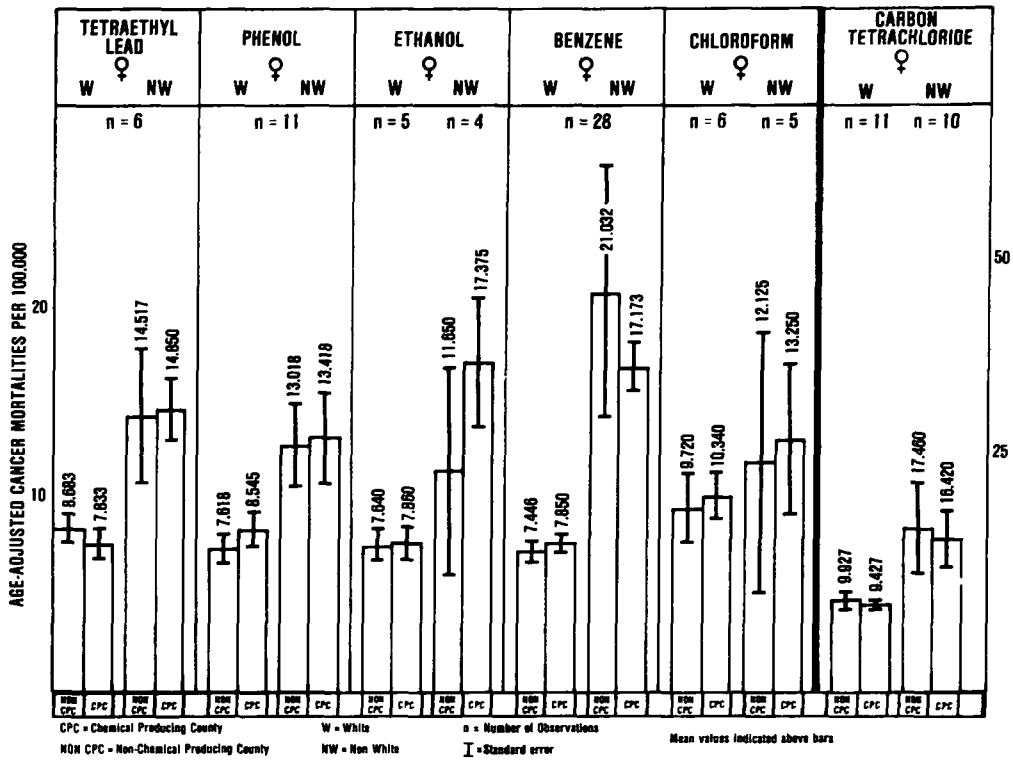


Figure 9. Mean age-adjusted cervix uteri cancer mortalities in CPCs and non-CPCs.

