

International Notes

Preliminary Report: 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin Exposure to Humans – Seveso, Italy

At approximately noon on Saturday, July 10, 1976, an explosion occurred during the production of 2.4.5-trichlorophenol in a factory in Meda, about 25 km north of Milan, in the Lombardia region of Italy. A cloud of toxic material was released and included 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). Debris from this cloud fell south-southeast of the plant on an area of about 2.8 km² (700 acres), including parts of the towns of Seveso, Meda, Cesano Maderno, and Desio. The size of the contaminated area was estimated primarily by measuring TCDD in the soil; additional criteria included the presence of dead animals (e.g., birds, rabbits, chickens) and detection of dermal lesions among persons in the area. The contaminated area was divided into three zones (A, B, and R) depending on the concentration of TCDD in the soil (Table 1). An additional zone, Zone S, outside the contaminated area was examined as a control zone.* Zone A, the most heavily contaminated section, was further divided into seven subzones, A1-A7, based on increasing distance from the factory. The total amount of TCDD deposited in the contaminated area was initially estimated at about 165 g (1); subsequently, it has been estimated to be at least 1.3 kg (2).

Within 20 days of the explosion, the Italian authorities had evacuated the 211 families (735 persons) from the area later defined as Zone A and had taken immediate measures to minimize the risk of exposure to residents in nearby areas (primarily

*Zone R was originally thought to be a reference zone, but on subsequent detection of TCDD concentrations in soil, Zone S was added.

		Size	Soil sampling	TCDD concentration (µg/m ²			
Zone	Population	(acres)	square (m ²)	Mean	Highest		
A	735	198	2,500	230.0	5477.0		
В	4,699	665	22,500	3.0	43.9		
R	31,800	3,532	22,500	0.9	9.7		

TABLE 1. Distribution* of TCDD contamination in soil[†] – Seveso, Italy, 1976

*These data may be underestimated by approximately 25% (2).

[†]Measured in a soil sample, 20 cm of depth, taken at the center of a square.

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those in Zone B). The Italian authorities were assisted by several national and international technical commissions in assessing adverse health effects. Residents of zones A, B, and R underwent extensive medical examinations from 1976 to 1985; chloracne, detected in a small segment of the population, was the only abnormal finding (3–6). Only one potentially exposed person was measured for TCDD; she was a 55-year-old woman residing in a portion of Zone A (mean TCDD soil concentration of 185.4 μ g/m²), who died from pancreatic adenocarcinoma 7 months after the explosion. Her TCDD whole-weight levels varied from 6 parts per trillion (ppt) in blood to 1840 ppt in adipose tissue[†] (7).

In April 1988, a group of U.S. and Italian scientists convened to further examine the Seveso TCDD incident. Since more than 30,000 serum or plasma samples (volumes of 1–3 mL) had been collected from residents of the four zones from the end of July 1976 through 1985 and stored at –30 C (–22 F), the group agreed to assess whether methodology developed at CDC to measure TCDD in human serum (8) could be used to measure TCDD in these low-volume samples. This methodology, a lipid-based measurement highly correlated with paired measurements of TCDD in adipose tissue (p = 0.98) (9), has been used to evaluate U.S. Army veterans (10), U.S. Air Force Operation Ranch Hand veterans (11), and occupationally exposed persons (D.G. Patterson, Jr., et al., unpublished data).

The preliminary Seveso study evaluated serum samples from five Zone A residents who developed the most severe types (III or IV) of chloracne; four Zone A residents who did not develop chloracne or other health problems (in 1976, each was \geq 15 years of age); and five persons from Zone S. All these samples had been collected in 1976 and were sent without identification to CDC for analysis. These samples were analyzed for TCDD (8) on both a whole-weight and a lipid basis, using triglycerides and total cholesterol data provided for those samples by the laboratory of the hospital of Desio-Milan to calculate total lipids.

The TCDD levels detected are the highest ever reported in humans (Figure 1). The three highest levels are from children who developed chloracne. Levels for the other two chloracne cases were similar to those in residents without chloracne. TCDD was not detected in four of the five controls. In one control, a level of 137 ppt TCDD on a lipid basis was detected; this value may represent either an actual level or the detection of a residue of <1% from a sample analyzed immediately before this sample.

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Editorial Note: Little is known about TCDD exposures and adverse health effects in humans. However, these Seveso samples are unique in that they were taken in proximity of time to an acute human exposure to TCDD. Thus, they and the subsequent samples allow for correlating TCDD levels and adverse health effects, if any, and for determining the half-life of TCDD in humans. This population has no apparent adverse health effects other than chloracne (4).

These serum measurements confirm overt exposure to TCDD in those persons tested who resided in Zone A. The levels are of the same magnitude as those found in occupational studies (12,13) that estimate initial TCDD levels by extrapolating to

[†]Levels of TCDD in serum or adipose tissue of the general population are <20 ppt.

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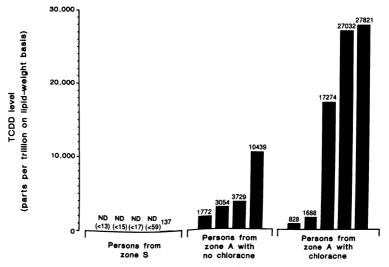
TCDD – Continued

the time of last exposure (by assuming first-order kinetics and a half-life of 7 years) (14). Although the three highest TCDD serum levels occurred in persons who developed chloracne, no threshold level for chloracne is obvious.

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FIGURE 1. Serum TCDD levels* of Zone A residents (with and without chloracne) and Zone S controls – Seveso, Italy



*Levels of TCDD in serum or adipose tissue of the general population are <20 ppt. ND=Nondetectable at specified levels.

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Current Trends

Licensure of Screening Tests for Antibody to Human T-Lymphotropic Virus Type I

Screening tests for antibody to human T-lymphotropic virus type I (HTLV-I), the first-recognized human retrovirus, have been licensed by the Food and Drug Administration (FDA). These tests have been recommended by the FDA for screening of blood and cellular components donated for transfusion. They have also been approved as diagnostic tests, which may be useful in evaluating patients with clinical diagnoses of adult T-cell leukemia/lymphoma (ATL) and tropical spastic paraparesis (TSP)/HTLV-I-associated myelopathy (HAM), both of which have been associated with HTLV-I infection. Because licensure will probably result in widespread use of these tests, the information presented below is provided for physicians and public health officials who may need to interpret HTLV-I test results and to counsel persons whose serum specimens are reactive in these tests. Users of the new HTLV-I screening tests are cautioned that additional, more specific tests are necessary to confirm that serum specimens that are repeatably reactive in these screening tests are truly positive for HTLV-I antibody. Users should also be aware that neither the screening tests nor more specific tests can distinguish between antibody to HTLV-I and antibody to the closely related human retrovirus, human T-lymphotropic virus type II (HTLV-II).

HTLV-I does not cause AIDS, and the finding of HTLV-I antibody in human blood does not imply infection with human immunodeficiency virus (HIV) or a risk of developing acquired immunodeficiency syndrome (AIDS).

BACKGROUND: HTLV-I

HTLV-I was isolated in 1978 and first reported in 1980 (1). Although a member of the family of retroviruses, HTLV-I is *not* closely related to HIV, the virus that causes AIDS. HTLV-I does not cause depletion of T-helper lymphocytes, and it is not generally associated with immunosuppression.

HTLV-I differs from HIV in its morphologic and genetic structure and in that HTLV-I antigens should not cross-react with the antigens of HIV. The HTLV-I genome contains four major genes: *gag*, which encodes core proteins of 15,000 (p15), 19,000 (p19), and 24,000 (p24) daltons; *pol*, which encodes a polymerase (reverse transcriptase) protein of 96,000 daltons; *env*, which encodes envelope glycoproteins of 21,000 (gp21) and 46,000 (gp46) daltons; and *tax*, which encodes a transactivator protein of 40,000 daltons (p40x).

Seroprevalence

HTLV-I infection is endemic primarily in southwestern Japan, the Caribbean, and some areas of Africa (2). Seroprevalence in well-characterized areas appears to increase with patient age, with rates in females markedly higher than those in males beginning in the 20–30-year age range. Seroprevalence rates as high as 15% in the general population and 30% in older age groups have been reported in some areas of Japan (3). In the Caribbean islands, rates may be as high as 5% in the general population and 15% in older age groups (4).

In the United States, HTLV-I infection has been identified mainly in intravenousdrug users (IVDUs), with seroprevalence rates ranging from 7% to 49% (5,6). Elevated rates have also been reported in female prostitutes (in whom IV-drug use is a major risk factor) (7) and in recipients of multiple blood transfusions (8). Seropositivity is rare among homosexual men and among patients in sexually transmitted disease clinics (9,10), and it appears to be nonexistent in hemophilic men without other risk factors (11). Systematic determination of HTLV-I seroprevalence in the general population of the United States has not been undertaken. However, in a study of 39,898 random blood donors in eight U.S. cities, 10 (0.025%) were seropositive for HTLV-I (12).

Transmission

Transmission of HTLV-I infection by blood transfusion is well documented in Japan, with a seroconversion rate of 63% in recipients of the *cellular* components of contaminated units (whole blood, red blood cells, and platelets) (*13*). Transmission by the plasma fraction of contaminated units has not resulted in infection; this finding has been attributed to the fact that HTLV-I is highly cell-associated. Transmission among IVDUs is presumed to occur by sharing of needles and syringes contaminated with infectious blood.

Transmission from mother to child occurs through breastfeeding; breastfed infants of seropositive mothers have an approximately 25% probability of becoming infected (14). However, infection has also occurred in infants who are not breastfed, suggesting that intrauterine and/or perinatal transmission may occur.

Sexual transmission of HTLV-I appears to be relatively inefficient (15). Transmission from male to female, however, appears to be more efficient than from female to male (16).

Disease Associations

HTLV-I has been etiologically associated with adult T-cell leukemia/lymphoma (ATL), a malignancy of mature T-lymphocytes characterized by skin lesions, visceral involvement, circulating abnormal lymphocytes, hypercalcemia, and lytic bone lesions (*17*). ATL has been recognized in Japan, the Caribbean, and Africa. No systematic attempt has been made to record cases of ATL in the United States, but 74 cases were reported to the National Institutes of Health between 1980 and 1987 (*18*). Approximately half of these cases occurred in persons of Japanese or Caribbean

ancestry; most of the remainder were in blacks from the southeastern United States. ATL tends to occur equally in men and women, with peak occurrence in persons 40–60 years of age.

It is thought that a person must be infected with HTLV-I for years to decades before ATL develops. The lifetime risk of ATL among HTLV-I-infected persons has been estimated to be approximately 2% in two studies in Japan (*19,20*). In Jamaica, the lifetime risk of ATL among persons infected before the age of 20 years was estimated to be 4% (*21*).

