Mortality of Workers Employed at Organochlorine Pesticide Manufacturing Plants - An Update

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ABSTRACT

A previously published mortality study among workers at four organochlorine pesticide manufacturing plants was updated through 1987. The plants under study manufactured chlordane (plant 1), heptachlor and endrin (plant 2), aldrin, and dieldrin and endrin (plant 3), and dichloro -diphenyl -trichloroethane (DDT) (plant 4). Dibromochloropropane (DBCP) was also produced at plant 3 during part of the study period. The update added eleven years of observation, and increased the total number of deaths from 298 to 650. The mortality for "all causes" and "all malignant neoplasms" at each of the plants was lower than expected. There was a statistically significant increase in liver/biliary tract cancer among workers at plant 3 (5 obs, SMR 393; CI 1.27-9.20). These results are somewhat consistent with the experimental animal findings where benign and malignant tumors of the liver were found after exposure to aldrin and dieldrin, however; the observed deaths in the workers were a mixture of intra- and extrahepatic tumors. Because of the small number of deaths and lack of exposure information, dose response analyses were limited. Additional study of this group should include a case control analysis of the liver/biliary cancer deaths at plant 3, and continued follow up of the total cohort.

Introduction:

In a previous study, the mortality of four cohorts employed at organochlorine pesticide manufacturing plants was reported (1). The report concluded that additional follow up was necessary due to the small number of deaths and the relatively short period of observation. The mortality of the cohorts has now been updated from 1976 through December 31, 1987, and the results are reported here.

The primary purpose of the study was to assess the carcinogenic risk among workers exposed to the following organochlorine pesticides: chlordane (plant 1), heptachlor and endrin (plant 2), aldrin, dieldrin, and endrin (plant 3), and dichloro-diphenyl-trichloroethane (DDT) (plant 4). Dibromochloropropane (DBCP) an organobromine pesticide was also manufactured at plant 3. According to the latest information from EPA's Integrated Risk Information System (IRIS) (2), all of these pesticides have been classified as probable human carcinogens except DBCP which has not been evaluated by IRIS and Endrin which was considered "not classifiable." Benign and malignant liver tumors were the most common tumors found in the

animal studies. The International Agency for Research on Cancer (IARC) (3) has determined that DDT and DBCP are potential human carcinogens. The others have not been classified by IARC. The National Toxicology Program (4) has classified DDT and DBCP as animal carcinogens. The carcinogenic classification of these pesticides is summarized in Table 1.

A number of recent epidemiologic studies (5,6,7) designed to examine the carcinogenicity of several of these same pesticides have also been conducted. The studies have not demonstrated an association between exposure to these pesticides and an increase in cancer; however, most of the studies are based on small populations, and the power to detect risks for rare causes of death such as liver cancer is weak.

Methods:

Each of the four study cohorts was defined as all white male workers who were employed for at least six months prior to December 31, 1964 at the plants under study. The personnel records from each of the plants were used to identify the cohorts. When the work history information was provided in sufficient detail, workers in non-production jobs such as office workers were excluded. In most cases this was possible and therefore, few non-production workers who had limited access to the process areas were included in the study. Additional work histories for cohort members who were active at the time records were collected for the original study, were not obtained for the update. No independent record systems were used to verify the completeness of the cohort. However, checks were run on the data to determine if there were missing records. No obvious gaps in the data were found.

For this update, follow-up of vital status was extended from the end of 1976 to December 31, 1987. Vital status for members of the cohort was determined by searching records maintained by the Social Security Administration, the Internal Revenue Service, and

the National Death Index. Death certificates for deceased workers were obtained from the state vital records offices, and the underlying cause of death was coded by a qualified nosologist according to the revision of the International Classification of Diseases (ICD) in effect at the time of death.