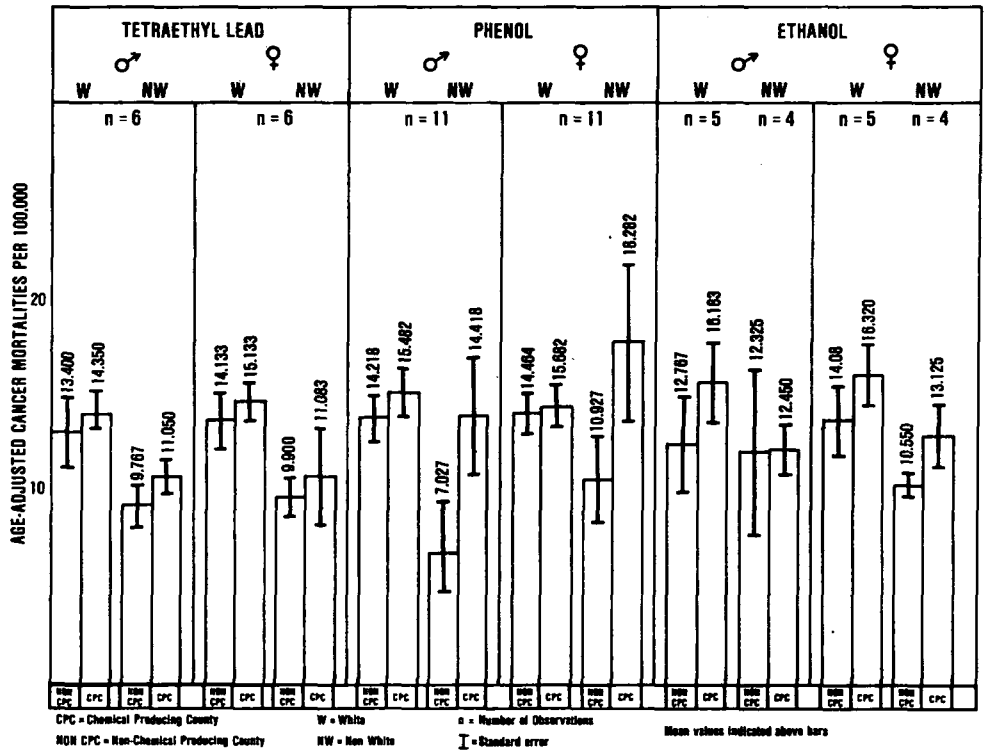


Figure 10. Mean age-adjusted large intestine cancer mortalities in CPCs and non-CPCs.

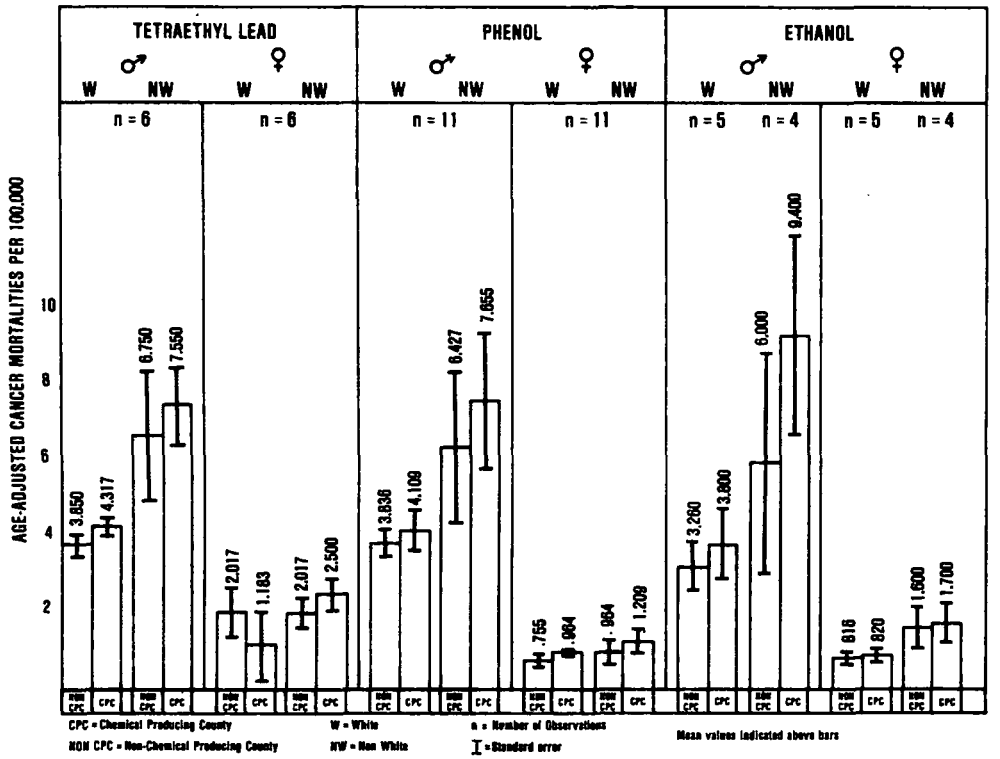


Figure 11. Mean age-adjusted esophageal cancer mortalities in CPCs and non-CPCs.