Because of the long latent period of ATL, the risk of this disease among persons infected by blood transfusion (many of whom are elderly and may not survive their underlying disease) is not thought to be substantial. In fact, no cases of ATL associated with blood transfusion have been reported.

HTLV-I has also been associated with a degenerative neurologic disease known as tropical spastic paraparesis (TSP) in the Caribbean and as HTLV-I–associated myelopathy (HAM) in Japan (22,23). TSP/HAM is characterized by progressive difficulty in walking, lower extremity weakness, sensory disturbances, and urinary incontinence. Although most cases have been reported from countries in which HTLV-I is endemic, a few cases have occurred in the United States (24). TSP/HAM occurs in persons of all age groups, with peak occurence in ages 40–49 years. Rates are higher in females than in males. The lifetime risk of TSP/HAM among persons infected with HTLV-I is unknown but appears to be very low. The latent period for this disease appears to be less than for ATL, and the disease probably can be caused by blood transfusion. Of 420 Japanese patients with HAM from whom information was available, 109 (26%) gave a history of blood transfusion; the mean interval between transfusion and onset of neurologic symptoms was estimated to be 4 years (M. Osame, unpublished data).

HTLV-I does not cause AIDS, and the finding of HTLV-I antibody in human blood does not imply infection with HIV or a risk of developing AIDS.

BACKGROUND: HTLV-II

HTLV-II is closely related to HTLV-I. The genome of HTLV-II encodes viral proteins that are similar to those of HTLV-I, and there is extensive serologic cross-reactivity among proteins from HTLV-I and HTLV-II.

No specific information is available regarding the seroepidemiology or the modes of transmission of HTLV-II. There is some evidence that some of the HTLV-I seropositivity in the United States, especially in IVDUs, may be caused by HTLV-II (5).

Two cases of disease have been associated with HTLV-II infection. HTLV-II was first isolated from a patient with a rare T-lymphocytic hairy cell leukemia (25). In the second case, HTLV-II was isolated from a patient who had the more common B-lymphocytic form of hairy cell leukemia and who also suffered from a T-suppressor lymphoproliferative disease (26). No serologic evidence of HTLV-II infection has been found in 21 additional cases of hairy cell leukemia (27). Thus, the disease associations of HTLV-II are unclear, and nothing is known regarding lifetime risk of disease among infected persons.

SEROLOGIC TESTS FOR HTLV-I

Interpretation

The screening tests that have been licensed by the FDA are enzyme immunoassays (EIAs) to detect HTLV-I antibody in human serum or plasma. Specimens with absorbance values greater than or equal to the cutoff value determined by the

manufacturer are defined as initially reactive. Initially reactive specimens must be retested in duplicate to minimize the chance that reactivity is due to technical error. Specimens that are reactive in either of the duplicate tests are considered repeatably reactive. Specimens that do not react in either of the duplicate repeat tests are considered nonreactive. Additional, more specific serologic tests are necessary to confirm that serum specimens repeatably reactive in the screening tests are positive for HTLV-I antibody. Users of the screening tests must have available to them additional, more specific tests to properly interpret repeatably reactive screening tests. Such tests are available in research institutions, industry, and some diagnostic laboratories. No such tests have been licensed by the FDA.

Tests used to confirm HTLV-I seropositivity must be inherently capable of identifying antibody to the core (gag) and envelope (env) proteins of HTLV-I. (The immunoreactivities of the polymerase [pol] and transactivator [tax] proteins of HTLV-I have not been well-defined in current test systems.) Specific tests include Western immunoblot (WIB) and radioimmunoprecipitation assay (RIPA). Indirect fluorescent antibody (IFA) testing for HTLV-I has been used in some laboratories, but IFA does not detect antibody to specific HTLV-I gene products.

WIB appears to be the most sensitive of the more specific tests for antibody to *gag* protein products p19, p24, and (*gag*-derived) p28, whereas RIPA appears to be most sensitive for antibody to the *env* glycoproteins gp46 and (*env* precursor) gp61/68. Based on experience with these tests in several laboratories, the following confirmatory criteria for HTLV-I seropositivity have been adopted by the Public Health Service Working Group: a specimen must demonstrate immunoreactivity to the *gag* gene product p24 and to an *env* gene product (gp46 and/or gp61/68) to be considered "positive." Serum specimens not satisfying these criteria but having immunoreactivities to at least one suspected HTLV-I gene product (such as p19 only, p19 and p28, or p19 and *env*) are designated "indeterminate." Serum specimens with no immunoreactivity to any HTLV-I gene products in additional, more specific tests are designated "negative." Both WIB and RIPA may be required to determine whether a serum specimen is positive, indeterminate, or negative.

Although additional, more specific tests have been somewhat standardized, the quantities and the molecular weights of HTLV-I proteins produced by various cell lines vary considerably. Hence, the cell of origin for HTLV-I antigens used in either WIB or RIPA, as well as the method of antigen preparation, may markedly influence test sensitivity and interpretation of immunoreactivity against individual HTLV-I proteins. Laboratories performing these tests, however, should be able to detect antibody to the *gag* and *env* gene products of HTLV-I in WIB and/or RIPA.

Sensitivity, Specificity, and Predictive Value

Using the WIB and RIPA available in research laboratories and the confirmatory criteria described above to define the presence of HTLV-I antibody, the sensitivities of the three EIAs that have been licensed by the FDA have been estimated from the performance of the tests on a reference panel of 137 antibody-positive serum specimens. All three EIAs were repeatably reactive for 137 of 137 panel serum specimens, yielding an estimated sensitivity of 97.3%–100% by the binomial distribution at 95% confidence. Specificity* of the EIAs was estimated for each test from

^{*}Specificity was calculated as follows: (total donations screened minus total number repeatably reactive in EIA) divided by (total donations screened minus number confirmed as positive by additional testing).

screening of at least 5000 normal U.S. blood donors in nonendemic areas. Estimated specificities ranged from 99.3% to 99.9% by the binomial distribution at 95% confidence. However, a specificity >99% but <100% may still yield a low positive predictive value when the screening test is used in a low-prevalence population. For example, in the study of U.S. blood donors cited above, 68 donors were repeat reactors in the screening test, but only 10 (15%) were determined to be HTLV-I-seropositive in more specific testing. This relatively low positive predictive value emphasizes the need for additional, more specific testing of specimens repeatably reactive in the EIA.

Neither the EIAs nor the additional, more specific tests can distinguish between antibodies to HTLV-I and HTLV-II. More sophisticated techniques, such as virus isolation and gene amplification (polymerase chain reaction [PCR]) are required to differentiate HTLV-I from HTLV-II infection.

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	48	th Week End	ing	Cumulati	ve, 48th We	ek Ending
Disease	Dec. 3, 1988	Dec. 5, 1987	Median 1983-1987	Dec. 3, 1988	Dec. 5, 1987	Median 1983-1987
Acquired Immunodeficiency Syndrome (AIDS)	215	U*	110	27,974	19,027	7,262
Aseptic meningitis	152	163	173	6,295	10,548	10,083
Encephalitis: Primary (arthropod-borne						
& unspec)	10	13	16	710	1,215	1,215
Post-infectious	2	3	1	112	95	98
Gonorrhea: Civilian	10,158	14,387	15,056	638,020	710,087	819,309
Military	99	214	237	10,630	15,042	19,346
Hepatitis: Type A	588	514	455	24,055	22,741	21,041
Type B	406	474	474	20,767	23,417	23,737
Non A, Non B	43	35	59	2,314	2,699	3,245
Unspecified	59	62	103 17	2,171 905	2,863	4,746
Legionellosis	13 10	10 4	4	905	880 184	713
Leprosy Malaria	16	10	12	922	828	221 921
Measles: Total [†]	26	10	15	2.743	3,548	2,704
Indigenous	26	10	12	2,423	3,129	2,272
Imported	20	10	1	320	419	304
Meningococcal infections	31	44	44	2,562	2,676	2,461
Mumps	129	224	67	4,288	11,859	3,044
Pertussis	71	60	38	2,657	2,345	2,345
Rubella (German measles)	· 1	5	5	189	329	601
Syphilis (Primary & Secondary): Civilian	830	645	521	37.175	32,694	25,709
Military	1	3	3	145	150	152
Toxic Shock syndrome	2	8	8	317	311	345
Tuberculosis	378	451	451	19,472	19,757	19,757
Tularemia		1	3	170	186	186
Typhoid Fever	5	19	4	361	333	345
Typhus fever, tick-borne (RMSF)	11	4	4	605	586	731
Rabies, animal	76	76	76	3,958	4,357	5,009

TABLE I. Summary – cases of specified notifiable diseases, United States

TABLE II. Notifiable diseases of low frequency, United States

	Cum. 1988		Cum. 1988
Anthrax Botulism: Foodborne Infant Other (Oreg. 1) Brucellosis (Mich. 4) Cholere Congenital rubella syndrome Congenital syphilis, ages < 1 year Diphtheria	26 33 4 67 7 4 426	Leptospirosis (Fla. 1, Hawaii 1) Plague Poliomyelitis, Paralytic Psittacosis (Ariz. 1, Wash. 2, Calif. 2) Rabies, human Tetanus Trichinosis	43 14 1 86 - 48 40

*Because AIDS cases are not received weekly from all reporting areas, comparison of weekly figures may be misleading. There were no cases of internationally imported measles reported for this week.

