FIRST NCI/EPA/NIOSH COLLABORATIVE WORKSHOP: PROGRESS ON JOINT ENVIRONMENTAL AND OCCUPATIONAL CANCER STUDIES

Tuesday Afternoon, May 6

EPIDEMIOLOGICAL/STATISTICAL SESSION (CONTINUED)

SESSION CHAIRPERSON

Dr. Joseph Fraumeni
National Cancer Institute

Mortality Study of Workers Employed at Organochlorine Pesticide Manufacturing Plants

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ABSTRACT

A retrospective cohort study was conducted to examine the mortality of workers employed in the manufacture of the chlorinated hydrocarbon pesticides, chlordane, heptachlor, DDT and aldrin/dieldrin/endrin. Four manufacturing plants were selected for study, and each cohort included all workers employed for at least 6 months prior to January, 1964. The entire study group totalled approximately 2100 individuals. Vital status ascertainment for these cohorts ranged from 90% to 97% complete, the cut-off date for follow-up was December 31, 1976.

In general, there are too few deaths in this study to make any meaningful conclusions. The standardized mortality ratio (SMR) for all causes in each cohort is below expected (100), ranging from 66 to 82, probably reflecting the "healthy worker effect". For "all malignant neoplasms", the SMR's range from 68 to 91 and for respiratory cancer from 55 to 132. In the aldrin/dieldrin/endrin cohort, pneumonia and "other respiratory diseases" were significantly above that expected. These causes of death need to be examined in more detail.

It is recommended that these cohorts be followed for several more years and the mortality patterns re-examined.

Introduction

The organochlorine (OC) pesticides such as DDT, Aldrin, Dieldrin, Lindane, Chlordane, Heptachlor, Toxaphene and Mirex have been an important class of compounds in terms of production volume and use. Due to the widespread use of these pesticides during the past 30 years, there has been an opportunity for widespread exposure to workers who manufacture, formulate and apply these compounds, and ultimately to those in the general population through ingestion of contaminated food and general environmental pollution.

There has been a great deal of concern about the long term, latent health effects from exposure to OC pesticides. These chemicals have a tendency to penetrate cell membranes and to be stored in the body fat.[1] Some of these chemicals have been shown to be toxic to the liver and kidney in exposed humans[2-3] and benign and malignant tumors in the liver have been induced in experimental animals chronically exposed to several of the OC compounds[4-9]. There are also reports of effects on the hematopoietic system among individuals exposed to Chlordane[10], DDT[11], Dieldrin[11] and Lindane[12-14].

In order to determine whether or not exposure to certain OC pesticides is associated with an increased risk of mortality due to chronic diseases an epidemiologic mortality study was initiated by the National Institute for Occupational Safety and Health (NIOSH). The

study was carried out under contract by the University of Illinois, School of Public Health. The contract stipulated that the study design be a retrospective cohort mortality study. The study cohort was defined as all workers at selected pesticide plants who were employed for at least six months, either continuously or intermittently between January 1, 1940 and December 31, 1964.

The original intent of this study was to examine the mortality of workers employed in OC pesticide formulating plants. However, after visits had been made to numerous formulating plants around the country it was determined that this part of the pesticide industry was not suitable for an epidemiologic study. The formulating plants are usually small operations, where work is seasonal, the turnover rate is high, exposures are multiple and records needed to conduct an epidemiologic study are not generally kept. Therefore, the emphasis of the study was shifted to OC pesticide manufacturing plants. These plants offerred a more suitable population for the investigation. However, the exposures at these plants are probably lower than at formulating plants and also include the chemical precursors of the final technical grade pesticide.

An attempt was made to include OC pesticide manufacturing plants in the U.S. which began operations at least 25-30 years ago, which had relatively large workforces and had records available to identify a study cohort. A list of the major OC pesticide manufacturing plants

was assembled from sources such as the Farm Chemical Handbook, trade commission reports, and EPA. Based on accummulated information, a number of potential facilities were contacted and walk through surveys were conducted to make the final selection for the study.