The cohorts were restricted to white male workers, since they represented the vast majority of the workforce at all the plants. Those with unknown race were assumed to be white. The NIOSH life table analysis system (LTAS) (8) was used to calculate standardized mortality ratios for specific causes of death. The LTAS distributes the person years at risk (PYAR) for each worker into strata by age and calendar year and further stratifies the PYAR by length of employment and time since first employment (latency). PYAR for each worker were accumulated from the time the worker achieved six months of employment to the end of the study (12/31/87) or the date of death whichever occurred first. For those lost to follow-up, PYAR were accumulated to the end of the study. The PYAR were multiplied by the corresponding age and calendar specific, white, male, U.S. mortality rates to yield expected numbers of death. For plant 3, Colorado State and county (Denver, Arapahoe, Jefferson, Douglas, and Adams County)

mortality rates were also used to calculate expected deaths. The use of regional rates was added to the analysis for plant 3 because of the known high incidence rate for respiratory disease in the Denver area. Two sided 95% confidence intervals were computed for each cause-specific SMR using the Byar approximation when deaths were eight or greater, or the Fisher exact method when the deaths were fewer than eight (9).

There were no historical exposure measurements available for the study. At each plant the workers were potentially exposed to multiple chemicals used in the manufacturing process as well as to the pesticides themselves. A list of potential exposures was included in the original publication (1). There are data from plant 4 on levels of exposure to DDT among 35 workers employed in 1967. The DDT in the fat of workers ranged from 38 to 647 ppm compared to an average of 8 ppm in the general population; and it was estimated that the average daily intake of DDT among the high exposed group was 17.5 to 18 mg/man/day compared to 0.04 mg/man/day in the general population (10).

Results:

The results of the vital status ascertainment are given in Table 2. The total number of cohort members differs slightly from the original study because some workers in plant 1 were included in the update that were previously excluded; and workers who previously had an unknown race status were subsequently excluded if they were determined to be non-white. The increase in number of deaths for each cohort compared to the initial study is given in Table 2. Among the 650 deaths, death certificates were obtained for all except eight individuals. These individuals were considered dead with an unknown cause of death.

Table 3 gives the cause specific mortality results by major cause and by individual plant. For all plants combined, the observed mortality for "all causes" is significantly less than expected and cause specific mortality is less than expected for all major causes of death except for cerebrovascular disease and respiratory disease.

The "all causes" mortality is less than expected for each of the four plants and significantly lower than expected at plant 3 (337

obs, SMR 0.87; CI 0.78-0.97). The mortality for "all malignant neoplasms" is also equal to or lower than expected in each of the plants. Cerebrovascular disease is higher than expected in three of the plants. There is a statistically significant excess of deaths from this cause at plant 4, (11 obs, SMR 2.38; CI 1.19-4.26), but a significant deficit at plant 3 (13 obs, SMR 0.56; CI 0.30-0.97). Diseases of the respiratory system at plant 3 is significantly elevated compared to U.S. rates, (37 obs, SMR 1.56; CI 1.10-2.16). When Colorado State and the surrounding county rates were used to calculate expected deaths (Table 4), the increase in non-malignant respiratory disease (ICD 460-519) at plant 3 is substantially reduced and is no longer statistically significant, although it is still slightly elevated. The risk for cerebrovascular disease is no longer significantly low when these regional rates were used. No other specific cause of death was affected by the use of regional rates.

Table 5 gives the cancer specific mortality by plant and for all plants combined. When all the cohorts are combined, the only excess risks by specific cancer site are for stomach and liver cancer. When examined by specific plant, stomach cancer is

non-significantly elevated in three out of the four plants. Respiratory cancer is 33% higher than expected at plant 1. There was a significant increase in bladder cancer at plant 2. All of these bladder cancer deaths occurred after 10 years of employment and two occurred after 20 years of latency and 20 years of employment (2 obs, SMR 15.38; 95% CI 2.58-50.82).

Probably the most important finding is the statistically significant increase in liver/biliary tract cancer (9th revision ICD codes 155.0, 155.1, 156) among the workers at plant 3 (5 obs, SMR 3.93; 95% CI 1.27-9.20). The increased risk at plant 3 was statistically significant when state or county rates were used to calculate expected deaths (Table 4). According to death certificate information, three of the deaths from plant 3 were due to biliary tract or bile duct cancer, one from gall bladder, and one hepatoma. Among the workers in plant 3, all of the liver cancer deaths occurred after 15 years of latency (1.03 were expected, SMR=4.85). There were 3 observed versus 0.32 expected deaths (SMR=9.38) in the less than 2 years employment strata; and 2 observed versus 0.45 expected (SMR=4.44) in the greater than 2 years employment strata.