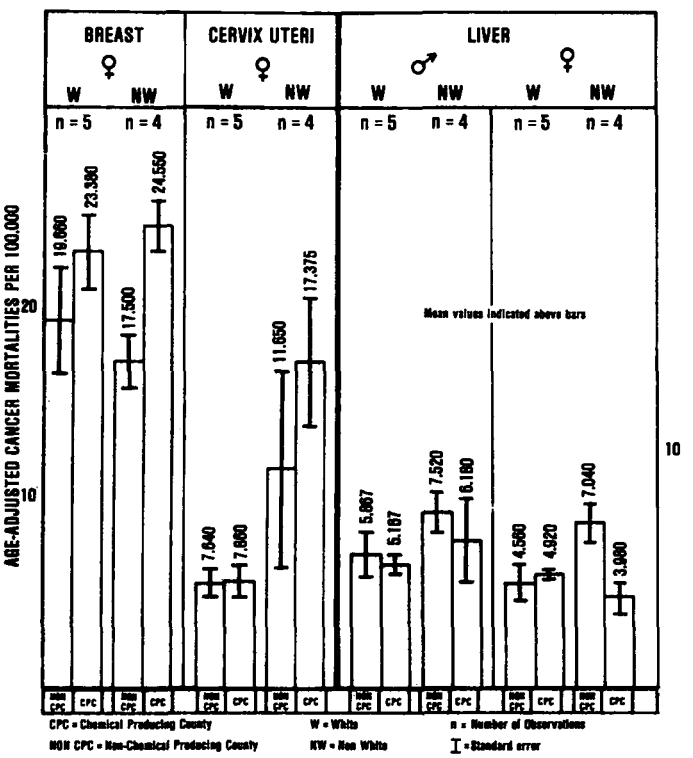


Figure 12. Mean age-adjusted cancer mortalities in ethanol CPCs and non-CPCs.

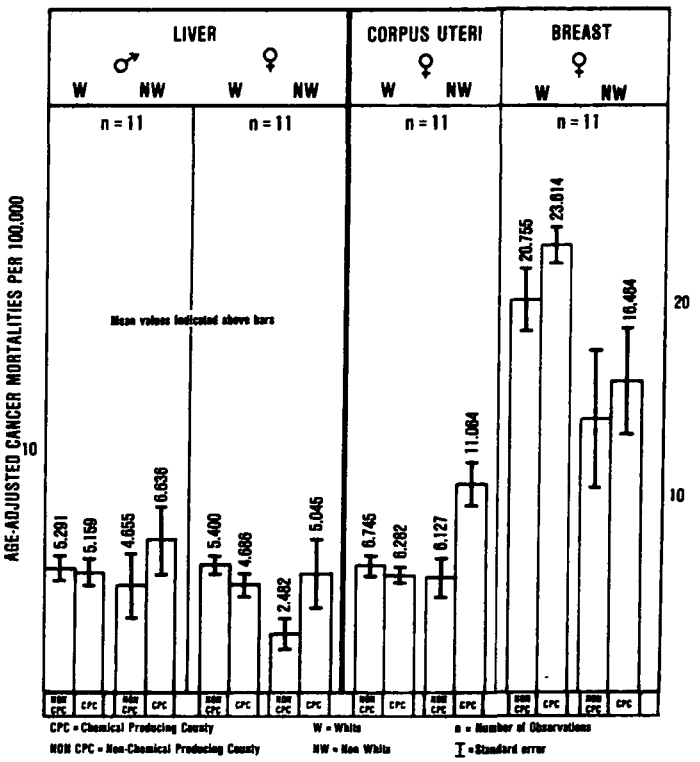


Figure 13. Mean age-adjusted cancer mortalities for phenol CPCs and non-CPCs.