	1	Aseptic	Encer	halitis			н	epatitis (Viral), by	type	I	
Reporting Area	AIDS	Menin- gitis	Primary	Post-in- fectious		ilian)	A	В	NA,NB	Unspeci- fied	Legionel- losis	Leprosy
	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1987	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988
UNITED STATES	27,974	6,295	710	112	638,020	710,087	24,055	20,767	2,314	2,171	905	164
NEW ENGLAND	1,183	386	24	4	20,116	22,058	798	1,122	112	87	50	15
Maine	27	20	2	-	366	654	18	51	5	2 4	4	-
N.H.	38 10	40 29	1	3	241 110	373 203	45 15	69 53	11	4	5 5	:
Vt. Mass.	650	157	8	1	6.777	7.736	373	668	71	61	33	14
R.I.	81	87		-	1,884	2,003	84	80	11	-	3	1
Conn.	377	53	6	-	10,738	11,089	263	201	8	16	-	-
MID. ATLANTIC	9,303	700	53	4	100,706	111,902	1,847	2,994	181	302	208 78	8
Upstate N.Y. N.Y. City	1,256 5,070	374 134	34 8	1 3	14,832 41,750	16,415 59,753	701 342	710 1,257	72 19	19 224	45	7
N.J.	2,206	61	11	-	14,530	15,133	420	676	60	42	40	i
Pa.	771	131	•	-	29,594	20,601	384	351	30	17	45	-
E.N. CENTRAL	1,993	1,038	182	13	108,086	108,894	1,603	2,198	205	123	229	7
Ohio	467	414	62	3	24,444 8,252	24,859 8,789	313 152	513 326	35 19	19 28	83 27	:
Ind. III.	80 924	96 92	28 32	10	8,252 32,315	31,217	552	475	74	34	21	6
Mich.	417	389	43	-	34,568	34,588	374	628	53	39	58	
Wis.	105	47	17	•	8,507	9,441	212	256	24	3	40	1
W.N. CENTRAL	684	253	55	12	27,416	28,556	1,291	931	100	34	74	1
Minn.	146	30	14	4	3,636	4,255	93	130	23	4 2	4 18	-
lowa Mo.	39 364	36 105	9 1	3	2,057 15,807	2,791 15,231	44 787	77 556	13 44	18	23	:
N. Dak.	304	5	4	-	175	270	6	14	3	5	1	-
S. Dak.	Ż	18	5	2	450	561	27	4	3	-	14	•
Nebr.	34	11	12	2	1,416	1,877	46	40	2	- 5	5	:
Kans.	90	48	10	1	3,875	3,571	288	110	12		9	1
S. ATLANTIC	4,959	1,364	103	40	180,346	185,789	2,232	4,334	359 8	332	135 13	1
Del.	62 497	43 194	3 10	3	2,849 18,661	3,167 21,420	44 273	128 662	40	26	20	1
Md. D.C.	448	21	1	1	13,525	12,395	16	45	4	1	1	-
Va.	338	199	32	4	13,262	13,553	348	309	72	225	11	-
W. Va.	20	36	22	-	1,261	1,297	14	66	5 87	4	31	•
N.C. S.C.	264 167	165 21	21	1	25,686 14,431	27,962 14,192	327 40	782 500	12	5	26	
Ga.	691	153	1	2	34,208	33,135	574	631	14	6	21	-
Fla.	2,472	532	13	29	56,463	58,668	596	1,211	117	61	12	-
E.S. CENTRAL	713	429	61	8	50,725	53,462	711	1,330	174	13	48	2
Ky.	89	145	21	1	5,177	5,359	466	265	61	2	20	•
Tenn.	324	48	15	2	17,597	18,830	157 55	604 347	40 62	10	8 14	2
Ala. Miss.	195 105	180 56	25	5	15,198 12,753	16,755 12,518	33	114	11	1	6	-
W.S. CENTRAL	2,349	755	86	3	67,880	79,845	3,010	1,913	198	514	27	39
Ark.	76	15	6		6,804	9,007	323	104	6	17	4	-
La.	340	117	24	1	13,698	13,158	152	332	25	16	7	7
Okla. Tex.	127 1,806	75 548	8 48	2	6,485 40,893	8,634 49,046	466 2,069	166 1,311	42 125	29 452	16	32
MOUNTAIN	820	223	28	4	13,709	18,492	3,171	1,503	235	161	44	1
Mont.	16	4	- 20	-	384	517	40	54	10	4	2	-
Idaho	10	3	-	-	308	635	126	105	9	4	-	-
Wyo.	6	2	:	-	188	399	5	12	3	70	3	-
Colo. N. Mex.	299 49	70 23	3 3	1	2,969 1,362	4,190 2,016	220 509	185 220	64 18	/0	8	1
Ariz.	261	79	13	1	5,037	6,288	1,766	586	72	55	19	
Utah	60	25	4	2	499	595	286	128	37	18	3	•
Nev.	119	17	5	-	2,962	3,852	219	213	22	9	5	-
PACIFIC	5,970	1,147	118	24	69,036	101,089	9,392	4,442	750	605	90	90
Wash.	361	-	7	4	6,481	8,265	2,144	813	183	70	22	7
Oreg.	173 5,320	1,017	106	- 20	2,990	3,708	1,273 5,430	540 2,985	85 469	21 497	4 61	1 67
Calif. Alaska	5,320	25	3	20	58,051 970	86,828 1,517	5,430	∠,985 54	469	497	-	1
Hawaii	97	105	2	-	544	771	12	50	5	5	3	14
Guam	1	-		-	136	180	9	13	-	2	1	5
P.R.	1,229	69	4	1	1,210	1,763	52	241	41	41	-	3
V.I.	32	:	-	-	414	268	1	7	2	- 5	-	-
Amer. Samoa C.N.M.I.	:	-	-	-	74 47	82	7	2 3	-	5	-	2 1
	_		_	-		-						·

TABLE III. Cases of specified notifiable diseases, United States, weeks ending December 3, 1988 and December 5, 1987 (48th Week)

N: Not notifiable

	Malaria			les (Rui			Menin- gococcal	_{Ми}	mps		Pertussi	•		Rubella	•
Reporting Area			enous		orted*	Total	Infections								
<u></u>	Cum. 1988	1988	Cum. 1988	1988	Cum. 1988	Cum. 1987	Cum. 1988	1988	Cum. 1988	1988	Cum. 1988	Cum. 1987	1988	Cum. 1988	Cum. 1987
UNITED STATES	922	26	2,423	-	320	3,548	2,562	129	4,288	71	2,657	2,345	1	189	329
NEW ENGLAND	70	-	83	-	54	281	219	-	117	-	176	160	-	9	2
Maine N.H.	3 3	:	7	:	-	3	10	-		-	24	28	-	-	1
Vt.	5	-	67	-	44	162 26	23 17		105 5	-	47 5	39 4	-	5	-
Mass.	33	-	2	-	2	66	95	-	7	-	60	54	-	3	1
R.I. Conn.	7 19	-	.7	-	- 8	2 22	21	-	-	-	17	5	-	1	-
		-		-			53	-	-	-	23	30	-	-	-
MID. ATLANTIC Upstate N.Y.	162 39	-	903 19	:	50 18	586 41	280 130	3 1	350 97	7	235	288	-	14	12
N.Y. City	89	-	46	-	6	466	65		101	4	142 8	162 19	-	27	10 1
N.J.	11	-	309	-	12	39	63	2	55	2	17	20	-	3	i
Pa.	23	•	529	-	14	40	22	-	97	1	68	87	-	2	-
E.N. CENTRAL Ohio	48	:	141	-	108	383	357	34	851	2	241	256	-	31	40
Ind.	11 4	-	2 57	-	83	5	129 29	17 4	130 77	-	49 74	74	-	1	-
III.	3	-	56	-	16	203	74	9	308	-	44	17 17	:	26	29
Mich.	23	-	26	-	5	29	85	4	219	2	37	47	-	4	23
Wis.	7	-	-	-	4	146	40	-	117	-	37	101	-	-	2
W.N. CENTRAL Minn.	18	-	11	-	3	230	95	14	205	1	128	138	-	2	2
lowa	6 2	-	10	-	1	39	20	-	-	-	49	13	-	-	-
Mo.	6	-	1	-	i	189	37	2	36 40	1	34 22	58 34	-	•	1
N. Dak.	-	-	-	-	-	1	1	-	-	-	11	14	-	-	-
S. Dak. Nebr.	1	-	-	-	-	-	4	-	1	-	5	3	-	-	-
Kans.	3	-	-	-		1	12 21	12	11 117	-	7	1 15	-	2	ī
S. ATLANTIC	121	1	396	-	22	170	440	42	727	4	243	305	-		
Del.	1	-	-	-	-	32	2	42	1	-	243	305	-	18	19 2
Md.	21	1	12	-	5	10	53	35	164	-	46	19	-	1	3
D.C. Va.	12 20	-	218	-	2	1	9 52	6	285	1	1	-	-	.:	1
W. Va.	3	-	6	-	-		52	1	136 18	i	24 9	52 39	-	11	1
N.C.	16	-	-	-	5	6	67	-	51	i	66	119	-	1	1
S.C. Ga.	10 6		-	-	-	2	37	-	6	-	1		-	-	-
Fla.	32	-	160	-	10	10 108	69 144	-	31 35	1	36 53	23 48	•	2 3	2 9
E.S. CENTRAL	20	_	70			8	239		443				-		
Ky.	1	-	35	-	-	-	239	1	210	2	100 12	48 2	-	2	3 2
Tenn. Ala		-	1	-	-	-	130	1	215	-	29	15	-	2	1
Ala. Miss.	10 9	-	34	:	-	4	40		15	-	55	24	-	-	-
W.S. CENTRAL		-		-	•	•	15	N	N	-	4	7	-	-	-
Ark.	82 4	-	14	-	3 1	448	172	22	835	30	233	304	-	11	11
La.	12	-	-	-	<u>!</u>	-	20 48	8 4	133 301	9	34 18	13 50	-	4	2
Okla.	10	-	8	-	-	4	19	-	197	-	62	163	-	1	5
Tex.	56	-	6	-	2	444	85	10	204	21	119	78	-	6	4
MOUNTAIN	43	17	135	-	33	497	75	4	208	17	796	211	-	6	25
Mont. Idaho	5 2	17	23	:	31	128	2	:	2	-	2	6	-	-	8
Wyo.	-	-	-	-	1	2	8	1	6 4	2	330 2	71 5	-	-	1
Colo.	15	-	112	-	1	9	19	-	32	-	29	68	-	2	1
N. Mex. Ariz.	2 13	-	-	-	-	318	11	N	N	1	53	13	-	-	-
Utah	4	-		-	-	36 1	18 15	2	139 7	14	352	38	-	-	5
Nev.	2	-	-	-	-	3	2	1	18	-	27 1	10	-	3 1	10
PACIFIC	358	8	670	-	47	945	685	9	552	10	505	635			-
Wash.	24	-	7	-	-	44	63	2	59	2	113	635 97	1	96	215 2
Oreg. Calif.	16 304	- 8	6	-	2	102	43	N	N	2	50	71	-	-	2
Alaska	304	-	653 1	-	37	794 1	555 7	7	451	4	275	225	1	68	139
Hawaii	11	-	3	-	8	4	17	-	13 18	2	7 60	6 236	:	28	2 70
Guam	-	-	-		1	2		_	3	-		200			
P.R.	2	-	226	-	:	771	12		10	:	15	20	:	1	1 3
V.I. Amer. Samoa	-	-	-	-	-	•	-	-	33	-				-	1
C.N.M.I.	1	-	-	:	-	1	3	-	3	-	-	-	-	-	•
	•	-	•	-	-	-	1	-	2	-	-	-	-	-	-

TABLE III. (Cont'd.) Cases of specified notifiable diseases, United States, weeks ending December 3, 1988 and December 5, 1987 (48th Week)

*For measles only, imported cases includes both out-of-state and international importations. N: Not notifiable

