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INTERNAL CORRESPONDENCE

CHEMICALS AND PLASTICS

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To (Name) Mr. J. B. Hollingsworth
Division Dr. T. T. Szabo
Location Mr. H. R. Guest
Mr. J. L. Carvajal
Dr. E. Q. Hull

Date April 30, 1976

Originating Dept.

Answering letter date

Copy to

Subject MCA Vinyl Chloride Technical
Panel Meeting April 28, 1976

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The MCA Vinyl Chloride Technical Panel met April 28 to review the status of the various research programs. Some brief notes on each program are as follows:

1. Teratology was discussed by Dr. B. A. Schwetz. The study involved exposure of rats, mice and rabbits to 50 ppm, 500 ppm and 2,500 ppm vinyl chloride concentrations in the early days of pregnancy. Excess maternal deaths were observed at the 2,500 ppm exposure. This was enhanced by ethanol in the drinking water. There appeared to be a slight teratogenic effect in mice though results were not statistically significant.

Dr. Schwetz stated that based on present-day exposures and the results of the study that teratogenesis should be of no concern to pregnant women and women of child bearing age. He noted that since the first 15 to 35 days of human pregnancy were most critical with regard to teratogenesis, it would not be possible to identify pregnant women and control their exposure.

2. Metabolism was discussed by Dr. P. G. Watanabe. The study confirmed the existence of detoxification in the liver of exposed animals. Vinyl chloride is not a carcinogen but its metabolite chloroacetaldehyde is carcinogenic. During detoxification it reacts with glutathione in the liver and is excreted as thiodiglycolic acid, or one of two conjugated cysteines. Based on the work to date, Dr. Watanabe believes the fate of ingested vinyl chloride is dose dependent. High vinyl chloride concentrations overcome the body's detoxification mechanism resulting in tissue damage (neoplasms). The Dow work integrated with the inhalation studies shows that 50 ppm is a threshold concentration in rats.

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3. Animal studies were discussed by Dr. M. L. Keplinger. There have been no basic changes in the conclusions from the Industrial Biotest work. Vinyl chloride is clearly carcinogenetic to three rodent species and is dose related.
4. Mutagenetic work was reviewed by Dr. R. V. Johnston. The cytogenetic study of rats taken from Industrial Biotest showed no significant changes in cell structure as a result of vinyl chloride exposure. Literature on mutagenesis was listed showing vinyl chloride to be a mutagen to bacteria. Three small population studies of humans showed mutagenesis from vinyl chloride. An extensive study by ICI on humans showed no mutagenesis.
5. Epidemiology was discussed by Dr. W. R. Gaffey. The "Tabershaw-Cooper Associates" (now Equitable Health Sciences) study has been expanded to a population of 10,173 workers with 707 deaths (95% followup) covering the period 1942 to 1972. In the long exposure, high concentration group excess deaths were noted from digestive, respiratory and "other" cancers. The "other" category was distorted by brain cancers. A draft of the final report will be available in two to three weeks.

The Infante birth defects studies generated much discussion but no practical results. The Painesville, Ohio study is discredited by C.D.C. The Pottstown, Pennsylvania study via questionnaires is poorly regarded though there appears to be no way to counter it. The Kanawha Valley study protocol is attached.

The proposed research program at Dow and the University of Louisville was discussed and endorsed. (This is shown in a separate letter.)



R. N. Wheeler, Jr.

RNWJr. /pm
Attachments

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CENTER FOR DISEASE CONTROL

Morbidity and Mortality



Vol. 25, No. 8

WEEKLY
REPORT

For
Week Ending
February 28, 1976

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE PUBLIC HEALTH SERVICE
DATE OF RELEASE: MARCH 5, 1976 - ATLANTA, GEORGIA 30333

EPIDEMIOLOGIC NOTES AND REPORTS ANGIOSARCOMA OF THE LIVER - Wisconsin

In the period June 1973-November 1975, 4 cases of angiosarcoma of the liver (ASL) were diagnosed at the Marshfield Clinic, Marshfield, Wisconsin. One was based on hepatic arteriography and 3 on tissue findings later confirmed by pathologic review at the National Cancer Institute (NCI).