Four facilities were eventually chosen for the study. Table 1 gives a description of the four plants. Plant No. 1 is located in Illinois and has manufactured chlordane since 1946. Plant No. 2 located in Tennessee, has produced Heptachlor since 1951. Also, in 1953 Endrin was manufactured in a pilot operation at this plant and by 1955 full scale commercial production of Endrin began. Other products at plant No. 2 have included hydrogen gas, chlorine, and chlorendic anhydride. Plant No. 3 located in Colorado, has manufactured a variety of pesticides. In 1946 production of Aldrin and Dieldrin began and continued until the 1970's. Endrin production began in 1953 and continued until 1965. In 1955 this plant started manufacturing an organobromine pesticide and in 1956 the production of organophosphates was started. Plant No. 4 is located in California and DDT has been its sole product since 1947.

Methods

The study population consisted of four separate cohorts from the four pesticide plants. For purposes of future analysis some of these cohorts may be combined. However, in this presentation the results of

each cohort will be examined separately. The cohorts were defined as all workers at each plant who had achieved at least 6 months employment prior to December 31, 1964. This cutoff date was selected to allow for accrual of sufficient time or latency for manifestation of disease.

An effort was made to determine the vital status (alive or deceased) of each member in the study cohorts as of December 31, 1976. Vital status was ascertained through records maintained by federal and state agencies, including the Social Security Administration and state motor vehicle offices. For those individuals whose vital status could not be determined through those sources U.S. Postal Mail Correction Services and other follow-up searches were used. For all those known to be deceased, death certificates were obtained and causes of death were coded by a nosologist according to the International Classification of Diseases (ICDA) in effect at the time of death. Those who had an unknown vital status were assumed to be alive as of December 31, 1976 so that the true risk of mortality was not overestimated. Those who died after December 31, 1976 were considered alive for purposes of this analysis.

In each cohort, person-years at risk of dying were accumulated for each worker starting when six months of employment were completed and ending either at the date of death or at the study end date of 12/31/76 whichever occurred first. Using a modified life table

analysis program, similar to that described by Cutler[15], the person-years for each cohort were combined into five-year calendar and five-year age time periods and multiplied by the corresponding U.S. white male cause-specific mortality rate to yield the expected number of deaths. Person-years were additionally distributed by five-year exposure and five-year latency (number of years from date first employed) categories. The observed and corresponding expected deaths were compared and differences were tested using the Poisson Distribution.

Results

The results of the vital status ascertainment and the total number of person-years for each cohort are given in Table 2. Even with the efforts previously described several of the cohorts have an unknown vital status of 10 percent.

Table 3 summarizes the observed and expected deaths by specific cause. For the category of "all causes", the SMR's (observed deaths/expected deaths x 100) range from 66 to 86. Assuming the record systems used to identify the cohorts were complete, these low SMR's probably reflect the healthy worker effect which has been noted in other studies of occupational groups[16], and possibly the lack of complete vital status ascertainment. Mortality due to all malignant neoplasms was also lower than expected, with SMR's ranging from 68 to

91. Other major causes of death in the cohorts including diseases of the circulatory system are also generally lower than expected. The only major category where there was a significant increase in observed deaths over expected was for pneumonia (11 observed vs. 4.3 expected, SMR = 255, $p<0.01$) and for "other respiratory diseases" (11 observed vs. 5.2 expected deaths, SMR = 213, $p<0.05$), at plant 3.

Table 4 summarizes observed and expected deaths by specific type of cancer. As stated previously, there is a deficit in observed deaths due to "all malignant neoplasms" in each plant studied. Although there are no statistically significant excesses or deficits in mortality among any specific cancer site, several sites are of note. In plant 1 there are 3 observed deaths due to stomach cancer when 0.99 were expected. In plant 3 there are slight excesses in cancer of the esophagus (2 observed vs. 0.85 expected), cancer of the rectum (3 observed vs. 1.24 expected), liver cancer (2 observed vs. 0.57 expected), and cancer of the lymphatic and hematopoietic system (6 obs. vs. 4.09 exp.). In addition there is a deficit in respiratory cancer (7 observed vs. 12.64 expected).