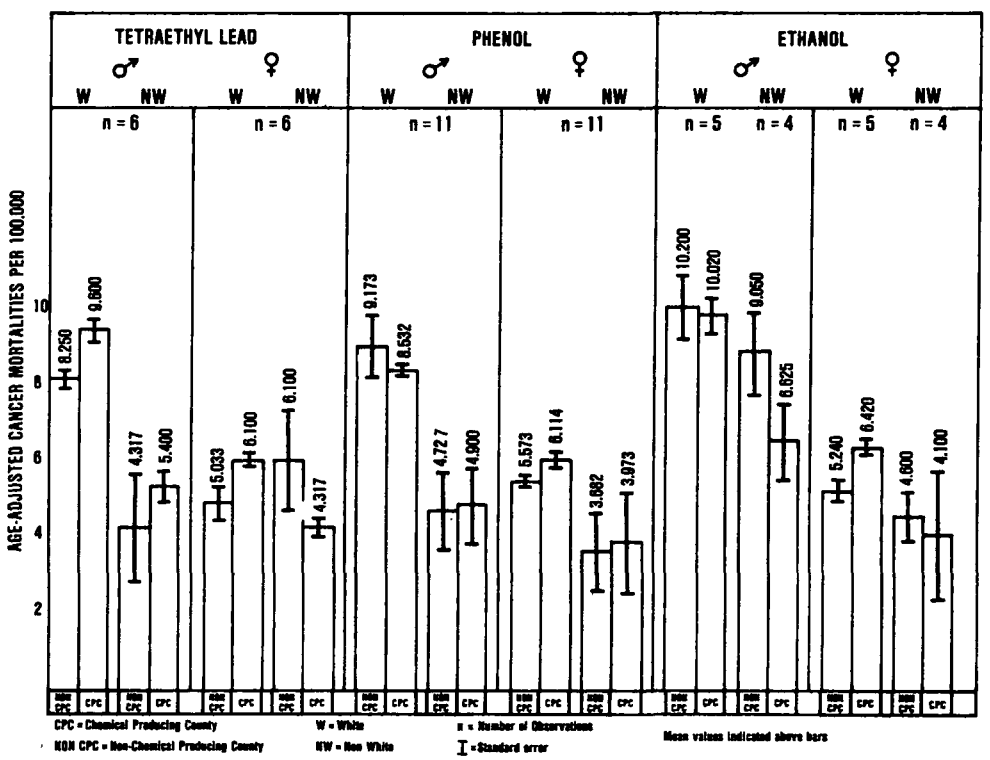


Figure 14. Mean age-adjusted leukemia/aleukemia mortalities for CPCs and non-CPCs.