An additional 6 cases, confirmed on pathologic review at NCI and identified through a national ASL case surveillance project initiated at CDC in 1974, have been diagnosed since 1964 in Wisconsin residents. This total of 10 cases is roughly twice as large as might be expected (5.5 cases), based on CDC surveillance estimates of crude ASL incidence for the entire United States. (Five additional cases had been re-

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ported for Wisconsin; 4 were not confirmed, and 1 was in a resident of another state.)

In view of this relative frequency of cases, together with the recent succession of cases at the Marshfield Clinic, epidemiologic information concerning each of these 10 cases

TABLE I. CASES OF SPECIFIED NOTIFIABLE DISEASES: UNITED STATES
(Cumulative totals include revised and delayed reports through previous weeks)

DISEASE	WEEK ENDING		MEDIAN 1971-1975	CUMULATIVE, FIRST 8 WEEKS		
	February 28 1976	February 22 1975		February 28 1976	February 22 1975	MEDIAN 1971-1975
Aseptic meningitis	28	35	33	307	292	292
Brucellosis	7	2	2	38	21	15
Chickenpox	5,764	4,311	---	39,288	29,653	---
Diphtheria	4	14	6	62	64	23
Encephalitis	13	17	15	129	96	120
Primary	4	7	5	32	33	33
Post-infectious	242	210	172	1,935	1,568	1,314
Hepatitis, Viral	726	707	890	5,409	5,428	7,619
Type A	133	150	---	1,365	1,123	---
Type unspecified	3	6	6	43	42	42
Malaria	805	578	622	4,318	2,668	4,767
Measles (rubella)	53	41	41	284	270	270
Meningococcal infections, total	53	41	40	281	263	263
Civilian	---	---	1	3	7	9
Military	1,366	1,425	1,892	9,366	11,136	14,246
Mumps	22	24	---	194	201	---
Pertussis	449	285	645	1,870	1,437	3,534
Rubella (German measles)	2	---	---	6	9	8
Tetanus	570	505	---	4,572	4,105	---
Tuberculosis	1	1	1	21	8	13
Tularemia	3	2	4	58	29	37
Typhoid fever	---	---	---	3	10	9
Typhus, tick-borne (Rky. Mt. spotted fever)	---	---	---	---	---	---
Veneral Diseases:						
Gonorrhea	18,313	17,273	---	151,748	141,258	---
Civilian	562	419	---	4,708	4,776	---
Military	528	531	---	4,132	3,938	---
Syphilis, primary and secondary	8	3	---	66	52	---
Civilian	39	45	56	249	284	448
Military	---	---	---	---	---	---

TABLE II. NOTIFIABLE DISEASES OF LOW FREQUENCY

	Cum.		Cum.
Anthrax	2	Poliomyelitis, total:	2
Botulism	4	Paralytic: N.Y.C. 1	2
Congenital rubella syndrome	5	Pittacosis: Ohio 1, Calif. 3	19
Leprosy: Pa. 1, Calif. 2	20	Rabies in man:	---
Leptospirosis	7	Trichinosis: N.J. 1	33
Plague	---	Typhus, murine: Tex. 1	2

1)Delayed Report for week ending 2/21/76

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ANGIOSARCOMA - Continued

has been assembled through medical record review and family interview (Table 1). Particular attention has been given to potential occupational or environmental exposure to chemicals known to induce ASL (vinyl chloride, arsenic, and thorium dioxide).

Most cases (7 of 10) were diagnosed since 1970, probably reflecting better case ascertainment in recent years. Ages ranged from 36-71 (mean age 58 years). All but 1 patient were male, and all but 1 were white. Places of usual residence for all cases were widely scattered across the state, roughly corresponding with the state's population (Figure 1).

One patient had a history of prior thorium dioxide exposure. For none of the remaining cases was tumor etiology obvious. Occupations and histories of exposure to potential environmental toxins differed widely, with no more than 1 case associated with any particular industry or mode of exposure. In several instances, however, individual patient occupations or exposures were suggestive of possible oncogenicity (Table 1). Patient 10 had had exposure to a vinyl compound; he had worked in paper and plywood products with potential contact with various chemicals, including polyvinyl acetate. Two other patients gave histories of possible exposure to arsenic: patient 9, who had exposure to insecticides while farming, and patient 8, who worked with wood probably treated with arsenic preservatives in the manufacture of bleachers. All but 3 of the 10 patients were known to have lived on farms during their lifetimes, 2 for all of their lives (Table 1).