An analysis by latency is presented in Table 5 for "all malignant neoplasms". In this type of analysis, one looks for trends to examine whether or not the risk of mortality as measured by the SMR is associated with any particular latency period. Plants 2 and 4 show a consistent increase in the risk of cancer mortality with an increase

in the latency period, however the numbers involved in this analysis are small. Since respiratory disease was increased in plant 3, latency was also examined for this cause of death. There was a statistically significant increase in mortality due to respiratory disease during the 10-20 year latency period (12 obs. vs. 4.51 exp.; $p<0.05$), which decreased during the greater than 20 years latency period (8 obs. vs. 3.96 exp.; $0.05<p<0.1$).

Discussion

Plants 1 and 2 have been previously studied by Wang[17]. In her study, the definition of the cohort included workers from both plants who were employed for at least 3 months between 1946 and 1975. Therefore, although there is overlap between this cohort and plants 1 and 2 included in the present study, the cohorts are not exactly alike. In Wang's study, there was no observed excess in mortality due to specific cancer sites except for a small increase in respiratory cancer. The only cause of death where there was a significant excess was for cerebrovascular disease. Neither of these findings were seen in the present study - cerebrovascular disease in plants 1 and 2 combined was 8 obs. vs. 7.88 exp. and respiratory cancer was 9 obs. vs. 7.87 exp. As noted previously there was an increase in stomach cancer observed in plant 1, however, the small numbers involved in this study preclude any clear association.

In the plant 3 cohort there was a significant increase in deaths due to non-malignant respiratory disease especially among those with at least 10 years of latency. In contrast to this finding there was a deficit in deaths due to respiratory cancer. These findings need to be examined further to determine whether there is a true association between respiratory disease and employment at this plant.

There was no excess in cause specific deaths in the plant 4 cohort. However, when the deaths from malignant neoplasms are examined by latency, there is an increase in risk with an increase in the length of the latent period. The numbers in this analysis are small and this trend could be due to chance alone.

Due to the small number of workers included in this study the statistical power does not enable one to conclude that there is no association between cause-specific mortality and employment at the study plants. The primary reason for these small numbers is due to the rapid turnover at these plants, and thus most workers who were hired left before they achieved 6 months of employment. This was especially true at plant 4 where approximately seventy percent of the employees worked less than 6 months.

Although this study has not identified a specific cancer risk associated with employment at certain types of OC pesticide manufacturers, it points to several causes (stomach cancer in plant 1,

esophagus, rectum, liver and lymphatic/hematopoietic cancer in plant 3) that should be examined further. An attempt should be made to determine if there are any common exposures among those who died from these causes of death. Additional analyses are also necessary to determine, if possible, whether or not the excess in respiratory disease is associated with specific occupational exposure at plant 3. Finally, the mortality experience in each of these cohorts should be followed for several more years with a better ascertainment of vital status to increase the statistical power of the analysis so that more confident conclusions can be made.

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Table 1

Description of Plants Included in the Study of Organochlorine Pesticide Manufacturers

	PLANT 1	PLANT 2	PLANT 3	PLANT 4
Date began OC pesticide production	1946	1951	1946	1947
OC pesticides produced	- Chlordane	- Heptachlor - Endrin	- Aldrin - Dieldrin - Endrin	- DDT
Other pesticides produced	none	none	- organobromines - organophosphates	none
Other chemicals at plant	- Chlorine - Dicyclopentadiene	- Chlorine - Chlorendic anhydride Hexachlorocyclopentadiene - vinyl chloride	- Numerous precursors	- tri-chloroacetaldehyde - sulfuric acid -Monochlorobenzene
Location	Illinois	Tennessee	Colorado	California

Table 2

Vital Status Follow-up of Workers in Study of Organochlorine Pesticide Manufacturers

	PLANT 1	PLANT 2	PLANT 3	PLANT 4
Known to be alive	259 (79%)	265 (87%)	870 (75%)	278 (79%)
Known to be deceased	59 (18%)	24 (8%)	173 (15%)	42 (11%)
Unknown vital status	9 (3%)	16 (5%)	112 (10%)	34 (10%)
Total	327	305	1,155	354
Person-years of observation	8,354	5,672	24,939	7,601