U: Unavailable [†]International [§]Out-of-state

Reporting Area		(Civilian) Secondary)	Toxic- shock Syndrome	Tuber	culosis	Tula- remia	Typhoid Fever	Typhus Fever (Tick-borne) (RMSF)	Rabies, Animal
	Cum. 1988	Cum. 1987	Cum. 1988	Cum. 1988	Cum. 1987	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988
UNITED STATES	37,175	32,694	317	19,472	19,757	170	361	605	3,958
NEW ENGLAND	1,121	586	24	506	589	4	35	12	15 1
Maine N.H.	12 6	1 3	4 5	20 11	22 18	:		-	5
Vt.	3	4	2	4	15		1	2	-
Mass. R.I.	412 31	282 12	10	298 39	324 58	3	21 6	7 2	-
Conn.	657	284	3	134	152	1	7	3	9
MID. ATLANTIC	8,875	6,029	48	4,034	3,605	-	70	19	459
Upstate N.Y. N.Y. City	548 6,119	240 4,466	22 6	503 2,218	474 1,778	-	15 42	11 6	43
N.J.	918	666	3	668	641	-	11	-	14
Pa.	1,290	657	17	645	712	-	2	2	402
E.N. CENTRAL	1,092	808 101	46 31	2,139 412	2,181 389	1	34 8	34 22	143 5
Ohio Ind.	104 49	56	1	222	228		2	2	29
111.	483	408	1	929	987	:	18	7	31
Mich. Wis.	423 33	188 55	13	480 96	487 90	1	4 2	2 1	35 43
W.N. CENTRAL	222	171	44	485	567	77	4	91	429
Minn.	18	20	5	85	112	3	2	2	125
lowa	23	26	7	53	38	- 47	2	- 55	13 21
Mo. N. Dak.	146 1	78 1	12	234 15	308 13	4/	2		102
S. Dak.	:	11	4	33	24	16	-	7	112
Nebr. Kans.	28 6	15 20	4 9	14 51	25 47	3 7	-	1 26	19 37
S. ATLANTIC	13,363	11,187	19	4,144	4,215	5	43	198	1,372
Del.	96	66	13	38	39	2	-	1	57
Md.	651	579	3	390	361	-	3	22	307 13
D.C. Va.	652 402	383 306	-	177 378	145 402	2	12	17	341
W. Va.	37	13	-	68	96	-	1	2	96
N.C. S.C.	765 698	670 668	9 3	479 438	534 430	-	2	107 23	8 121
Ga.	2,389	1,556	-	681	760	1	8	23	283
Fla.	7,673	6,946	3	1,495	1,448	-	14	3	146
E.S. CENTRAL	1,859	1,764	24	1,592	1,785	11	3 1	91 30	277 113
Ky. Tenn.	63 796	23 699	10 10	343 476	407 544	5 5	-	39	69
Ala.	533	465	3	481	509	-	1	10	88
Miss.	467	577	1	292	325	1	1	12	7
W.S. CENTRAL	4,018	4,089	29 2	2,468 284	2,305 277	53 34	8	144 31	508 84
Ark. La.	237 803	233 759	-	311	285		4	2	10
Okla.	137	169	9	229	224	16 3	4	93 18	33 381
Tex.	2,841	2,928	18	1,644	1,519	-			
MOUNTAIN Mont.	788 3	659 9	35	539 31	599 16	11	11 1	12 6	350 196
Idaho	4	5	5	19	29	-	-	2	11
Wyo. Colo.	1 99	3 115	3	5 74	2 146	2 5	3	3 1	38 28
N. Mex.	99 47	54	2	90	94	2	1	-	11
Ariz.	157	284	16	232	255	1	6	-	41
Utah Nev.	16 461	23 166	9	29 59	25 32	1	-	-	9 16
PACIFIC	5,837	7,401	48	3,565	3,911	8	153	4	405
Wash.	196	153	8	212	227	1	14	1	-
Oreg.	284 5 314	280 6,950	1	135	121 3,323	1 4	7 126	1 2	384
Calif. Alaska	5,314 15	6,950	38	3,018 44	3,323 58	2	120	-	384 21
Hawaii	28	14	1	156	182	-	5	-	-
Guam	3	2	-	30	26	-	:	-	-
P.R. V.I.	625 2	832 9	-	219 6	277 2	-	5	-	66
Amer. Samoa	-	-	-	4	10	-	1	-	-
C.N.M.I.	1	-	-	24	-	-	-	-	-

TABLE III. (Cont'd.) Cases of specified notifiable diseases, United States, weeks ending December 3, 1988 and December 5, 1987 (48th Week)

U: Unavailable

	T	All Ca	uses, B	y Age	(Years)		TT		<u> </u>		18.05 0	y Age	Vearel		r
Reporting Area	All	≥65	T I	25-44	1-24	<1	P&i** Total	Reporting Area	All	>65	45-64	1	1-24	<1	P&I** Total
	Ages								Ages	200	40-04	20-44	1-24		Total
NEW ENGLAND Boston, Mass.	754 187	499 108	145 42	60 19	25 7	25	62	S. ATLANTIC	1,260	762	274	135	51	37	58
Bridgeport, Conn.	56	40	42	6	1	11	29 2	Atlanta, Ga.	169	94	39	26	7	3	5
Cambridge, Mass.	27	22	3	2	-	-	5	Baltimore, Md. Charlotte, N.C.	165 102	89 53	45 32	20 13	4 3	7	5 8
Fall River, Mass.	32	24	4	2	2	-	2	Jacksonville, Fla.	152	94	24	20	8	6	Š
Hartford, Conn. Lowell, Mass.	64 26	33 17	16 8	9	4	2 1	1	Miami, Fla.	97	51	24	16	5	1	-
Lynn, Mass.	22	19	ĭ	2	-		-	Norfolk, Va. Richmond, Va.	87 84	57 54	18	6	3	3	9
New Bedford, Mass.	33	26	5	1	1	-	-	Savannah, Ga.	47	35	14 8	10 2	5 2	1	7
New Haven, Conn. Providence, R.I.	66 83	42 60	8 20	5 3	6	5	2	St. Petersburg, Fla.	113	95	10	ĩ	4	3	9
Somerville, Mass.	5	4	20	-		2	3	Tampa, Fla.	72	45	14	4	4	4	3
Springfield, Mass.	46	25	11	4	2	4	5	Washington, D.C. Wilmington, Del.	153 19	86 9	39 7	16 1	4	8	5
Waterbury, Conn.	41	31	8	2	:	-	4	E.S. CENTRAL	688					-	•
Worcester, Mass.	66	48	10	5	2	1	7	Birmingham, Ala.	116	451 79	148 22	50 6	14 1	25 8	40 2
MID. ATLANTIC Albany, N.Y.	2,785 58	1,827 43	542	276	78	61	168	Chattanooga, Tenn.	52	36	11	3	ź		. 3
Allentown, Pa.	24	43	8	1	3 1	3	3	Knoxville, Tenn.	60	39	10	8	-	3	5
Buffalo, N.Y.§	106	79	19	5	2	1	8	Louisville, Ky. Memphis, Tenn.	77 168	50 114	21 34	4	1	1	3
Camden, N.J.	44	25	11	1	6	1	-	Mobile, Ala.	39	24	34	12 4	4	4	15 1
Elizabeth, N.J. Erie, Pa.t	22 47	16 36	17	1	3 2	1	6 4	Montgomery, Ala.	31	22	7	1	-	i	i
Jersey City, N.J.§	59	40	11	6	1	i	4	Nashville, Tenn.	145	87	35	12	4	7	10
N.Y. City, N.Y.	1,449	958	260	172	33	26	76	W.S. CENTRAL	1,845	1,165	410	165	65	40	68
Newark, N.J. Paterson, N.J.	106 32	45 11	32 9	18	8	3	4	Austin, Tex. Baton Rouge, La.	64 20	45	14	2	2	1	3
Philadelphia, Pa.	306	170	82	8 33	2 11	2 9	1 21	Corpus Christi, Tex.§		13 37	10	2 1	1	-	1
Pittsburgh, Pa.†	88	61	20	°3	2	2		Dallas, Tex.	165	97	43	11	7	7	5
Reading, Pa.	37	24	8	4	1	-	4	El Paso, Tex.	103	64	28	8	1	2	11
Rochester, N.Y. Schenectady, N.Y.	130 20	104 15	20 4	3 1	-	3	19	Fort Worth, Tex Houston, Tex.§	95 734	64 436	18 169	5 89	6 24	2 16	4
Scranton, Pa.†	35	29	5	i		-	8	Little Rock, Ark.	77	430	22	4	24 5	10	18
Syracuse, N.Y.	117	77	24	9	2	5	5	New Orleans, La.	134	87	26	15	4	ź	1
Trenton, N.J. Utica, N.Y.	46	28	11	3	1	3	4	San Antonio, Tex.	177	116	29	18	9	5	6
Yonkers, N.Y.	20 39	15 32	4	1	-	1	3	Shreveport, La. Tulsa, Okla.	93 135	62 99	20 27	5 5	3 3	3	8 10
E.N. CENTRAL	2.551	1,647	555	185	65	97	114	MOUNTAIN	802	501	190	52	29	30	41
Akron, Ohio	84	60	18	3		3	2	Albuquerque, N. Me	x. 98	62	18	13	2	3	-
Canton, Ohio	36	29	5	2	-	-	3	Colo. Springs, Colo.	42	25	10	4	1	2	3
Chicago, III.§ Cincinnati, Ohio	564 147	362 102	125 27	45 4	10	22	16	Denver, Colo. Las Vegas, Nev.	144 129	90 75	31 43	13 7	6 2	4	6
Cleveland, Ohio	184	100	46	23	8 4	6 11	12 9	Ogden, Utah	32	25	4	ź	1		8 5
Columbus, Ohio	127	73	32	11	3	8	ĭ	Phoenix, Ariz.	167	97	42	4	10	14	8
Dayton, Ohio Detroit, Mich.	136 348	98 181	25	9	4		5	Pueblo, Colo. Salt Lake City, Utah	31 38	22 17	8 15	1	-		1
Evansville, Ind.	348 79	61	95 6	37 5	21 1	12 6	9 1	Tucson, Ariz.	121	88	19	1	2 5	3 2	3 7
Fort Wayne, Ind.	87	61	16	7	i	2	4	PACIFIC	2.088	1,375	404	, 179	74	49	118
Gary, Ind. Grand Banida, Mich	13	7	3	3	-	-	1	Berkeley, Calif.	18	1,375	404	1/9	/4	49	118
Grand Rapids, Mich. Indianapolis, Ind.	54 163	37 100	11 34	3	1	2	2	Fresno, Calif.	116	72	21	9	7	7	8
Madison, Wis.	43	29	- 34	13 3	5	11 2	3	Glendale, Calif.	21	18	2	1	-	-	-
Milwaukee, Wis.	160	115	32	5	4	4	11	Honolulu, Hawaii Long Beach, Calif.	74 103	41 70	25 16	3 12	3 3	2	8 9
Peoria, III. Rockford, III.	58	39	12	2	1	4	7	Los Angeles Calif.	492	321	89	52	19	4	16
South Bend, Ind.	53 53	39 37	9 13	4	-	1	6 4	Oakland, Calif.	73	47	17	4	2	3	5
Toledo, Ohio	118	85	26	2 4	2	1	12	Pasadena, Calif. Portland, Oreg.	35 124	28 87	2 23	1	4	-	3
Youngstown, Ohio	44	32	11		-	i	-	Sacramento, Calif.	124	125	23	10 14	2 6	2 5	5 19
W.N. CENTRAL	1,012	717	179	70	28	18	41	San Diego, Calif.	144	84	35	9	10	6	10
Des Moines, Iowa	104	80	15	5	20	1	9	San Francisco, Calif.	192	115	42	29	2	4	5
Duluth, Minn. Kansas City, Kans.	44 39	32	9	3	-	-	-	San Jose, Calif.	183 234	123	35	14	7	4	14 4
Kansas City, Mo.	110	23 77	11 20	2	2	1	1	Seattle, Wash. Spokane, Wash.	234	159 43	43 12	15 3	8 1	9 1	4
Lincoln, Nebr.	57	45	20 8	9 3	4	-	8 1	Tacoma, Wash.§	35	27	6	2			3
Minneapolis, Minn.	236	162	43	16	6	9	16		13,785*1				429	382	710
Omaha, Nebr. St. Louis, Mo.	114 159	78	20	10	4	2	3		.5,705	3,344	-,0-/	.,	423	302	/ 10
St. Paul, Minn.	89	107 71	35 10	12	4	1	:								
Wichita, Kans.	60	42	8	5 5	1 3	2	1								
				5	5	-	-								