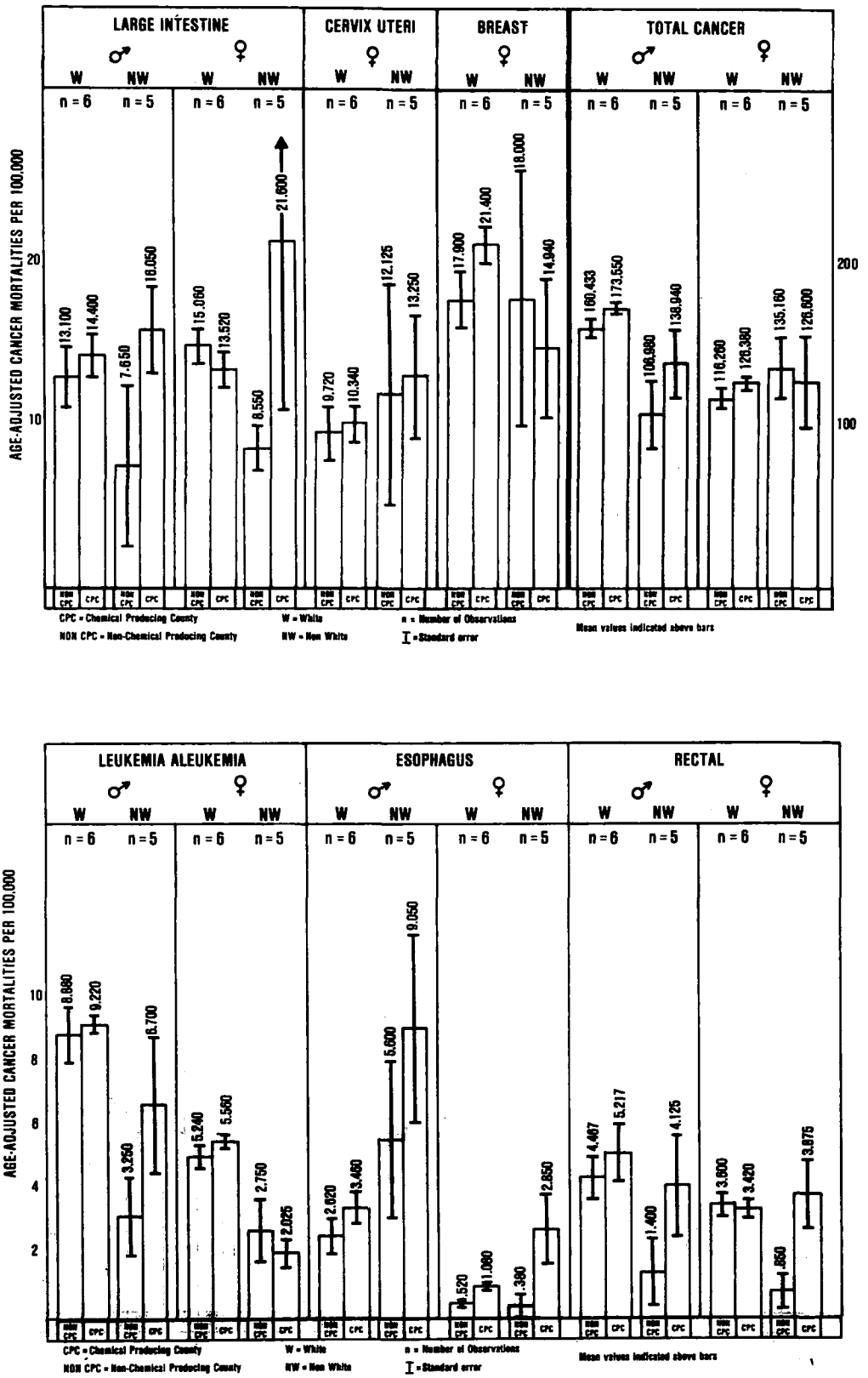


Figure 15. Mean age-adjusted cancer mortalities in chloroform CPCs and non-CPCs.

