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Item ID Number 01140

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Report/Article Title Results of a Pilot Study of Health Effects due to 2,3,7,8-Tetrachlorodibenzodioxin Contamination - Missouri

Journal/Book Title Morbidity and Mortality Weekly Report

Year 1984

Month/Day February 10

Color

Number of Images 4

Description Notes Alvin L. Young filed this item under the category "Human Exposure to Phenoxy Herbicides and TCDD"

Morbidity AND Mortality Weekly Report, 1984

33(5):54-56,61.

54

MMWR

February 10, 1984

Hepatocellular Carcinoma - Continued

mented by ultrasonography and CAT scan, was in an elderly man who declined biopsy and surgery. All patients with tumors were asymptomatic at the time of detection, and all had rising AFP levels or a single level above 1,000 ng/ml. Of the 10 remaining people with elevated AFP, one has had low-level elevations (50-90 ng/ml) and is being evaluated, and nine had transient elevations associated with acute HBV infection. These preliminary results suggest that AFP screening of HBsAg-positive persons can, at least sometimes, detect PHC at a stage when surgical resection may be curative.

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Editorial Note: PHC is a leading cause of cancer deaths in much of Asia and Africa. Worldwide, it is estimated that over 150 million chronic carriers of HBV infection—900,000 of whom live in the United States—are at risk for developing PHC (4).

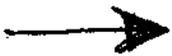
In the past, a PHC diagnosis usually followed the onset of symptoms, and the 5-year survival rate approached zero (5). Of the various treatments for PHC, only surgical resection has resulted in long-term survival. A recent study from the People's Republic of China demonstrated that surgery in asymptomatic patients with tumors less than 5 cm in diameter can result in improved survival (6).

Well-designed prospective studies are needed to evaluate the use of AFP screening in the early detection of PHC. These studies should include measures of sensitivity, specificity, and positive predictive value, as well as an analysis of cost-effectiveness. The preliminary Alaskan experience is promising and will hopefully result in recommendations concerning the use of prospective AFP testing among HBsAg carriers.

While early detection of PHC may improve survival rates, detection is only part of the health-care strategy directed against PHC. Because of the presumed etiologic link between chronic HBV infection and PHC, preventing PHC may be possible by preventing HBV infection. The success of future HBV vaccination programs may well determine the future incidence of PHC.

References

1. Heyward WL, Lanier AP, Bender TR, et al. Primary hepatocellular carcinoma in Alaskan Natives, 1969-1979. *Int J Cancer* 1981;28:47-50.
2. Schreeder MT, Bender TR, McMahon BJ, et al. Prevalence of hepatitis B in selected Alaskan Eskimo villages. *Am J Epidemiol* 1983;118:543-9.
3. Heyward WL, Lanier AP, Bender TR, et al. Early detection of primary hepatocellular carcinoma by screening for alpha-fetoprotein in high risk families: a case-report. *Lancet* 1983;ii:1161-2.
4. Report of a WHO meeting. Prevention of liver cancer. In: WHO Technical Report Series No. 691, 1983.
5. Moertel CG. The liver. In: Holland JF, Frei E III, eds. *Cancer medicine*. 2nd edition. Philadelphia: Lea & Febiger, 1982:1774-81.
6. Tang Z-y, Yu Y-q, Lin Z-y, Yang B-h, Zhou X-d, Cao Y-z. Clinical research of primary liver cancer: a 10 year (1970-1979) survey. *Chin Med J* 1983;96:247-50.



Results of a Pilot Study of Health Effects due to 2,3,7,8-Tetrachlorodibenzodioxin Contamination — Missouri

In 1971, waste oils containing 2,3,7,8-tetrachlorodibenzodioxin (TCDD) were sprayed on residential, recreational, and work areas in Missouri to control dust. In several of these areas, the extent of environmental contamination did not become apparent until late 1982 and into

TCDD Contamination - Continued

1983. Starting in January 1983, the Missouri Division of Health and CDC administered approximately 800 Health Effect Survey screening questionnaires to individuals initially solicited because of potential exposures at residential areas in eastern Missouri. In February, a group of 68 persons considered to have a high probability of exposure (i.e., who lived in, worked at, or recreated at these areas) and a group of 36 persons considered to have no exposure were selected after reviewing these questionnaires. These 104 persons received detailed medical examinations and a series of laboratory tests focused on detecting subclinical effects in key, target-organ systems (i.e., hepatic, dermatologic, immunologic, and neurologic systems).

Comparisons of these two groups produced no consistent indications of increased disease prevalence directly related to the putative exposures; no cases of chloracne, overt porphyria cutanea tarda (PCT)* or precursor conditions of PCT, or soft-tissue sarcomas were seen. An apparent trend of urinary-tract abnormalities was indicated by an increased prevalence of self-reported kidney/urinary problems, a higher proportion of leukocyturia, and a greater prevalence of microscopic hematuria in the group at high risk of exposure. None of the findings from the medical histories or the immune-function assays demonstrated statistically significant differences. There was, however, an indication of an increased prevalence of T_4/T_3 -cell ratios less than 1.0 in the high-risk group. No significant differences in standard and specialized liver-function test results were detected.