TABLE IV. Deaths in 121 U.S. cities,* week ending December 3, 1988 (48th Week)

*Mortality data in this table are voluntarily reported from 121 cities in the United states, most of which have populations of 100,000 or more. A death is reported by the place of its occurrence and by the week that the death certificate was filed. Fetal deaths are not included

**Pneumonia and influenza.

Theoremonia and infruenza.
 TBecause of changes in reporting methods in these 3 Pennsylvania cities, these numbers are partial counts for the current week.
 Complete counts will be available in 4 to 6 weeks.
 tTotal includes unknown ages.
 \$Data not available. Figures are estimates based on average of past available 4 weeks.

Screening Tests - Continued

USE OF HTLV-I SCREENING TESTS IN BLOOD BANKS

The FDA recommends that whole blood and cellular components donated for transfusion be screened for HTLV-I antibody using a licensed EIA screening test. The FDA further recommends that units that are repeatably reactive by EIA be quarantined, then destroyed, unless otherwise stipulated by the FDA. Source plasma (obtained from plasma donors) intended for use in further manufacturing need not be screened for HTLV-I antibody.

DONOR DEFERRAL AND NOTIFICATION

FDA recommends permanent deferral of donors whose sera are repeatably reactive in EIA and confirmed as positive for HTLV-I antibody by additional, more specific testing. Such donors should be notified and counseled accordingly.

Donors whose serum specimens are repeatably reactive in the EIA but not confirmed as positive for HTLV-I antibody need not be notified on the first occasion. Although the donated units must be destroyed, the donors remain eligible for future donation. If, however, the donors test repeatably reactive in the EIA on a subsequent donation, they should be deferred indefinitely as donors and notified and counseled accordingly.

GUIDELINES FOR COUNSELING

Counseling should be considered a routine adjunct depending on the results of HTLV-I testing. Given some of the uncertainties related to testing, e.g., the inability to distinguish between antibodies to HTLV-I and HTLV-II, and the low probability that disease will occur in seropositive persons, every effort should be made to minimize the anxiety provoked by a repeatably reactive screening test, particularly one that is not confirmed as HTLV-I-seropositive by additional testing.

Persons confirmed as seropositive for HTLV-I should be notified that they have antibody to HTLV-I and are likely infected with HTLV-I or HTLV-II. They should be given information concerning disease associations and possible modes of transmission. In addition, they should be advised that they have been permanently deferred as blood donors and should neither give blood for transfusion nor share needles that have been used for percutaneous injection or infusions with other persons. Breastfeeding of infants should be discouraged. The paucity of data concerning sexual transmission of HTLV-I/HTLV-II does not permit a firm recommendation concerning sex practices; specific recommendations, such as the use of condoms to reduce the potential risk of sexual transmission, should be developed in consultation with a health-care professional.

Persons whose serum specimens are repeatably reactive on more than one occasion in the EIA but not confirmed as positive for HTLV-I antibody in more specific testing should be informed that they have inconclusive test results that do not necessarily imply infection with HTLV-I or HTLV-II. Nevertheless, they should be notified that they have been deferred indefinitely as donors and should not donate blood for transfusion. Periodic follow-up of such donors with EIA, more specific serologic tests, and possibly sophisticated techniques such as virus isolation and/or PCR may provide more reliable information regarding the presence of viral infection. *Reported by: Public Health Service Working Group.*[†]

[†]D Anderson, MD, J Epstein, MD, L Pierik, J Solomon, PhD, Food and Drug Administration. W Blattner, MD, C Saxinger, PhD, National Cancer Institute; H Alter, MD, H Klein, MD, Clinical Center; P McCurdy, MD, G Nemo, MD, National Heart, Lung, and Blood Institute, National Institutes of Health. J Kaplan, MD, J Allen, MD, R Khabbaz, MD, M Lairmore, PhD, CDC.

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Progress in Chronic Disease Prevention

Years of Potential Life Lost due to Cancer - United States, 1968-1985

Although the incidence of cancer is relatively low in persons <65 years of age (82.9 deaths per 100,000 persons in this age group in 1985), it is the second leading cause of years of potential life lost (YPLL) for this age group, exceeded only by injuries. In 1985, 1,952,171 YPLL were attributable to cancer*. This is essentially unchanged (a 0.7% increase) from the YPLL for 1984. In 1985, white males contributed 43.1% of the cancer-attributable YPLL; white females, 40.1%; black males, 7.9%; and black females, 7.0%. Males and females of other races account for the remaining 1.9%.

Between 1968 and 1985, cancer mortality rates in persons <55 years of age declined 23% from 43 to 35 deaths per 100,000 persons in this age group, while rates in those \geq 55 years of age increased 17% from 775 to 905 per 100,000 persons (1). During this period, total YPLL remained relatively constant, with an average annual decline of <1%. However, the age-adjusted rate of cancer-attributable YPLL for the total population steadily decreased from 1968 to 1985 (Table 1), reflecting the overall decline in cancer mortality in younger persons.

Rates of cancer-attributable YPLL in 1985 were age-adjusted by race-gender groups: the highest rate occurred for black males (1208.1 per 100,000), followed by white males (949.4), black females (876.7), and white females (840.5). The rates in all four major race-gender groups also declined differentially (Figure 1). The average annual decline between 1968 and 1985 was approximately twice as great for black females (a decline of 18.9 per 100,000 per year) as for black males (9.4), white females (9.6), or white males (9.9).

Reported by: Div of Chronic Disease Control and Community Intervention, Center for Chronic Disease Prevention and Health Promotion, CDC.

Editorial Note: As life expectancy increases and all causes of death in earlier years of life decrease, mortality patterns and public health priorities may change. The patterns for cancer mortality and YPLL illustrate the complex shifts that may alter perception of the importance of cancer in young persons. Declines in mortality from infectious diseases

^{*}This report examines cancer mortality and YPLL for all mentions of cancer on death certificates, using multiple cause of death tapes from the National Center for Health Statistics. Cancer is selected as the underlying cause of death on 88% of death certificates mentioning cancer as a cause of death. Cancer-attributable YPLL is computed using differences between age at death from cancer and 65 years.

YPLL – Continued

and major chronic conditions such as cardiovascular disease in younger persons have increased the relative public health burden of cancer mortality.

YPLL reflects both the rate of disease and the size of the population at risk. Although the rate of cancer in younger persons, particularly those <55 years, is decreasing, some of the largest population increases by age group occur for persons 30–50 years of age, the result of higher birth rates during 1946–1964. Because cancer rates have decreased while the size of the population at risk has increased, virtually no change has occurred in the annual total number of cancer-attributable YPLL from 1968 to 1985. Thus, cancer-attributable YPLL has produced a constant disease burden.

Age-adjusted YPLL rates (Table 1) show an overall downward trend, however, reflecting diminishing cancer mortality rates in persons <55 years of age. This downward trend, which occurred in each of the four largest race-gender groups (Figure 1), is most prominent for black women, whose decrease is twice that of the other groups. This decrease does not appear to be attributable to greater population growth among black women, since their growth rate is identical to that of black men, whose decline in age-adjusted YPLL was the smallest of the four groups. Instead, the differential decline in age-adjusted YPLL appears to be related to a complex interaction between cancer incidence, mortality, and survival; this interaction may vary by tumor sites for different segments of the population. Since the number of cancer

Year	YPLL*	Age-adjusted rate [†]
1968	1,958,714	1,073.6
1969	1,953,236	1,062.0
1970	1,970,612	1,065.0
1971	1,960,541	1,048.1
1972	1,952,028	1,033.6
1973	1,956,137	1,027.5
1974	1,952,977	1,017.5
1975	1,919,724	993.6
1976	1,920,902	986.3
1977	1,930,981	983.4
1978	1,921,543	969.9
1979	1,906,684	953.8
1980	1,932,719	959.0
1981	1,906,013	939.0
1982	1,924,103	939.6
1983	1,921,549	929.3
1984	1,938,046	928.0
1985	1,952,171	925.9

TABLE 1. Cancer-attributable years of potential life lost before age 65 years (YPLL) and age-adjusted YPLL rates – United States, 1968–1985

*Calculated by multiplying the age-specific disease rate by population by the number of years between death and age 65. Deaths that occur after age 65 do not contribute to the total. *Rates are per 100,000 persons <65 years and are computed by applying age-specific rates for YPLL (i.e., YPLL/population for each age stratum) to a standard population. Here, the U.S. population structure under age 65 for 1980 was the standard.

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YPLL – Continued

cases occurring in certain population groups may be small, data are insufficient to address such interactions (1,2).

Although the decline in age-adjusted rates for cancer-attributable YPLL is encouraging, understanding the basis and public health implications of this decline requires further investigation. These efforts may need to focus on cancer incidence, mortality, and survival among younger population subgroups and on the relative impact of these measurements and programs designed to affect them (3).

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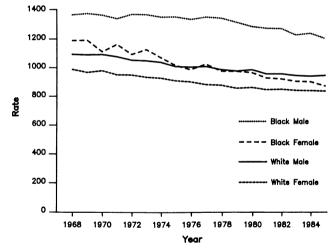


FIGURE 1. Age-adjusted rate of years of potential life lost due to cancer per 100,000 persons by race-gender group – United States, 1968–1985

Notice to Readers

MMWR Auxiliary Publications

The following documents have been published as part of *MMWR* Vol. 37. For information regarding purchase of these documents, contact the U.S. Government Printing Office (telephone [202] 783–3238 or MMS Publications (telephone [617] 893–3800)... For additional questions, contact Editorial Services, Epidemiology Program Office, CDC (telephone [404] 639–2100).