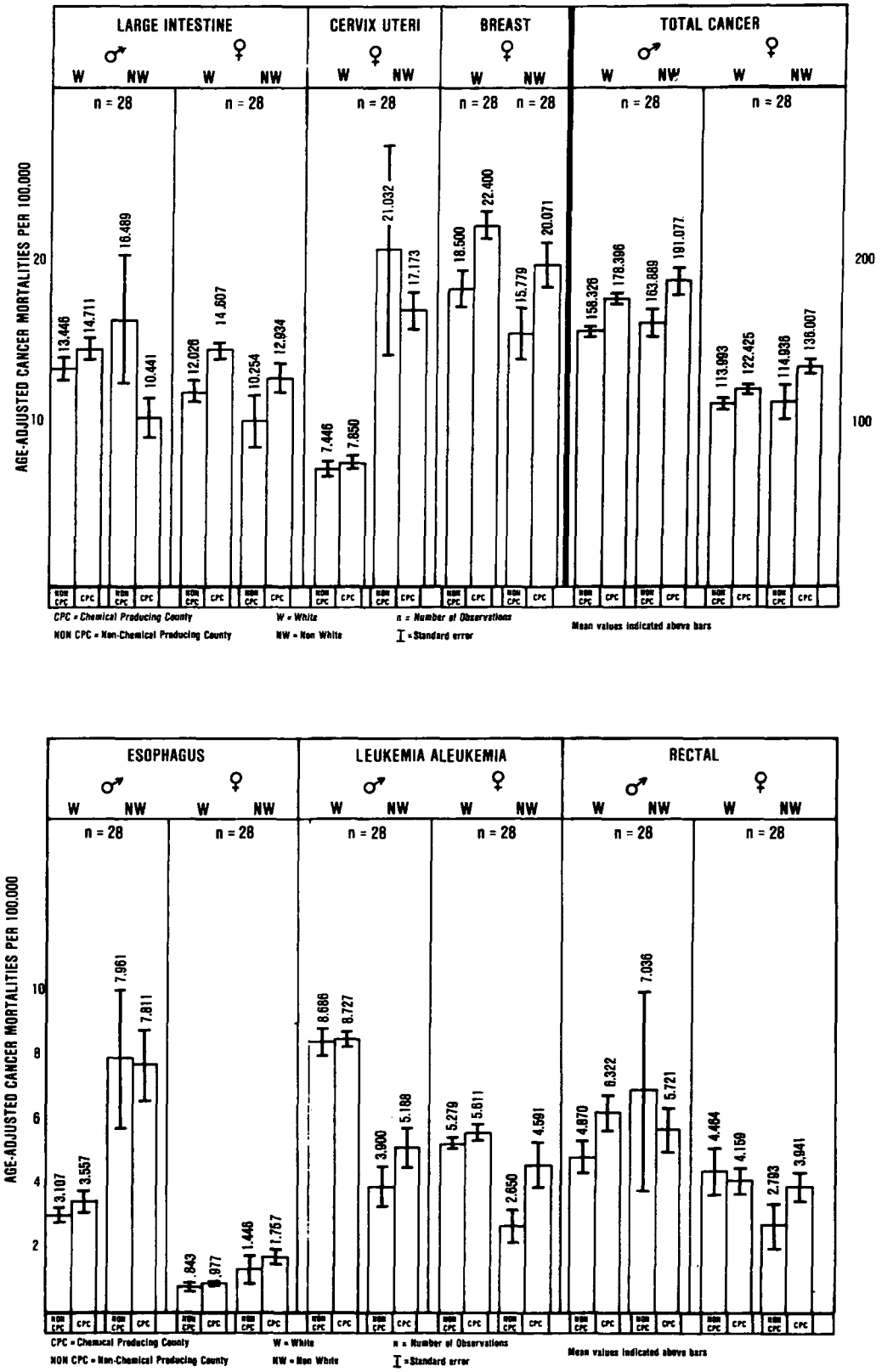


Figure 16. Mean age-adjusted cancer mortalities in benzene CPCs and non-CPCs.