(Reported by J Fiechter, MD, C Reyes, MD, Marshfield Clinic; K Rentmesster, MPH, HG Skinner, MD, State Epidemiologist, Wisconsin State Dept of Health and Social Services; Office of Extramural Coordination and Special Projects, National Institute of Safety and Health; and the Cancer and Birth Defects Div, Bur of Epidemiology, CDC.)

Editorial Note

These data indicate no obvious common-source origins among recent cases of ASL in Wisconsin. The marked male preponderance among cases, however, suggests occupational

Figure 1
GEOGRAPHIC DISTRIBUTION OF CONFIRMED CASES OF
ANGIOSARCOMA OF THE LIVER IN WISCONSIN RESIDENTS,
1964-1975, BY PLACE OF USUAL RESIDENCE



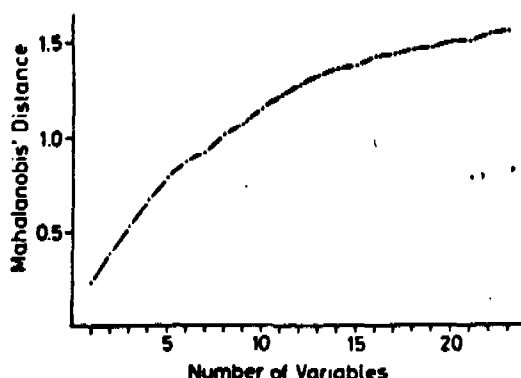
exposure as the potential origin for at least some of the cases. Anecdotal histories of occupational and environmental exposures in several of the cases may warrant further investigation, particularly in relation to contact with arsenic. Although at present only vinyl chloride monomer (1), thorium dioxide (2), and arsenic (3,4) are clearly implicated as causes of ASL, investigations such as the one described here may well provide useful leads in defining further causes.

References

1. Crech JL, Johnson MN: Angiosarcoma of liver in the manufacture of polyvinyl chloride. *J Occup Med* 16:150-151, 1974
2. MacMahon HE, Murphy AS, Bates MI: Endothelial-cell sarcoma of liver following Thorotrast injection. *Am J Pathol* 23:585-595, 1947
3. Lender JJ, Stanly RJ, Summer HW, et al: Angiosarcoma of the liver associated with Fowler's solution (potassium arsenite). *Gastroenterology* 68:1582-1586, 1975
4. Roth F: The sequela of chronic arsenic poisoning in Moselle vinters. *Ger Med Mon* 2:172-175, 1957

Table 1
Cases of ASL in Wisconsin Residents 1964-1975

Case Number	Month and Year Diagnosis	Month and Year Death	Usual Town Of Residence	Age at Diagnosis	Race	Sex	Occupation	Comments	Farm Residence
1	4/64	2/65	Sheybohan	71	W	M	University Teacher	Lived near a chemical company which made plastics and resin	Youth
2	2/67	2/67	Oshkosh	61	W	M	Foundry Welder	Exposure to welding materials and resins	None
3	6/67	6/67	Necedah	36	W	F	Secretary	Infectious hepatitis in childhood, mother and sister had liver disease	Youth
4	7/71	7/71	Milwaukee	68	B	M	Hog Scolder		None
5	4/72	4/72	Milwaukee	64	W	M	Unknown	Exposure to thorium dioxide	Unknown
6	4/74	6/74	Brown Deer	46	W	M	Lutheran Pastor	Lived near a paper mill, 1955-1959	Youth
7	7/74	7/74	Wausau	57	W	M	Rock Crusher; Glue-Liner for rubber raincoats, 1940s	Exposure to silica dust, rubber cement	Youth
8	9/74	9/74	Waupun	46	W	M	Postal Mail Carrier formerly employed by company that made bleachers	Contact with lumber, paints, wood preservatives, (ammoniacal-copper-arsenate wood preservative) (?)	Lifetime
9	8/74	8/74	Stratford	66	W	M	Retired Farmer	No tissue diagnosis; ASL diagnosed by hepatic arteriography	Lifetime
10	11/75	11/75	Marshfield	65	W	M	Press operator for paper and plywood company (23 years)	Contact with wood, plywood, glues, polyvinyl acetate, others	Youth



the discrimination of vector of means can be tested for significance.