Publications - Continued

Supplements:

Guidelines for the Prevention and Control of Congenital Syphilis (Vol. 37, No. S-1, January 15, 1988).

Guidelines for Effective School Health Education to Prevent the Spread of AIDS (Vol. 37, No. S-2, January 29, 1988).

Management of Patients with Suspected Viral Hemorrhagic Fever (Vol. 37, No. S-3, February 26, 1988).

1988 Agent Summary Statement for Human Immunodeficiency Virus and Report on Laboratory-Acquired Infection with Human Immunodeficiency Virus (Vol. 37, No. S-4, April 1, 1988).

Guidelines for Evaluating Surveillance Systems (Vol. 37, No. S-5, May 6, 1988).

CDC Recommendations for a Community Plan for the Prevention and Containment of Suicide Clusters (Vol. 37, No. S-6, August 19, 1988).

NIOSH Recommendations for Occupational Safety and Health Standards 1988 (Vol. 37, No. S-7, August 26, 1988).

CDC Surveillance Summaries:

Public Health Surveillance of 1990 Injury Control Objectives for the Nation (Vol. 37, No. SS-1, February 1988)

- Introduction: Moving from the 1990 Injury Control Objectives to State and Local Surveillance Systems;
- Deaths from Motor Vehicle-Related Injuries, 1978-1984;
- Deaths due to Injury in the Home among Persons under 15 Years of Age, 1970–1984;
- Deaths from Falls, 1978-1984;
- Drownings in the United States, 1978-1984;
- Hospitalizations due to Tap Water Scalds, 1978-1985;
- Deaths from Residential Fires, 1978-1984;
- Unintentional Firearm-Related Fatalities, 1970-1984;
- Homicides among Black Males 15-24 Years of Age, 1970-1984;

Suicides among Persons 15–24 Years of Age, 1970–1984.

- Vol. 37, No. SS-2, June 1988
 - Campylobacter Isolates in the United States, 1982-1986;
 - Water-Related Disease Outbreaks, 1985;

Salmonella Isolates from Humans in the United States, 1984–1986.

- Reports on Selected Racial/Ethnic Groups (Vol. 37, No. SS-3, July 1988)
 - Distribution of AIDS Cases, by Racial/Ethnic Group and Exposure Category, United States, June 1, 1981–July 4, 1988;
 - Plague in American Indians, 1956-1987;
 - Leading Major Congenital Malformations among Minority Groups in the United States, 1981–1986;
 - Differences in Death Rates due to Injury among Blacks and Whites, 1984;

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Publications - Continued

Dental Caries and Periodontal Disease among Mexican-American Children from Five Southwestern States, 1982–1983.

Rabies Surveillance, United States, 1987 (Vol. 37, No. SS-4, September 1988).

Clarification: Vol. 37, No. 47

p. 718 In the article, "HIV-Related Beliefs, Knowledge, and Behaviors among High School Students," the second sentence of the second full paragraph should be clarified to read: "Because response rates of schools from some sites were less than 100%, results from these sites cannot be generalized...."

Errata: Vol. 37, No. 42

- p. 654 In the article, "Human Plague United States, 1988," the vaccine dose given to the patient in case 7 is incorrect. The dose given in the first sentence under case 7 should be 1.0 mL.
- p. 656 Reference 3 should be: ACIP. Plague vaccine. MMWR 1982;31:301-4.

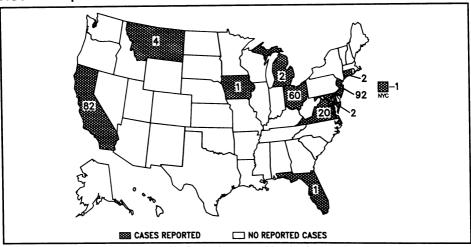


FIGURE I. Reported measles cases - United States, Weeks 44-47, 1988

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- 733 Preliminary Report: 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin Exposure to Humans – Seveso, Italy
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- 747 Years of Potential Life Lost due to Cancer – United States, 1968–1985
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International Notes

Preliminary Report: 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin Exposure to Humans – Seveso, Italy

At approximately noon on Saturday, July 10, 1976, an explosion occurred during the production of 2,4,5-trichlorophenol in a factory in Meda, about 25 km north of Milan, in the Lombardia region of Italy. A cloud of toxic material was released and included 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). Debris from this cloud fell south-southeast of the plant on an area of about 2.8 km² (700 acres), including parts of the towns of Seveso, Meda, Cesano Maderno, and Desio. The size of the contaminated area was estimated primarily by measuring TCDD in the soil; additional criteria included the presence of dead animals (e.g., birds, rabbits, chickens) and detection of dermal lesions among persons in the area. The contaminated area was divided into three zones (A, B, and R) depending on the concentration of TCDD in the soil (Table 1). An additional zone, Zone S, outside the contaminated area was examined as a control zone.* Zone A, the most heavily contaminated section, was further divided into seven subzones, A1–A7, based on increasing distance from the factory. The total amount of TCDD deposited in the contaminated area was initially estimated at about 165 g (1); subsequently, it has been estimated to be at least 1.3 kg (2).

Within 20 days of the explosion, the Italian authorities had evacuated the 211 families (735 persons) from the area later defined as Zone A and had taken immediate measures to minimize the risk of exposure to residents in nearby areas (primarily

*Zone R was originally thought to be a reference zone, but on subsequent detection of TCDD
concentrations in soil, Zone S was added.

		Size	Soil sampling	TCDD concentration (µg/r			
Zone	Population	(acres)	square (m²)	Mean	Highest		
A	735	198	2,500	230.0	5477.0		
в	4,699	665	22,500	3.0	43.9		
R	31,800	3,532	22,500	0.9	9.7		

TABLE 1. Distribution* of TCDD contamination in soil[†] – Seveso, Italy, 1976

*These data may be underestimated by approximately 25% (2).

[†]Measured in a soil sample, 20 cm of depth, taken at the center of a square.

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TCDD - Continued

those in Zone B). The Italian authorities were assisted by several national and international technical commissions in assessing adverse health effects. Residents of zones A, B, and R underwent extensive medical examinations from 1976 to 1985; chloracne, detected in a small segment of the population, was the only abnormal finding (3–6). Only one potentially exposed person was measured for TCDD; she was a 55-year-old woman residing in a portion of Zone A (mean TCDD soil concentration of 185.4 μ g/m²), who died from pancreatic adenocarcinoma 7 months after the explosion. Her TCDD whole-weight levels varied from 6 parts per trillion (ppt) in blood to 1840 ppt in adipose tissue[†] (7).

In April 1988, a group of U.S. and Italian scientists convened to further examine the Seveso TCDD incident. Since more than 30,000 serum or plasma samples (volumes of 1–3 mL) had been collected from residents of the four zones from the end of July 1976 through 1985 and stored at –30 C (–22 F), the group agreed to assess whether methodology developed at CDC to measure TCDD in human serum (8) could be used to measure TCDD in these low-volume samples. This methodology, a lipid-based measurement highly correlated with paired measurements of TCDD in adipose tissue (p = 0.98) (9), has been used to evaluate U.S. Army veterans (10), U.S. Air Force Operation Ranch Hand veterans (11), and occupationally exposed persons (D.G. Patterson, Jr., et al., unpublished data).

The preliminary Seveso study evaluated serum samples from five Zone A residents who developed the most severe types (III or IV) of chloracne; four Zone A residents who did not develop chloracne or other health problems (in 1976, each was \geq 15 years of age); and five persons from Zone S. All these samples had been collected in 1976 and were sent without identification to CDC for analysis. These samples were analyzed for TCDD (8) on both a whole-weight and a lipid basis, using triglycerides and total cholesterol data provided for those samples by the laboratory of the hospital of Desio-Milan to calculate total lipids.

The TCDD levels detected are the highest ever reported in humans (Figure 1). The three highest levels are from children who developed chloracne. Levels for the other two chloracne cases were similar to those in residents without chloracne. TCDD was not detected in four of the five controls. In one control, a level of 137 ppt TCDD on a lipid basis was detected; this value may represent either an actual level or the detection of a residue of <1% from a sample analyzed immediately before this sample.

Reported by: P Mocarelli, MD, Institute of General Pathology, Univ of Milan and Hospital of Desio, Milan; F Pocchiari, PhD, Instituto Superiore di Sanita, Rome. N Nelson, PhD, New York Univ Medical Center, New York, New York. Center for Environmental Health and Injury Control, CDC.

Editorial Note: Little is known about TCDD exposures and adverse health effects in humans. However, these Seveso samples are unique in that they were taken in proximity of time to an acute human exposure to TCDD. Thus, they and the subsequent samples allow for correlating TCDD levels and adverse health effects, if any, and for determining the half-life of TCDD in humans. This population has no apparent adverse health effects other than chloracne (*4*).

These serum measurements confirm overt exposure to TCDD in those persons tested who resided in Zone A. The levels are of the same magnitude as those found in occupational studies (12,13) that estimate initial TCDD levels by extrapolating to

*Levels of TCDD in serum or adipose tissue of the general population are <20 ppt.

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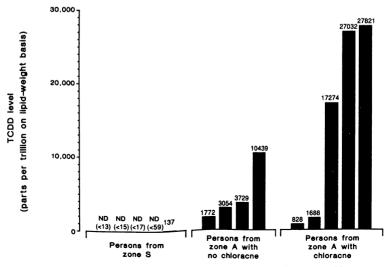
TCDD - Continued

the time of last exposure (by assuming first-order kinetics and a half-life of 7 years) (14). Although the three highest TCDD serum levels occurred in persons who developed chloracne, no threshold level for chloracne is obvious.

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FIGURE 1. Serum TCDD levels* of Zone A residents (with and without chloracne) and Zone S controls – Seveso, Italy



*Levels of TCDD in serum or adipose tissue of the general population are <20 ppt. ND=Nondetectable at specified levels.