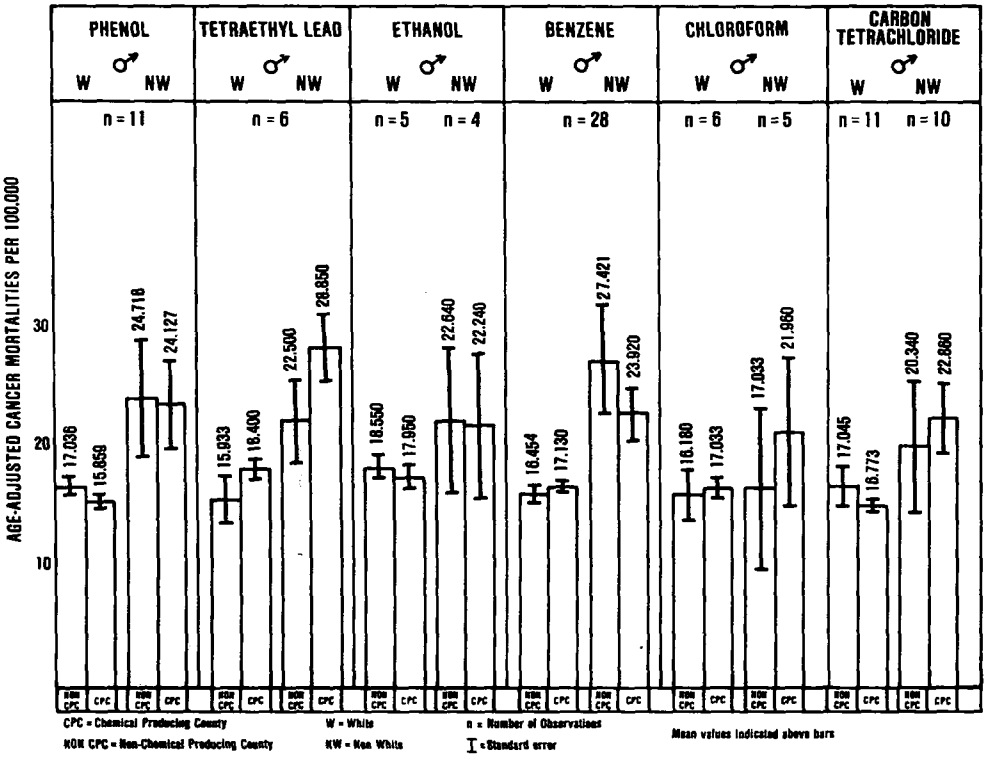


Figure 17. Mean age-adjusted prostate cancer mortalities in CPCs and non-CPCs.