A high discrimination between the malformation group and its matched controls appeared within the first 10 of the 24 variables (see accompanying figure). Even with 8 variables a significant discrimination of both groups at 1% level was found. The discrimination of both groups was also possible without the two factors "abortions/stillbirths in mothers family" and "hours of daily work".

So, even in a sample with factors each not discriminating significantly between both groups, the linear discriminance analysis makes it possible to discriminate between the malformation group and the matched controls. This might mean that the malformations in our sample can be explained as caused by the combination of many minor factors (extrinsic and intrinsic).

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J. KLEINERFUCHT
K.-H. DEGENHARDT

CONGENITAL MALFORMATIONS AND VINYL CHLORIDE

Sia.—In March, 1975, a significantly increased rate of central-nervous-system (C.N.S.) malformations was reported in 3 Ohio communities where polyvinyl chloride (P.V.C.) polymerisation plants are located;¹ the increase was not uniform and appeared more prominent in the Painesville area. There are two P.V.C. plants in Painesville, one of which began operation in 1949 and the other in 1967. The birth-defect rates were computed from birth-certificate data for 1970-73 and were compared with similar data for the entire State of Ohio. This study suggested a relationship between the higher rates of C.N.S. malformations and the increased risk of exposure to vinyl chloride in these communities.

By analysing data collected through the Center for Disease Control's hospital-based birth defects monitoring programme (S.D.M.P.),² we examined this relationship. Two hospitals participating in the S.D.M.P. are located in cities with polymerisation plants: one is in Pennsylvania, the other in Painesville, Ohio. C.N.S.-malformation rates for white infants born in these two hospitals in 1970-74 were compared with the rates for white infants in each State. No increase was seen in the Pennsylvania hospital, but an increase, primarily in anencephaly and spina bifida, was noted in the Painesville hospital for the total time 1970-74 (see table).

ANENCEPHALUS AND SPINA BIFIDA IN OHIO STATE AND IN PAINESVILLE HOSPITAL, OHIO

Year	Anencephalus				Spina Bifida			
	Painesville		State		Painesville		State	
	No. of cases	Rate*	No. of cases	Rate	No. of cases	Rate	No. of cases	Rate
1970	0	0.0	26	5.0	3	31.2	58	11.1
1971	1	10.3	30	7.3	3	30.9	44	8.5
1972	3	35.6	31	6.3	0	0.0	47	9.6
1973	2	24.9	27	5.4	2	24.9	46	9.2
1974	0	0.0	22	4.5	1	15.1	29	6.0
1970-1974	6	14.1	144	5.7	9	21.2	224	8.9

* Per 10 000 total white births.

The S.D.M.P. hospital data for Painesville were compared with birth-certificate data from the Ohio department of health, and 1 additional case not included in the S.D.M.P. report was added, bringing the total to 15. 30 controls, the first normal white infant born before and after each case, were selected from the hospital birth registry. Medical records for cases and controls were reviewed to confirm the diagnosis and to obtain, among other data, the parents' occupation and residence at the time of the infant's birth. In addition, parents of 14 of the affected children were interviewed about previous occupations and residences.

The study revealed that none of the interviewed parents of affected infants had ever worked at either of the two P.V.C.-polymerisation plants in Painesville (1 set of parents could not be located, but records show that they did not work at these plants at the time of their infant's birth); 2 of the fathers of controls had worked at one of the plants at the time of their infants' births. None of the parents in either group lived within 2 miles of the two plants. The chi-square test of significance was used in comparing the distances from residence and work-place of mothers and fathers to the P.V.C. plants for cases and controls. Comparisons were made at distances of 1 to 10 miles, in 1-mile increments. A significantly larger proportion of control mothers than case mothers worked (including housewives) within a 10-mile radius of the P.V.C. plant, at the 95% confidence level. This is probably a chance occurrence among multiple comparisons.