TCDD – Continued

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Current Trends

Licensure of Screening Tests for Antibody to Human T-Lymphotropic Virus Type I

Screening tests for antibody to human T-lymphotropic virus type I (HTLV-I), the first-recognized human retrovirus, have been licensed by the Food and Drug Administration (FDA). These tests have been recommended by the FDA for screening of blood and cellular components donated for transfusion. They have also been approved as diagnostic tests, which may be useful in evaluating patients with clinical diagnoses of adult T-cell leukemia/lymphoma (ATL) and tropical spastic paraparesis (TSP)/HTLV-I-associated myelopathy (HAM), both of which have been associated with HTLV-I infection. Because licensure will probably result in widespread use of these tests, the information presented below is provided for physicians and public health officials who may need to interpret HTLV-I test results and to counsel persons whose serum specimens are reactive in these tests. Users of the new HTLV-I screening tests are cautioned that additional, more specific tests are necessary to confirm that serum specimens that are repeatably reactive in these screening tests are truly positive for HTLV-I antibody. Users should also be aware that neither the screening tests nor more specific tests can distinguish between antibody to HTLV-I and antibody to the closely related human retrovirus, human T-lymphotropic virus type II (HTLV-II).

HTLV-I does not cause AIDS, and the finding of HTLV-I antibody in human blood does not imply infection with human immunodeficiency virus (HIV) or a risk of developing acquired immunodeficiency syndrome (AIDS).

BACKGROUND: HTLV-I

HTLV-I was isolated in 1978 and first reported in 1980 (1). Although a member of the family of retroviruses, HTLV-I is *not* closely related to HIV, the virus that causes AIDS. HTLV-I does not cause depletion of T-helper lymphocytes, and it is not generally associated with immunosuppression.

Screening Tests - Continued

HTLV-I differs from HIV in its morphologic and genetic structure and in that HTLV-I antigens should not cross-react with the antigens of HIV. The HTLV-I genome contains four major genes: *gag*, which encodes core proteins of 15,000 (p15), 19,000 (p19), and 24,000 (p24) daltons; *pol*, which encodes a polymerase (reverse transcriptase) protein of 96,000 daltons; *env*, which encodes envelope glycoproteins of 21,000 (gp21) and 46,000 (gp46) daltons; and *tax*, which encodes a transactivator protein of 40,000 daltons (p40x).

Seroprevalence

HTLV-I infection is endemic primarily in southwestern Japan, the Caribbean, and some areas of Africa (2). Seroprevalence in well-characterized areas appears to increase with patient age, with rates in females markedly higher than those in males beginning in the 20–30-year age range. Seroprevalence rates as high as 15% in the general population and 30% in older age groups have been reported in some areas of Japan (3). In the Caribbean islands, rates may be as high as 5% in the general population and 15% in older age groups (4).

In the United States, HTLV-I infection has been identified mainly in intravenousdrug users (IVDUs), with seroprevalence rates ranging from 7% to 49% (5,6). Elevated rates have also been reported in female prostitutes (in whom IV-drug use is a major risk factor) (7) and in recipients of multiple blood transfusions (8). Seropositivity is rare among homosexual men and among patients in sexually transmitted disease clinics (9,10), and it appears to be nonexistent in hemophilic men without other risk factors (11). Systematic determination of HTLV-I seroprevalence in the general population of the United States has not been undertaken. However, in a study of 39,898 random blood donors in eight U.S. cities, 10 (0.025%) were seropositive for HTLV-I (12).

Transmission

Transmission of HTLV-I infection by blood transfusion is well documented in Japan, with a seroconversion rate of 63% in recipients of the *cellular* components of contaminated units (whole blood, red blood cells, and platelets) (13). Transmission by the plasma fraction of contaminated units has not resulted in infection; this finding has been attributed to the fact that HTLV-I is highly cell-associated. Transmission among IVDUs is presumed to occur by sharing of needles and syringes contaminated with infectious blood.

Transmission from mother to child occurs through breastfeeding; breastfed infants of seropositive mothers have an approximately 25% probability of becoming infected (14). However, infection has also occurred in infants who are not breastfed, suggesting that intrauterine and/or perinatal transmission may occur.

Sexual transmission of HTLV-I appears to be relatively inefficient (15). Transmission from male to female, however, appears to be more efficient than from female to male (16).

Disease Associations

HTLV-I has been etiologically associated with adult T-cell leukemia/lymphoma (ATL), a malignancy of mature T-lymphocytes characterized by skin lesions, visceral involvement, circulating abnormal lymphocytes, hypercalcemia, and lytic bone lesions (*17*). ATL has been recognized in Japan, the Caribbean, and Africa. No systematic attempt has been made to record cases of ATL in the United States, but 74 cases were reported to the National Institutes of Health between 1980 and 1987 (*18*). Approximately half of these cases occurred in persons of Japanese or Caribbean

ancestry; most of the remainder were in blacks from the southeastern United States. ATL tends to occur equally in men and women, with peak occurrence in persons 40–60 years of age.

It is thought that a person must be infected with HTLV-I for years to decades before ATL develops. The lifetime risk of ATL among HTLV-I–infected persons has been estimated to be approximately 2% in two studies in Japan (19,20). In Jamaica, the lifetime risk of ATL among persons infected before the age of 20 years was estimated to be 4% (21).

Because of the long latent period of ATL, the risk of this disease among persons infected by blood transfusion (many of whom are elderly and may not survive their underlying disease) is not thought to be substantial. In fact, no cases of ATL associated with blood transfusion have been reported.

HTLV-I has also been associated with a degenerative neurologic disease known as tropical spastic paraparesis (TSP) in the Caribbean and as HTLV-I–associated myelopathy (HAM) in Japan (22,23). TSP/HAM is characterized by progressive difficulty in walking, lower extremity weakness, sensory disturbances, and urinary incontinence. Although most cases have been reported from countries in which HTLV-I is endemic, a few cases have occurred in the United States (24). TSP/HAM occurs in persons of all age groups, with peak occurence in ages 40–49 years. Rates are higher in females than in males. The lifetime risk of TSP/HAM among persons infected with HTLV-I is unknown but appears to be very low. The latent period for this disease appears to be less than for ATL, and the disease probably can be caused by blood transfusion. Of 420 Japanese patients with HAM from whom information was available, 109 (26%) gave a history of blood transfusion; the mean interval between transfusion and onset of neurologic symptoms was estimated to be 4 years (M. Osame, unpublished data).

HTLV-I does not cause AIDS, and the finding of HTLV-I antibody in human blood does not imply infection with HIV or a risk of developing AIDS.

BACKGROUND: HTLV-II

HTLV-II is closely related to HTLV-I. The genome of HTLV-II encodes viral proteins that are similar to those of HTLV-I, and there is extensive serologic cross-reactivity among proteins from HTLV-I and HTLV-II.

No specific information is available regarding the seroepidemiology or the modes of transmission of HTLV-II. There is some evidence that some of the HTLV-I seropositivity in the United States, especially in IVDUs, may be caused by HTLV-II (5).

Two cases of disease have been associated with HTLV-II infection. HTLV-II was first isolated from a patient with a rare T-lymphocytic hairy cell leukemia (25). In the second case, HTLV-II was isolated from a patient who had the more common B-lymphocytic form of hairy cell leukemia and who also suffered from a T-suppressor lymphoproliferative disease (26). No serologic evidence of HTLV-II infection has been found in 21 additional cases of hairy cell leukemia (27). Thus, the disease associations of HTLV-II are unclear, and nothing is known regarding lifetime risk of disease among infected persons.

SEROLOGIC TESTS FOR HTLV-I

Interpretation

The screening tests that have been licensed by the FDA are enzyme immunoassays (EIAs) to detect HTLV-I antibody in human serum or plasma. Specimens with absorbance values greater than or equal to the cutoff value determined by the

manufacturer are defined as initially reactive. Initially reactive specimens must be retested in duplicate to minimize the chance that reactivity is due to technical error. Specimens that are reactive in either of the duplicate tests are considered repeatably reactive. Specimens that do not react in either of the duplicate repeat tests are considered nonreactive. Additional, more specific serologic tests are necessary to confirm that serum specimens repeatably reactive in the screening tests are positive for HTLV-I antibody. Users of the screening tests must have available to them additional, more specific tests to properly interpret repeatably reactive screening tests. Such tests are available in research institutions, industry, and some diagnostic laboratories. No such tests have been licensed by the FDA.

Tests used to confirm HTLV-I seropositivity must be inherently capable of identifying antibody to the core (gag) and envelope (env) proteins of HTLV-I. (The immunoreactivities of the polymerase [pol] and transactivator [tax] proteins of HTLV-I have not been well-defined in current test systems.) Specific tests include Western immunoblot (WIB) and radioimmunoprecipitation assay (RIPA). Indirect fluorescent antibody (IFA) testing for HTLV-I has been used in some laboratories, but IFA does not detect antibody to specific HTLV-I gene products.

WIB appears to be the most sensitive of the more specific tests for antibody to *gag* protein products p19, p24, and (*gag*-derived) p28, whereas RIPA appears to be most sensitive for antibody to the *env* glycoproteins gp46 and (*env* precursor) gp61/68. Based on experience with these tests in several laboratories, the following confirmatory criteria for HTLV-I seropositivity have been adopted by the Public Health Service Working Group: a specimen must demonstrate immunoreactivity to the *gag* gene product p24 and to an *env* gene product (gp46 and/or gp61/68) to be considered "positive." Serum specimens not satisfying these criteria but having immunoreactivities to at least one suspected HTLV-I gene product (such as p19 only, p19 and p28, or p19 and *env*) are designated "indeterminate." Serum specimens with no immunoreactivity to any HTLV-I gene products in additional, more specific tests are designated "negative." Both WIB and RIPA may be required to determine whether a serum specimen is positive, indeterminate, or negative.

Although additional, more specific tests have been somewhat standardized, the quantities and the molecular weights of HTLV-I proteins produced by various cell lines vary considerably. Hence, the cell of origin for HTLV-I antigens used in either WIB or RIPA, as well as the method of antigen preparation, may markedly influence test sensitivity and interpretation of immunoreactivity against individual HTLV-I proteins. Laboratories performing these tests, however, should be able to detect antibody to the *gag* and *env* gene products of HTLV-I in WIB and/or RIPA.

Sensitivity, Specificity, and Predictive Value

Using the WIB and RIPA available in research laboratories and the confirmatory criteria described above to define the presence of HTLV-I antibody, the sensitivities of the three EIAs that have been licensed by the FDA have been estimated from the performance of the tests on a reference panel of 137 antibody-positive serum specimens. All three EIAs were repeatably reactive for 137 of 137 panel serum specimens, yielding an estimated sensitivity of 97.3%–100% by the binomial distribution at 95% confidence. Specificity* of the EIAs was estimated for each test from

^{*}Specificity was calculated as follows: (total donations screened minus total number repeatably reactive in EIA) divided by (total donations screened minus number confirmed as positive by additional testing).

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Screening Tests - Continued

screening of at least 5000 normal U.S. blood donors in nonendemic areas. Estimated specificities ranged from 99.3% to 99.9% by the binomial distribution at 95% confidence. However, a specificity >99% but <100% may still yield a low positive predictive value when the screening test is used in a low-prevalence population. For example, in the study of U.S. blood donors cited above, 68 donors were repeat reactors in the screening test, but only 10 (15%) were determined to be HTLV-I-seropositive in more specific testing. This relatively low positive predictive value emphasizes the need for additional, more specific testing of specimens repeatably reactive in the EIA.