This pilot study of a group of individuals presumed to be at high risk of exposure was intended to provide a perspective on the types and degrees of abnormalities likely to be seen in such TCDD exposures. The results appear negative, but no overall definitive conclusion should be based solely on this initial study. The insights provided need to be examined in more refined epidemiologic studies using different designs and strategies (especially in larger, more homogeneous population groups in which exposure status can be better characterized). These studies should be focused primarily, but not exclusively, on discerning any effects on the immune and neurologic systems and the urinary tract and liver.

Reported by K Webb, S Ayres, R Slavin, A Knutsen, S Roodman, St. Louis University, WB Gedney, St. Joseph Hospital, Kirkwood, W Schramm, RL Hotchkiss, R Miller, HD Donnell, State Epidemiologist, Missouri Div of Health; Special Studies Br, Chronic Diseases Div, Clinical Chemistry Div, Center for Environmental Health, CDC.

Editorial Note: Animal toxicity studies are commonly used to predict health effects in humans (although the existence of species-specific and even organ-specific effects of TCDD make extrapolations tenuous). The organ systems most prominently affected in animals are the liver (acute toxicity and hepatocarcinogenesis), the immune system (thymic atrophy and decreased cell-mediated immunity), and the skin (chloracne-like changes); effects on reproduction have also been noted (1,2).

Most direct knowledge of TCDD effects on human health has been obtained from workers exposed to dioxin during the production or subsequent handling of 2,4,5-trichlorophenol (2,4,5-TCP) or 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) (3). In some workplaces, exposed persons had chloracne but no systemic illnesses; other reports have noted that workers fatigued easily and experienced weight loss, myalgias, insomnia, irritability, and decreased libido. The liver has been shown to become tender and enlarged, and sensory changes, particularly in the lower extremities, have been reported. Total serum lipids may be increased, and the prothrombin times may be prolonged (4). PCT has also been observed (5). The most specific of the dioxin-related findings are chloracne (which can also be caused by other structurally similar compounds, such as polychlorinated biphenyls [PCBs] and chlorinated naphthalenes) and PCT (which also has a variety of potential causes). A number of studies ad-

*An acquired form of porphyria characterized by chronic skin lesions and other symptoms.

TCDD Contamination - Continued

Investigating the association of TCDD exposures to soft-tissue sarcoma have been conducted in the industrial setting. These include two case-control studies in Sweden in which investigators reported a sixfold increase in the risk of soft-tissue sarcomas among persons exposed to chlorophenols and phenoxy herbicides (6).

Information on health effects involving nonoccupational environmental exposure is sparse. In 1976, after an explosion at a Seveso, Italy, chemical plant, chloracne developed in exposed children; some elevated liver-function test results were detected in the exposed population, and the incidence of abnormal nerve conduction tests was reported significantly elevated in subjects with chloracne (7). In Missouri, after playing in dirt in a riding arena contaminated with up to 33 parts per million TCDD, a child had hemorrhagic cystitis (8).

Public health policy in situations such as this environmental contamination with TCDD must continue to focus on the prevention of any potential health effects (particularly delayed or long-term), even if effects are not demonstrated in a pilot study. For this reason, appropriate efforts to prevent human exposure must continue, in this and other similar situations, until a more complete understanding of public health risks is obtained.

(Continued on page 61)

TABLE I. Summary—cases specified notifiable diseases, United States

Disease	5th Week Ending			Cumulative, 5th Week Ending		
	February 4, 1984	February 5, 1983	Median 1979-1983	February 4, 1984	February 5, 1983	Median 1979-1983
Acquired Immunodeficiency Syndrome (AIDS)	51	N	N	257	N	N
Asplenic meningitis	86	93	59	440	450	369
Encephalitis: Primary arthropod-borne & unsp. (C)	20	12	12	64	83	78
Post-infectious	-	-	2	3	5	9
Gonorrhea: Civilian	14,831	17,887	19,785	78,872	91,339	92,424
Military	379	427	822	2,009	2,417	2,782
Hepatitis: Type A	443	643	543	1,890	2,305	2,250
Type B	474	387	361	1,937	1,930	1,630
Non A, Non B	67	79	N	281	289	N
Unspecified	121	149	183	517	677	868
Legionellosis	5	11	N	31	49	N
Leprosy	1	2	4	15	28	14
Malaria	14	14	14	53	52	57
Measles: Total*	91	3	40	137	40	164
Indigenous	91	1	N	132	28	N
Imported	-	2	N	5	12	N
Meningococcal infections: Total	59	66	62	232	266	273
Civilian	59	55	62	232	268	269
Military	-	1	-	-	8	1
Mumps	60	75	104	291	381	472
Pertussis	14	30	29	108	90	90
Rubella (German measles)	8	23	39	37	72	207
Syphilis (Primary & Secondary): Civilian	592	652	645	2,587	3,950	2,915
Military	2	6	7	33	51	42
Toxic Shock syndrome	8	8	N	28	43	N
Tuberculosis	395	442	471	1,585	1,726	1,843
Tularemia	-	5	2	5	13	11
Typhoid fever	2	4	8	19	30	30
Typhus fever, tick-borne (RMSF)	2	-	1	6	6	8
Rabies, animal	70	99	95	257	443	422