Vinyl chloride is a known carcinogen,³ and concern has arisen over its possible teratogenic effects. This concern stems from recent studies which have shown (a) an increase in mutagenicity in bacterial assay systems⁴ and (b) increased breakage in chromosomes of P.V.C.-polymerisation workers.⁵ The Ohio Department of Health study¹ is the only report that has suggested that vinyl chloride causes birth defects in man. Although the follow-up study reported here confirmed a moderate increase in C.N.S. malformations in Painesville, Ohio, no association was found with vinyl-chloride exposure.

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LARRY D. EDMONDS,
HENRY FALK
JACK E. NISSIM

LATE COMPLICATIONS OF TUBAL LIGATION

Sia.—Unlike Mr Darwish and Dr Saafan (Nov. 15, p. 975), we are uncertain as to the etiology of the late sequelae of female sterilisation, which they refer to as menstrual irregularity, but in our study it will be noted we looked specifically at menorrhagia and dysmenorrhoea. It seems unlikely that a psychosomatic cause could be present when two different tech-

1. Infante, P. in Proceedings of the Conference on Occupational Carcinogenesis at the New York Academy of Science, 25 March, 1974. New York, in the press.

2. Center for Disease Control. Congenital Malformations Surveillance Annual Summary 1974. Atlanta, 1975.

3. Center for Disease Control. Morbidity and Mortality Weekly Report, Feb. 9, 1974, p. 49.

4. Rasmussen, L., Johansson, A., Ramel, C., Wachsmann, C. A. *Am J Hyg*, 1974, 3, 194.

5. Ponder-Kuzman, F., Lombert, B., Lindsten, J. *Lancet*, 1975, i, 459.



DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE
CENTER FOR DISEASE CONTROL
1600 CLAYTON ROAD, NE
ATLANTA, GEORGIA 30333

March 25, 1976

MAR 29 1976

Mr. Milton Freifeld
Manufacturer's Chemist Association
1825 Connecticut Avenue
Washington, D. C. 20009

Dear Mr. Freifeld:

Enclosed is a copy of the protocol for the study presently underway in Charleston, West Virginia. I have made a few marginal notes where changes were made. The main change was the decision from the outset to interview cases and controls (regardless of exposures among the case group). We had at one time considered searching occupational records at the plant for names of cases and controls, but discarded the idea in favor of the interviews.

If you have questions about this, do not hesitate to contact me. I would like to emphasize our desire to maintain the confidentiality of hospitals that participate in the BDMP. This includes, according to our agreement with the Commission on Professional and Hospital Activities, no mention of the number of hospitals in a community which participate in BDMP. We would appreciate your cooperation in this respect. Also note that the data contained in this protocol are preliminary and were compiled only as background for planning of the study. Final data will be part of the finished report.

Sincerely yours,

J. William Flynt, Jr., M.D.
Chief, Birth Defects Branch
Bureau of Epidemiology

Enclosure

cc: Dr. John Finklea
Mr. Larry Edmonds

JWF:crs

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Introduction

The continuing concern about a causal association prompted a review of all 36 counties with VCP plants to determine those with BDMP hospitals. A similar determination had been done previously selecting for analysis those communities with a VCP plant and most likely served by a single hospital also in the BDMP (and having a VCP plant). Painesville, Ohio, and Pottstown, Pennsylvania, were identified in this manner. Pottstown had no excess of malformations while Painesville was noticed to have a twofold increase in CNS defects. On the second review, a total of 17 counties with VCP plants also have at least 1 BDMP hospital. Four of the 17 counties have a significantly higher number of CNS defects than expected, based on a total U.S. rate 1970-73. In two counties BDMP monitors less than 50 percent of the total reported births (Table 1). The 2 remaining counties are Lake Co., Ohio (Painesville),

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the site of previous investigation, and Kanawha County (Charleston), West Virginia. There are 4 hospitals in Kanawha County reporting 4,476 births in 1973 (Table 2). The 2 largest hospitals are enrolled in the BDMP; 1 is located in Charleston and the other in South Charleston, the site of the VCP plant. The current BDMP data show for Kanawha County a significantly higher number of CNS defects than expected (59 observed vs. 37 expected). The expected number of cases are based on the total U.S. rates for 1970-74 (Table 3).