At plant 4, there were two deaths from liver/biliary tract cancer. However, for one of these deaths, an adenocarcinoma of the liver, the primary site was unspecified . The other death was due to gall bladder cancer. The expected number of primary and unspecified cancer deaths for this cause at plant 4 was 0.45. More details on the liver cancer deaths from plants 3 and 4 are given in Table 6.

At plants 1 and 2 there were no observed deaths from liver/biliary tract cancer, however, only 0.64 deaths were expected at plant 1 and 0.26 deaths were expected at plant 2.

Discussion:

This update of a previously published study of pesticide manufacturers adds eleven years of follow-up, more than doubling the total number of deaths and provides approximately 40 years of observation for each cohort. A minimun of 23 years (12/31/64 to 12/31/87) has elapsed since each cohort member was first employed at the study plants, allowing for diseases with long latency periods to develop.

The excess in liver/biliary tract cancer deaths observed in plants 3 and 4 is important because it may be relevant to the a priori disease of interest based on the animal studies. In subchronic studies of rats and mice, the liver was the primary target organ. Both aldrin and dieldrin induced hepatocellular carcinomas and benign liver tumors in mice. Studies in rats resulted in effects on the liver, but no increase in malignant liver tumors (6). When interpreting these results a number of factors must be considered. First, there is a lack of quantitative information on exposures to the pesticides as well as to precursor chemicals used in the manufacturing process. Second, since DBCP was manufactured at plant 3 between 1955 and

1976 and it is classified as an animal carcinogen, it has to be considered a potential confounding exposure. Two of the liver/biliary tract cancer deaths were included in the NIOSH DBCP registry. This is a national exposure registry that includes plant 3 and is based on voluntary submittal of names from employers of workers with at least 30 hours employment in areas of the plant with potential exposure to DBCP. Third, the liver cancers are not a homogeneous type, but are a mixture of extrahepatic (biliary tract and gall bladder) and intrahepatic tumors, whereas; the experimental animal studies resulted in intrahepatic tumors. Whether this is important etiologically, is not known. Similar experimental and epidemiological results have been found in studies of polychlorinated biphenyls (11) and dinitrotoluene (personal communication from Dr. Leslie Stayner, NIOSH). Fourth, there does not appear to be a dose response relationship for liver/biliary tract cancer when dose is measured as length of employment. However, this type of analysis is crude since there is no exposure data and a positive dose response is difficult to demonstrate with so few deaths. There were only two deaths from liver cancer that had worked at the plant for more than two years.

The statistically significant excess in bladder cancer deaths at plant 2 was unexpected since no known bladder carcinogens were known to be used at this plant. The small number of deaths (3 observed) makes it difficult to interpret these findings. It should be noted, however, that since this is a mortality analysis and bladder cancer survival has improved, a better estimate of the true risk would require an analysis of incident cases rather than deaths. In other studies of occupationally induced bladder cancer, mortality alone did not detect a risk, whereas overall incidence revealed large excess risks (12,13).

The findings for cerebrovascular disease mortality were of interest because three of the four plants had an excess risk with plant 4 having a statistically significant excess. This observation was not part of an a priori hypothesis and could have been due to chance or differences among the study population in risk factors related to cerebrovascular disease. The potential exposures at these plants have not been previously associated with an increased risk for cerebrovascular disease. However, according to a recent report (14), a study of workers' compensation claimants in California found a significant increase in deaths from cerebrovascular disease (PMR 2.95, 95% CI 1.18-

6.08) among workers in the agriculture industry. Since these claims were filed between 1945 and 1963, these workers had potential exposure to organochlorine pesticides.

There are two epidemiologic studies of workers from pesticide manufacturing plants that have been completed since the publication of the original study. Both of these studies were designed to evaluate the cancer risks among workers exposed to organochlorine pesticides. Shindell and Ulrich (5) performed a mortality analysis on the workers from plant 1 where chlordane was manufactured. This included all workers with at least three months of employment between January 1946 and June 1985, and therefore; there was considerable overlap between their study cohort and plant 1 in this report. The results of the two studies are similar; there was a deficit in cancer mortality (SMR=0.91) and an increase in cerebrovascular disease (SMR=1.71).