TABLE II. Notifiable diseases of low frequency, United States

	Cum. 1984		Cum. 1984
Anthrax	-	Plague (Wash. 1)	1
Botulism: Foodborne	-	Polioomyelitis: Total	-
Infant (Calif. 1)	5	Paralytic	-
Other	1	Psittacosis (Upstate N.Y. 2)	6
Brucellosis (Upstate N.Y. 1, Ohio 1, Mo. 1, Va. 1, Calif. 2)	11	Rabies, human	-
Cholera	-	Tetanus (Kans. 1, Calif. 1)	2
Congenital rubella syndrome	-	Trichinosis	2
Diphtheria	-	Typhus fever, flea-borne (endemic, murine)	2
Leptospirosis (Ohio 1, Hawaii 1)	2		

*There were no cases of internationally imported measles reported for this week.

*TCDD Contamination – Continued**References*

1. Gupta BN, Vos JG, Moore JA, Zinkl JG, Bullock BC. Pathologic effects of 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin in laboratory animals. *Environ Hlth Per* 1973;5:125-40.
2. Kociba RJ, Keyes DG, Beyer JE, et al. Results of a two-year chronic toxicity and oncogenicity study of 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin in rats. *Toxicol Appl Pharmacol* 1978;46:279-303.
3. Kimbrough RD, ed. Topics in environmental health. Volume 4. Halogenated biphenyls, terphenyls, naphthalenes, dibenzodioxins and related products. Amsterdam, The Netherlands: Elsevier/North Holland Biomedical Press, 1980.
4. Jensen NE, Walker AE. Chloracne: three cases. *Proc R Soc Med* 1972;65:687-8.
5. Poland AP, Smith D, Metter G, Possick P. A health survey of workers in a 2,4-D and 2,4,5-T plant with special attention to chloracne, porphyria cutanea tarda, and psychologic parameters. *Arch Environ Health* 1971;22:316-27.
6. Eriksson M, Hardell L, Berg NO, Moller T, Axelson O. Soft-tissue sarcomas and exposure to chemical substances: a case-referent study. *Br J Ind Med* 1981;38:27-33.
7. Reggiani G. Acute human exposure to TCDD in Seveso, Italy. *J Toxicol Environ Health* 1980;6:27-43.
8. Carter CD, Kimbrough RD, Liddle JA, et al. Tetrachlorodibenzodioxin: an accidental poisoning episode in horse arenas. *Science* 1975;188:738-40.

*Current Trends***Measles Surveillance – Canada**

A provisional total of 915 measles cases was reported in Canada for 1983. This appears to be the lowest incidence reported since national reporting of measles began in 1924. However, complete data are available only through 1982.

In 1982, 1,064 cases of measles were reported in Canada, a rate of 4.3 cases per 100,000 population. Compared with 1981 and 1980, this reflects a 55% and a 92% reduction, respectively, and a 99% reduction compared with the 10-year prevaccine period 1949-1958 (Figure 1).

All provinces except Prince Edward Island reported measles cases in 1982. Although Ontario accounted for the largest proportion of cases (48%), it reported a 41% reduction in incidence rate compared with 1981.

The age distribution of measles patients in 1982 was available for all provinces except Ontario, for which data were available from January to June 1982. Children under 1 year of age accounted for 19% of cases; under 5 years, 27%; and under 10 years, 75%. The highest rate (43 cases per 100,000 persons) occurred among infants, followed by preschoolers (1-4 years), with a rate of 15.1 per 100,000 persons. In Ontario, 21% of children were less than 5 years old; school-aged children (5-19 years) accounted for 73% of 224 cases.

All provinces are attempting to eliminate measles either by compulsory vaccination at school entry or by voluntary approaches, and some have reported that up to 95% of children are now immunized by the time they reach school age. New Brunswick and Ontario (representing 39% of Canada's population) introduced legislation in 1981 and 1982, respectively, making immunization against measles and five other diseases (diphtheria, tetanus, pertussis, polio, and rubella) compulsory for school attendance.

Reported by Health and Welfare, Canada; Weekly Epidemiological Record 1983;58:331-2, World Health Organization; Div of Immunization, Center for Prevention Svcs, CDC.