In light of continuing concern regarding the possible teratogenicity of VC monomer; the Kanawha County setting was selected for a second investigation concerning the possible association of increased rates of CNS defects with parental occupation or residential exposure to the S. Charleston VCP plant.

Additional background data on Kanawha County vital statistics are provided in the appendix.

Objectives

The objective of this study will be to further investigate the possible relationship of parental exposure to vinyl chloride and the increased incidence of central nervous system defects. The case-control study method will be used to compare the frequency of maternal and paternal occupational exposure to VCP. Similarly, residential exposure will be examined by comparing distances of parental residences at birth from the VCP plant in South Charleston.

3

Methods

A. Case Ascertainment

All cases of Central Nervous System (ICDA NO. 740.0-743.9) defects that occurred among resident live births and fetal deaths in Kanawha County between January 1, 1970 and December 31, 1974 will be identified. Two sources of case identifications will be employed:

1. Birth and fetal death certificates will be reviewed in the West Virginia Department of Vital Statistics.
2. Records at all Kanawha County hospitals will be reviewed. This will include cases reported through the Birth Defects Monitoring Program. A consolidated list of unduplicated cases will be compiled and diagnosis verified according to hospital records. Non-residential and unverified cases will be retained for further comparative analysis, but not matched with controls.

B. Selection of Controls

Controls will be selected from Kanawha County resident birth certificates after all cases have been identified. Two controls will be selected for each case of CNS defects. The controls selected will be the next matched normal live birth preceding and following. The cases and controls will be matched for maternal age (15-19, 20-24, etc.), paternal education (0-8, 9-12, 13 over), and race. A maximum of twenty-five birth certificates will be reviewed preceding and following the case to find the two matching controls. If a preceding (following) age-education-race matched control is not found, then the first preceding (following) birth matched on race

and either education or age will be chosen, education being the first choice. If there is still no-match, then the first preceding (following) birth matched only on race will be selected.

Data Collection

A. Birth Certificates and Fetal Death Certificates

The information to be collected on case and controls from the birth certificates is contained on page 1 of the work sheet.

B. Hospital Record

The information to be collected on cases from hospital charts is listed on page 2 of the work sheet. Hospital records will be reviewed for controls only in cases where birth certificates information is incomplete.

C. Interview

Parents of cases will be interviewed in standard fashion regarding prior residences and occupations. The information to be obtained is listed on Worksheets 3-5.

Parents of controls will not be interviewed unless 1 or more parents of cases are found to be employed in the vinyl chloride plant or a significantly greater proportion of the parents of cases live near the vinyl chloride plant. In the event that 1 or more parents of cases works at vinyl chloride plant, only 1 control will be interviewed for each matching case. The control chosen for interview will be the closest match.

*Parents of controls
will not be interviewed
unless 1 or more parents
of cases are found to be
employed in the vinyl
chloride plant or a
significantly greater
proportion of the
parents of cases live
near the vinyl chloride
plant.*

*Control for each
case - matched
prior to interview*

Data Analysis

An analysis of matched triptes will be made using the method of Mantel and Haenszel.

The possible relationship between parents' occupational exposure and CNS defects in their children will be analyzed 3 ways by dichotomizing cases and controls on employment of mother, father, and either parent in a vinyl chloride plant. Only current employment will be used for controls unless significance is attained. Then past employment data will be obtained from the controls in the same manner as for cases and the resulting data re-analyzed.

The effect of distance of residence from the vinyl chloride plant will be analyzed by dichotomizing on distance in increments of 1 mile in the range 1-10 miles.