A mortality study of aldrin/dieldrin workers at a plant located in the Netherlands was recently completed (6). This study included 570 workers who were employed for at least one year between 1954 and 1970. There were only 75 deaths in this study so it is difficult to draw any conclusions. There were 32

observed and 30.9 expected neoplasms and there were no significant increases in specific cancer types. There was one liver cancer whereas 0.6 was expected. There were also no trends when risk for various causes was examined by exposure groupings.

Wong et al, (7) studied the mortality of chemical workers from three manufacturing plants where there was exposure to brominated chemicals, including DBCP, and to DDT. There was a lack of historical exposure information and many of the workers were exposed to multiple chemicals. Among a small group of 238 workers with potential exposure to DBCP there was a significant excess in arteriosclerotic heart disease (6 observed and 2.17 expected). In the Wong study, there were 740 workers exposed to DDT and 112 deaths. There were no liver cancer deaths and no statistically significant excess in cause specific mortality. The authors point out that there were inherent deficiencies in the study including small sample size, multiple exposures, and lack of information on historical exposure.

In summary, this updated analysis has provided additional information on the possible carcinogenicity of organochlorine pesticides. The most important result is the statistically

significant increase in liver/biliary tract cancer among workers at plant 3 where aldrin/dieldrin were the primary organochlorine pesticides produced and the nonsignificant increase at plant 4 which manufactured DDT. The study is limited by the lack of exposure data including the potential for confounding exposures to other chemical compounds, and the relatively small size of the cohorts to adequately assess mortality from rare diseases.

Since one of the primary limitations of this study is the lack of exposure information and the small number of liver cancer deaths, further follow-up and a subsequent nested case control study should be considered. Of course the feasibility of this type of study is dependent on the availability of at least qualitative exposure data.

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Table 1. Review of Pesticide Toxicity

Pesticide	IRIS			IARC			NTP	
	Animal	Human	Class	Animal	Human	Class	Animal	Human
Aldrin	S	I	B2	L	I	3	---	---
Dieldrin	S	I	B2	L	I	3	---	---
Chlordane	S	I	B2	L	I	3	---	---
Heptachlor	S	I	B2	L	I	3	---	---
Endrin	I	I	D	I	ND	3	---	---
DDT	S	I	B2	S	I	2B	S	I
DBCP	---	---	---	S	I	2B	S	I

S = sufficient
I = inadequate
L = limited
D = not classifiable as to carcinogenicity to humans
B2 = probable human carcinogens
2B = possibly human carcinogens
3 = not classifiable as to carcinogenicity to humans

IRIS = Integrated Risk Information System (2)
IARC = International Agency for Research on Cancer (3)
NTP = National Toxicology Program (4)

Table 2. Vital Status follow-up as of 12/31/87 of Workers in Organochlorine Pesticide Plants.

	Plant 1 (Chlordane)	Plant 2 (Heptachlor, Endrin)	Plant 3 (Aldrin, Dieldrin)	Plant 4 (DDT)	Total
Alive	245	240	803	230	1518
Deceased (Deceased in origianl report)	159 (59)	64 (24)	337 (173)	90 (42)	650 (298)
Unknown	1	1	13	8	23
Total	405	305	1,153	328	2191
Person-years (Person-years in original report)	13,191 (8,354)	8,477 (5,672)	34,479 (24,939)	9,797 (7,601)	65,944 (46,566)

Table 3. Cause-specific mortality among workers in organochlorine pesticide plants, based on U.S. mortality rates.