Table 3

Observed/Expected Deaths According to Major Cause Among Workers in Study of Organochlorine Pesticide Manufacturers

CAUSE OF DEATH (7th Revision ICD No.)	PLANT 1	PLANT 2	PLANT 3	PLANT 4
All Malignant Neoplasms (140-205)	11/15.89 (69)[1]	6/ 6.60 (91)	31/ 37.79 (82)	6/ 8.86 (68)
Nervous System Diseases (330-334)	7/ 6.03 (116)	1/ 1.85 (54)	9/ 13.32 (68)	-
Circulatory System Disease (400-468)	28/40.25 (70)	12/15.35 (78)	69/ 90.17 (77)	17/20.61 (82)
Non-malignant Respiratory System Disease (470-527)	1/ 4.58 (22)		22/ 10.40 (212)*	1/ 2.28 (44)
Accidents (800-962)	6/ 6.28 (96)	1/ 3.96 (25)	11/ 18.45 (60)	4/ 5.46 (73)
Suicide (963, 970-979)	1/ 2.12 (47)	-	10/ 5.99 (167)	-
All Other Causes	5/12.01 (42)	4/ 8.57 (47)	21/ 29.74 (71)	14/11.79 (119)
All Causes	59/87.16 (68)	24/36.33 (66)	173/205.86 (84)	42/49.00 (86)

1 $SMR = \frac{\text{Observed Deaths}}{\text{Expected Deaths}} \times 100.$

* $p<0.01$

Blanks represent no observed deaths.

Table 4

Observed/Expected Deaths According to Specific Cancer Causes
Among Workers in Organochlorine Pesticide Manufacturing

CAUSE OF DEATH (Seventh Revision ICD No.)	PLANT 1	PLANT 2	PLANT 3	PLANT 4
All Malignant Neoplasms (140–205)	11/15.89 (69)[1]	6/6.60 (91)	31/37.79 (82)	6/8.86 (68)
Esophagus (150)	-	-	2/ 0.85 (235)	-
Stomach (152,153)	3/ 0.99 (303)	-	1/ 2.09 (48)	1/0.44 (227)
Intestine (152,153)	-	1/0.57 (175)	1/ 3.35 (30)	-
Rectum (154)	1/ 0.56 (178)	-	3/ 1.24 (242)	-
Liver (155,156A)	-	-	2/ 0.89 (225)	-
Pancreas (157)	1/ 0.91 (110)	-	1/ 2.10 (48)	1/0.49 (204)
Respiratory (160–164)	6/ 5.43 (110)	3/2.45 (122)	7/12.64 (55)	4/3.19 (125)
Bladder and Urinary (180–181)	-	1/0.15 (666)	1/ 1.10 (91)	-
Other and Unspecified (156B,165,190–199)	-	1/0.88 (114)	6/ 4.80 (125)	-
Lymphatic and Hematopoietic (200–205)	-	-	6/ 4.09 (147)	-
Others	-	-	1/-	-

1 $SMR = \frac{\text{Observed Deaths}}{\text{Expected Deaths}} \times 100.$

Blanks represent no observed deaths.

Table 5

Observed and Expected Deaths Due to Malignant Neoplasms According to Latency[1] Among Workers in Study of Organochlorine Pesticide Manufacturers

Years Since First Employed		Observed	Expected	SMR
PLANT 1	< 10 yrs.	1	1.52	66
	10 - < 20 yrs.	4	4.43	90
	> 20 yrs.	6	9.94	60
PLANT 2	< 10 yrs.	0	1.46	-
	10 - < 20 yrs.	3	3.30	91
	> 20 yrs.	3	1.85	162
PLANT 3	< 10 yrs.	4	7.55	53
	10 - < 20 yrs.	18	16.25	111
	> 20 yrs.	9	14.00	64
PLANT 4	< 10 yrs.	0	1.40	-
	10 - < 20 yrs.	1	3.68	27
	> 20 yrs.	5	3.78	132

[1] Latency = number of years from date first employed.