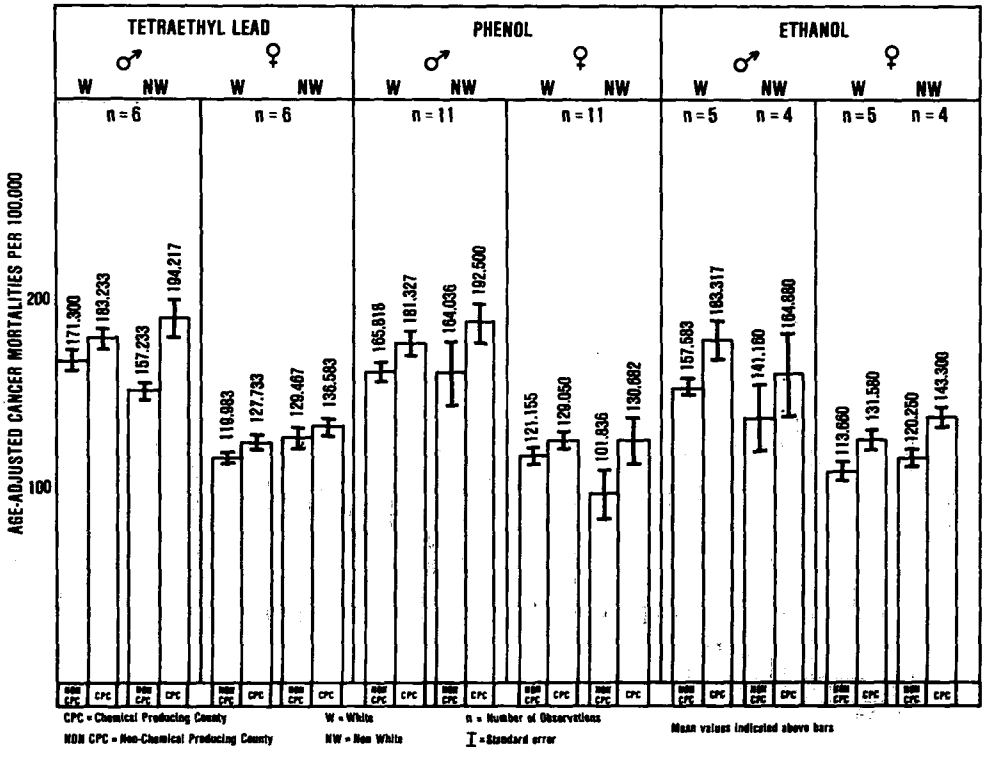


Figure 18. Mean age-adjusted total cancer mortalities in CPCs and non-CPCs.

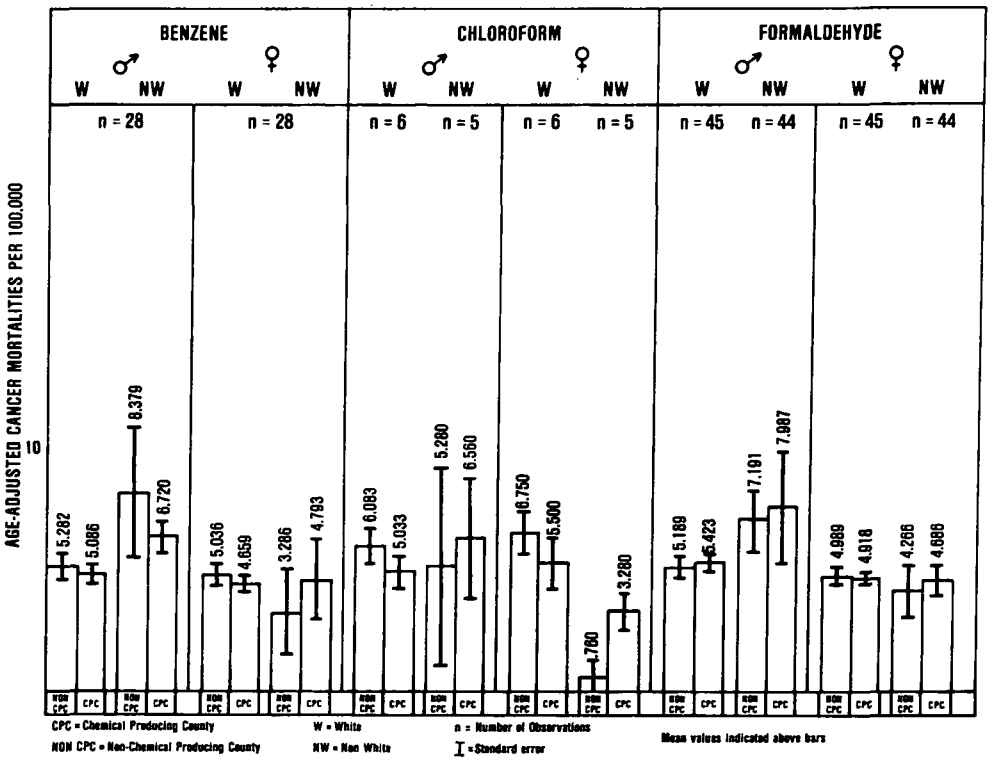


Figure 19. Mean age-adjusted liver cancer mortalities in CPCs and non-CPCs.

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16. ABSTRACT

This study is designed to determine whether there is an association between cancer mortality and the production of environmental carcinogens. Mortality rates of counties containing organic chemical production facilities are compared to rates of control counties. Twelve different cancer sites in lung, stomach, etc., and eight organic carcinogens were considered. Although a rigorous statistical analysis was not conducted, for most cancer sites mortality rates were found to be higher in counties of organic carcinogen production than in control counties. The study aids in the development of statistical techniques for determining the contribution of environmental contaminants to the rise in cancer rates. Research of this type assists in the identification of compounds that need to be regulated.

17. KEY WORDS AND DOCUMENT ANALYSIS

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