Cause of Death (9th Revision ICD)	Plant 1 (Chlordane) Obs	Plant 1 (Chlordane) SMR (95% CI)	Plant 2 (Heptachlor, Endrin) Obs	Plant 2 (Heptachlor, Endrin) SMR (95% CI)	Plant 3 (Aldrin, Dieldrin) Obs	Plant 3 (Aldrin, Dieldrin) SMR (95% CI)	Plant 4 (DDT) Obs	Plant 4 (DDT) SMR (95% CI)	Total Obs	Total SMR (95% CI)
All Malignant Neoplasms (140-208)	35	0.87 (.61-1.22)	18	1.00 (.60-1.59)	72	0.86 (.67-1.08)	18	0.87 (.52-1.39)	143	0.88 (.74-1.04)
Heart (390-429, except, 401, 403, 405, 415-417)	67	0.85 (.66-1.09)	25	0.80 (.52-1.18)	123	0.77 (.65-.93)	32	0.87 (.60-1.24)	247	0.81 (.71-.91)
Cerebrovascular (430-438)	18	1.52 (.90-2.41)	6	1.56 (.57-3.40)	13	0.56 (.30-.97)	11	2.38 (1.19-4.26)	48	1.10 (.81-1.46)
Respiratory System (460-519)	10	0.83 (.40-1.54)	5	1.11 (.36-2.60)	37	1.56 (1.10-2.16)	5	0.93 (.30-2.19)	57	1.25 (.95-1.62)
Digestive System (520-579)	6	0.68 (.25-1.48)	2	0.47 (.06-1.70)	18	0.93 (.55-1.48)	7	1.44 (.58-2.98)	33	0.89 (.61-1.24)
Cirrhosis of liver (571)	3	0.67 (.14-1.96)	1	0.40 (.01-2.22)	10	0.96 (.45-1.77)	6	2.15 (.79-4.70)	20	0.99 (.60-1.53)
Genito-Urinary System (580-629)	3	1.22 (.25-3.58)	0	---	4	0.82 (.22-2.11)	0	---	7	0.95 (.37-1.92)
Accidents (E800-949)	8	0.80 (.34-1.59)	3	0.52 (.11-1.53)	20	0.81 (.50-1.26)	5	0.73 (.24-1.72)	36	0.76 (.53-1.05)
Violence (E950-978)	3	0.67 (.14-1.96)	0	---	13	1.13 (.61-1.95)	2	0.62 (.08-2.26)	18	0.94 (.56-1.48)
All Other	9	---	5	---	37	---	10	---	61	---
All Causes	159	0.85 (.73-1.00)	64	0.81 (.63-1.04)	337	0.87 (.78-.97)	90	0.98 (.79-1.21)	650	0.87 (.80-.94)

Table 4. Cause-Specific Mortality for Plant 3 workers based on National, State and County Rates.

Cause of Death (9th Revision ICD)	National Rates Obs	National Rates SMR 95% CI	State Rates[1] Obs	State Rates[1] SMR 95% CI	County Rates[1] Obs	County Rates[1] SMR 95% CI
All Malignant Neoplasm (140-208)	72	0.86 (.67-1.08)	69	1.11 (.87-1.41)	69	1.04 (.82-1.33)
•liver, biliary, gallbladder (155.0, 155.1, 156)	5	3.93 (1.27-9.20)	5	5.10 (1.65-11.91)	5	4.86 (1.57-11.36)
•Respiratory Cancer (160-165)	20	0.66 (.40-1.02)	19	0.92 (.56-1.45)	19	0.87 (.52-1.36)
•Lymphatic and hemato. (200-208)	10	1.26 (.60-2.32)	10	1.51 (.72-2.79)	10	1.45 (.70-2.68)
Heart Disease (390-429, ex. 401, 403, 405, 415-417)	123	0.77 (.65-.93)	110	0.93 (.77-1.12)	110	0.91 (.75-1.09)
Cerebrovascular (430-438)	13	0.56 (.30-.97)	12	0.74 (.38-1.30)	12	0.76 (.39-1.33)
Respiratory Disease (460-519)	37	1.56 (1.10-2.16)	36	1.21 (.85-1.68)	36	1.11 (.78-1.54)
•Pneumonia	14	1.73 (.95-2.91)	13	1.36 (.73-2.34)	13	1.14 (.61-1.95)
•Pneumoconiosis and other (COPD)	15	1.72 (.96-2.84)	15	1.29 (.73-2.14)	15	1.20 (.67-1.98)
All Causes	337	0.87 (.78-0.97)	307	0.98 (.87-1.10)	307	0.98 (.84-1.06)

[1] State and County rates used in analysis start in 1960, therefore observed and expected deaths that occur before this date are exluded.