Table 6

Observed and Expected Deaths from Respiratory Disease According to Latency Among Workers at Plant 3 in Study of Organochlorine Pesticide Manufacturers

Years Since First Employed	Observed	Expected	SMR
< 10 yrs.	2	1.07	187
10 - < 20 yrs.	12	4.51	266*
> 20 yrs.	8	3.96	202

* $p<0.05$

Dr. Kraybill (NCI): This paper interested me very much, because of the selection of the chemicals, these pesticides, particularly DDT. We have been waiting for many years for the story about DDT. Correct me if I am wrong, but we have no human data yet to show that DDT or DDE has been carcinogenic in man. People opine that maybe if you did, then formulators, since they are getting a good exposure, probably more so than in the industrial plant, could show a causation. I am wondering if you could combine a population. Is that plant still producing DDT in California?

Mr. Brown (NIOSH): It is.

Dr. Kraybill (NCI): Are there formulators for DDT?

Mr. Brown (NIOSH): I am sure there are. Most of the formulators formulate thousands of different chemicals depending on the season and on the day.

Dr. Kraybill (NCI): Well, I am not an epidemiologist and that is why I am asking stupid questions. If you could combine them, maybe the numbers would be sufficient to draw a conclusion. But if you could do this on DDT and put that issue to bed once and for all, that would be a great contribution.

Mr. Brown (NIOSH): One thing that we may do is look at these plants and have a shorter cut-off period for length of employment and we would get more people in the study that way. I do not know if the short-term employment people are as important though.

Dr. Keefer (NCI): I was wondering also about the agricultural workers themselves who might be using these chemicals. I do not suppose you would have any better luck with them, but I wanted to ask.

I also wondered about other types of pesticides. I do not know about the usage patterns at all, but some of the dinitrobenzene types of pesticides and herbicides have been shown to contain relatively large amounts of nitrosamine contaminants. These are several orders of magnitude higher concentrations in the commodity than some of the things that we worry about like beer and scotch. I was wondering whether it would be possible to follow the experience of people with those kinds of pesticides as opposed to the chlorinated hydrocarbons or in addition to the chorinated hydrocarbons.

Mr. Brown (NIOSH): On your first question, agricultural workers are a very difficult group to follow for epidemiological work. They are migratory. There are usually no records of these people or who they are. Many of them are illegally in this country. It is a very difficult study to accomplish. Plus, it is out of NIOSH's purview; I think it is EPA's.

As far as doing work on other compounds, I think NIOSH is looking at other pesticides. In fact, they are looking at some other formulators. I am not sure if they are concentrating on looking at nitrosamines. I think John, who presented a paper on nitrosamines before, may be looking at some of those in his survey. As far as looking at the mortality outcomes, I do not think there are any plans to do that right now.

Dr. Caldwell: Is there any reason that you can think of as to why the people leave in such a short period of time? Did any of the health hazard walk-throughs maybe indicate that people are leaving in six months there because they are developing acute illness or acute hypersensitivity, so that there is a reason why they are gone.

Mr. Brown (NIOSH): I do not really know. A lot of this is seasonal work and a lot of people come in, especially in the DDT plant in Los Angeles, for the summertime and they produce tremendous quantities of DDT and then there is a slowdown and so they leave. A lot of people are there for summer jobs.

The other thing about studying other pesticides is we are trying to look at people who were employed by the government in some of these pest eradication programs. A lot of those people used DDT years back, such as in the fringe beetle eradication program and some other things through the Agriculture Department.

Dr. Galbraith (EPA): Was there any difference in the inert ingredients in the pesticide formulations in the four different plants?

Mr. Brown (NIOSH): Well, since these were not formulators, these plants only made the technical grade product. So there was not any addition of inert ingredients at these plants. They sold these concentrated technical grades to a formulator and the formulators are the ones who add the inert ingredients and mix them up.

Dr. Fraumeni (NCI): Thank you very much.

National Cancer Institute

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

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

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