Table I

Counties with Significant Increases in CNS Defects 1970-73
and Reported Births in 1973

	# CNS Defects in BDMP		# Births In BDMP	#Births In AHA*	Z: Monitored in BDMP
	obs.	exp.			
Oklahoma Co., Oklahoma	68	44.9	4,096	8,718 (9 hospitals)	47.0
New Castle Co. Delaware	9	4	366	5,634 (3 hospitals)	6.5
Lake Co. Ohio	31	18	1,821	1,851 (2 hospitals)	98.4
Kanawha Co. West Virginia	53	31	4,245	4,476 (4 hospitals)	94.8

*American Hospital Association Guide to the health care field (1974 edition)

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Table II

Reported Hospital Births in Kanawha County, W. Virginia from AHA Guide

Charleston.	3488
"	148
"	93
South Charleston	757
Total birth Kanawha County	4476

*BDMP Hospital

95% of reported hospital births are monitored by BDMP

Vital Statistics Data from West Virginia Vital Statistics Reports
1974 for Kanawha County

Total Births	3348
Hospital Births	3341
White Births (male)	1616
(female)	1471
Non-white Births (male)	126
(female)	135

Table III

Total Central Nervous System Defects - 1970-1974

	OBS.	EXP.*	RATE+
	43	29.2	31.3
	16	8.2	41.3
Kanawha County	59	37.4	33.5
Total U.S.	7655		21.2

Relative risk = 1.58

Anencephalus-Spina Bifida - 1970-1974

	OBS.	EXP.	RATE
Charleston	27	17.6	19.6
S. Charleston	13	5.0	33.5
Kanawha County	40	22.6	22.7
Total U.S.	4604		12.8

Relative risk = 1.77

BDMP Births - 1970-1974

Charleston	13,754
S. Charleston	3,877
Kanawha County	17,631
Total U.S.	3,606,848

* Expected calculated on BDMP total white U.S. rate 1970-1974

+ Rates per 10,000 total births for whites

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APPENDIX

VITAL STATISTICS, KANAWHA COUNTY, WEST VIRGINIA

POPULATION

Census Population, Kanawha County

	<u>1970</u>	<u>1971</u>	<u>1972</u>	<u>1973</u>	<u>1974</u>
S. Charleston	16,330				
Charleston	71,505				
St. Albans	14,356				
County	229,515	230,400	230,300	226,800	223,700

BIRTH RATE

	<u>1970</u>	<u>1971</u>	<u>1972</u>	<u>1973</u>	<u>1974</u>
Kanawha County	16.3	16.5	15.0	14.4	15.0
West Virginia	17.3	17.8	16.7	15.4	15.4

BIRTHS, KANAWHA COUNTY

	<u>1970</u>	<u>1971</u>	<u>1972</u>	<u>1973</u>	<u>1974</u>	
White Male	1724	1805	1658	1624	1616	
White Female	1726	1694	1535	1436	1471	
TOTAL	3450	3499	3193	3060	3087	162.89
Nonwhite Male	148	116	134	120	126	
Nonwhite Female	137	162	107	147	135	
TOTAL	285	278	241	267	261	

<u>City</u>	<u>NUMBER OF BIRTHS</u>				
	<u>1970</u>	<u>1971</u>	<u>1972</u>	<u>1973</u>	<u>1974</u>
Charleston			1035	972	1031
S. Charleston			245	240	227
St. Albans			200	218	198

<u>BIRTHS BY AGE OF MOTHER, KANAWHA COUNTY</u>							
	<u>10-14</u>	<u>15-19</u>	<u>20-24</u>	<u>25-34</u>	<u>35-44</u>	<u>45</u>	<u>Unk</u>
1970	7	711	1504	1309	201	3	
1971	9	761	1553	1274	180		
1972	6	723	1360	1186	156	3	
1973	14	743	1247	1186	136	1	
1974	10	671	1290	1252	124	1	

<u>RESIDENT DEATHS, KANAWHA COUNTY</u>					
	<u>1970</u>	<u>1971</u>	<u>1972</u>	<u>1973</u>	<u>1974</u>
Fetal Deaths	59 (15.8)	60 (15.9)	53 (15.4)	37 (11.1)	28 (8.4)
Congenital Anomaly	25 (10.9)	25 (10.9)	20 (8.7)	15 (6.5)	11 (4.9)
Premature Births	12 (5.2)	3 (1.3)	8 (3.5)	9 (3.9)	7 (3.1)

<u>FAMILIES WITH INCOME LESS THAN POVERTY LEVEL IN 1969 BY COUNTY</u>		
	<u># Families</u>	<u>% Of All Families</u>
Kanawha County	8020	13.0
West Virginia	81697	18.0