Table 5. Cause-Specific Cancer Mortality Among Workers in Organochlorine Pesticide Plants

Cause of Death (9th Revision ICD)	Plant 1 (Chlordane) Obs	Plant 1 (Chlordane) SMR (95% CI)	Plant 2 (Heptachlor, Endrin) Obs	Plant 2 (Heptachlor, Endrin) SMR (95% CI)	Plant 3 (Aldrin, Dieldrin) Obs	Plant 3 (Aldrin, Dieldrin) SMR (95% CI)	Plant 4 (DDT) Obs	Plant 4 (DDT) SMR (95% CI)	Total Obs	Total SMR (95% CI)
All Malignant Neoplasms (140-208)	35	0.87 (.61-1.22)	18	1.00 (.59-1.59)	72	0.86 (.67-1.08)	18	0.87 (.52-1.39)	143	0.88 (.74-1.04)
• Buccal cavity/Pharynx (140-149)	0	---	0	---	3	1.19 (.25-3.49)	0	---	3	0.61 (.16-1.67)
• Stomach (151)	4	2.10 (.57-5.37)	2	2.84 (.34-10.27)	1	0.27 (.01-1.51)	3	3.61 (.75-10.56)	10	1.40 (.67-2.58)
• Intestine (152-153)	1	0.26 (.007-1.48)	1	0.63 (.02-3.53)	7	0.91 (.37-1.89)	0	---	9	0.60 (.27-1.14)
• Rectum (154)	0	---	0	---	3	1.36 (.28-4.00)	0	---	3	0.70 (.18-1.92)
• Liver, biliary, gallbladder (155.0, 155.1, 156)	0	---	0	---	5	3.93 (1.27-9.20)	1	3.27 (.08-18.17)	6	2.42 (.88-5.27)
• Liver, not specified (155.2)	0	---	0	---	0	---	1	7.17 (.18-39.88)	1	0.85 (.04-4.18)
• Pancreas (157)	2	0.93 (.11-3.38)	0	---	4	0.91 (.25-2.33)	1	0.93 (.02-5.20)	7	0.82 (.33-1.68)
• Respiratory (160-165)	19	1.33 (.80-2.08)	6	0.88 (.32-1.92)	20	0.66 (.40-1.02)	8	1.03 (.45-2.04)	53	0.90 (.67-1.17)
• Kidney (189.0-189.2)	1	1.01 (.01-5.64)	0	---	3	1.42 (.29-4.17)	0	---	4	0.98 (.31-2.36)
• Bladder (188, 189.3-189.9)	0	---	3	7.12 (1.47-20.84)	0	---	0	---	3	0.69 (.18-1.87)
• Lymphatic/hemato. (200-208)	4	1.10 (.30-2.83)	1	0.58 (.01-3.23)	10	1.26 (.60-2.32)	0	---	15	0.98 (.55-1.62)
• Other neoplasms	4	0.45	5	1.25	16	0.86	4	0.87	29	0.80

Table 6. Detailed Information on Liver Cancer Deaths at Plants 1 and 3.

Case	Plant	Coded UCOD (Revision)	Cause on Death Certificate	Work History	Latency
1	3	156.1 (9th)	Hepatobiliary CA	laborer-plantwide 1 year, 1952-1953	32 years
2	3	156.9 (9th)	biliary tract CA	Laborer, maintenance-plantwide 9 1/2 years, 1952-62 (In DBCP registry) **	30 years
3	3	156.0 (8th)	gallbladder CA*	operator, eng., laborer 17 1/2 years, 1951-68 (In DBCP registry) **	26 years
4	3	156.1 (8th)	cholangitis, bile duct CA*	operator-insecticide 1 year, 1951-52	19 years
5	3	155.0 (8th)	hepatoma, diffuse*	maintenance, construction 6 months	21 years
6	4	155.1 (7th)	gallbladder CA	laborer - 1.3 years powder feeder - 1 month	15 years
7	4	197.8 (8th)	Adenocarcinoma of liver	specific job unknown	20 years

* The cause of death for these cases were confirmed by hospital reports. Cases 3 and 4 were adenocarcinomas and case 5 was primary carcinoma of the liver.

** Included in the NIOSH registry of workers with potential exposure to DBCP.