Neither the EIAs nor the additional, more specific tests can distinguish between antibodies to HTLV-I and HTLV-II. More sophisticated techniques, such as virus isolation and gene amplification (polymerase chain reaction [PCR]) are required to differentiate HTLV-I from HTLV-II infection.

	48	th Week End	ing	Cumulative, 48th Week Ending				
Disease	Dec. 3, 1988	Dec. 5, 1987	Median 1983-1987	Dec. 3, 1988	Dec. 5, 1987	Median 1983-1987		
Acquired Immunodeficiency Syndrome (AIDS)	215	U*	110	27,974	19,027	7,262		
Aseptic meningitis	152	163	173	6,295	10,548	10,083		
Encephalitis: Primary (arthropod-borne								
& unspec)	10	13	16	710	1,215	1,215		
Post-infectious	2	3	1	112	95	98		
Gonorrhea: Civilian	10,158	14,387	15,056	638,020	710,087	819,309		
Military	99	214	237	10,630	15,042	19,346		
Hepatitis: Type A	588	514	455	24,055	22,741	21,041		
Type B	406	474	474	20,767	23,417	23,737		
Non A, Non B	43	35	59	2,314	2,699	3,245		
Unspecified	59	62	103	2,171	2,863	4,746		
Legionellosis	13	10	17	905	880	713		
Leprosy	10	4	4	164	184	221		
Malaria	16	10	12	922	828	921		
Measles: Total [†]	26	10	15	2,743	3,548	2,704		
Indigenous	26	10	12	2,423	3,129	2,272		
Imported	-	-	1	320	419	304		
Meningococcal infections	31	44	44	2,562	2,676	2,461		
Mumps	129	224	67	4,288	11,859	3,044		
Pertussis	71	60	38	2,657	2,345	2,345		
Rubella (German measles)	1	5	5	189	329	601		
Syphilis (Primary & Secondary): Civilian	830	645	521	37,175	32,694	25,709		
Military	1	3	3	145	150	152		
Toxic Shock syndrome	2	8	8	317	311	345		
Tuberculosis	378	451	451	19,472	19,757	19,757		
Tularemia	-	1	3	170	186	186		
Typhoid Fever	5	19	4	361	333	345		
Typhus fever, tick-borne (RMSF)	11	4	4	605	586	731		
Rabies, animal	76	76	76	3,958	4,357	5,009		

TABLE I. Summary - cases of specified notifiable diseases, United States

TABLE II. Notifiable diseases of low frequency, United States

	Cum. 1988		Cum. 1988
Anthrax Botulism: Foodborne Infant Other (Oreg. 1) Brucellosis (Mich. 4) Cholera Congenital rubella syndrome Congenital syphilis, ages < 1 year Diphtheria	26 33 4 67 7 4 426	Leptospirosis (Fla. 1, Hawaii 1) Plague Poliomyelitis, Paralytic Psittacosis (Ariz. 1, Wash. 2, Calif. 2) Rabies, human Tetanus Trichinosis	43 14 1 86 - 48 40

Because AIDS cases are not received weekly from all reporting areas, comparison of weekly figures may be misleading.
[†]There were no cases of internationally imported measles reported for this week.

		Aseptic	Encer	ohalitis	Gond	orrhea	Н	epatitis (Viral), by	type	Logianat	
Reporting Area	AIDS	Menin- gitis	Primary	Post-in- fectious	(Civ	ilian)	A	В	NA,NB	Unspeci- fied	Legionel- Iosis	Leprosy
	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1987	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988
UNITED STATES	27,974	6,295	710	112	638,020	710,087	24,055	20,767	2,314	2,171	905	164
NEW ENGLAND	1,183	386	24	4	20,116	22,058	798	1,122	112	87	50	15
Maine N.H.	27 38	20 40	2		366	654	18	51	5	2	4	•
Vt.	10	29	17	3	241 110	373 203	45 15	69 53	11 6	4	5	•
Mass.	650	157	8	1	6,777	7,736	373	668	71	61	5 33	14
R.I.	81	87	-	-	1,884	2,003	84	80	11	-	3	1
Conn.	377	53	6	-	10,738	11,089	263	201	8	16	-	-
MID. ATLANTIC	9,303	700	53	4	100,706	111,902	1,847	2,994	181	302	208	8
Upstate N.Y. N.Y. City	1,256 5,070	374 134	34 8	1 3	14,832 41,750	16,415 59,753	701	710	72	19	78	-
N.J.	2,206	61	11	-	14,530	15,133	342 420	1,257 676	19 60	224 42	45 40	7
Pa.	771	131		-	29,594	20,601	384	351	30	17	40	1
E.N. CENTRAL	1,993	1,038	182	13	108,086	108,894	1,603	2,198	205	123	229	7
Ohio	467	414	62	3	24,444	24,859	313	513	35	123	83	
Ind.	80	96	28	-	8,252	8,789	152	326	19	28	27	-
01.	924	92	32	10	32,315	31,217	552	475	74	34	21	6
Mich.	417	389	43	-	34,568	34,588	374	628	53	39	58	-
Wis.	105	47	17	-	8,507	9,441	212	256	24	3	40	1
W.N. CENTRAL	684	253	55	12	27,416	28,556	1,291	931	100	34	74	1
Minn.	146	30	14	4	3,636	4,255	93	130	23	4	4	-
lowa	39	36	9 1	3	2,057	2,791	44	77	13	2	18	-
Mo. N. Dak.	364	105 5	4	-	15,807 175	15,231 270	787 6	556	44 3	18	23	-
S. Dak.	7	18	5	2	450	561	27	14	3	5	1 14	-
Nebr.	34	11	12	2	1,416	1,877	46	40	2		5	-
Kans.	90	48	10	ī	3,875	3,571	288	110	12	5	9	1
S. ATLANTIC	4,959	1,364	103	40	180,346	185,789	2,232	4,334	359	332	135	
Del.	62	43	3		2,849	3,167	44	128	303	332	135	1
Md.	497	194	10	3	18,661	21,420	273	662	40	26	20	1
D.C.	448	21	1	1	13,525	12,395	16	45	4	1	-1	
Va.	338	199	32	4	13,262	13,553	348	309	72	225	11	-
W. Va. N.C.	20 264	36 165	22 21	•	1,261	1,297	14	66	5	4	-	-
S.C.	167	21	21	1	25,686 14,431	27,962 14,192	327 40	782 500	87 12	-	31	-
Ga.	691	153	1	2	34,208	33,135	574	631	14	5 6	26 21	-
Fla.	2,472	532	13	29	56,463	58,668	596	1,211	117	61	12	:
E.S. CENTRAL	713	429	61	8	50.725	53,462	711	1,330	174			-
Ky.	89	145	21	1	5,177	5,359	466	265	61	13 2	48 20	2
Tenn.	324	48	15	-	17,597	18,830	157	604	40	-	8	:
Ala.	195	180	25	2	15,198	16,755	55	347	62	10	14	2
Miss.	105	56	-	5	12,753	12,518	33	114	11	1	6	-
W.S. CENTRAL	2,349	755	86	3	67,880	79,845	3,010	1,913	198	514	27	39
Ark.	76	15	6	-	6,804	9,007	323	104	6	17	4	
La.	340	117	24	1	13,698	13,158	152	332	25	16	7	7
Okia. Tex.	127 1,806	75 548	8 48	2	6,485 40,893	8,634 49,046	466	166	42	29	16	
				-			2,069	1,311	125	452	-	32
MOUNTAIN Mont.	820 16	223	28	4	13,709	18,492	3,171	1,503	235	161	44	1
Idaho	10	3	-	-	384 308	517 635	40 126	54 105	10	4	2	-
Wyo.	6	2		-	188	399	120	105	9	4	3	-
Colo.	299	70	3	-	2,969	4,190	220	185	64	70	8	1
N. Mex.	49	23	3	1	1,362	2,016	509	220	18	1	4	
Ariz.	261	79	13	1	5,037	6,288	1,766	586	72	55	19	-
Utah Nev.	60 119	25 17	4	2	499	595	286	128	37	18	3	-
			5	-	2,962	3,852	219	213	22	9	5	-
PACIFIC	5,970	1,147	118	24	69,036	101,089	9,392	4,442	750	605	90	90
Wash. Oreg.	361 173	-	7	4	6,481 2,990	8,265 3,708	2,144	813	183	70	22	7
Calif.	5,320	1,017	106	20	2,990	3,708 86,828	1,273 5,430	540 2.985	85	21	4	1
Alaska	19	25	3	- 20	970	1,517	5,430	2,985	469 8	497	61	67
Hawaii	97	105	2	-	544	771	12	50	5	12 5	- 3	1 14
Guam	1			-	136	180	9	13	-			
P.R.	1,229	69	4	1	1,210	1,763	52	241	41	2 41	1	5
V.I.	32	•	-	-	414	268	1	7	2	-	-	3
Amer. Samoa	-	-	-	-	74	82	7	2	-	5	-	2
C.N.M.I.					47		1	3				

TABLE III. Cases of specified notifiable diseases, United States, weeks ending December 3, 1988 and December 5, 1987 (48th Week)

N: Not notifiable

J.

	Malaria	Measles (Rubeola)					Menin- gococcal	M	mps		Pertussi		Rubella			
Reporting Area	Ivialaria	Indigenous		Imported*		Total	Infections									
	Cum. 1988	1988	Cum. 1988	1988	Cum. 1988	Cum. 1987	Cum. 1988	1988	Cum. 1988	1988	Cum. 1988	Cum. 1987	1988	Cum. 1988	Cum. 1987	
UNITED STATES	922	26	2,423	-	320	3,548	2,562	129	4,288	71	2,657	2,345	1	189	329	
NEW ENGLAND	70	-	83	•	54	281	219	-	117	-	176	160	-	9	2	
Maine N.H.	3 3	-	7 67	-	44	3 162	10 23	•	- 105	:	24 47	28 39	:	- 5	1	
Vt.	5	-	· ·		-	26	17	-	5	-	5	4	•	-	-	
Mass.	33 7	-	2	-	2	66	95	•	7	-	60	54	•	3	1	
R.I. Conn.	19	-	7	-	8	2 22	21 53	-	-	-	17 23	5 30	-	1		
MID. ATLANTIC	162		903	-	50	586	280	3	350	7	235	288	-	14	12	
Upstate N.Y. N.Y. City	39 89	-	19 46	:	18 6	41 466	130 65	1	97 101	4	142 8	162 19	:	2 7	10 1	
N.J.	11	-	309	-	12	39	63	2	55	2	17	20	-	3	i	
Pa.	23	-	529	-	14	40	22	-	97	1	68	87	-	2	-	
E.N. CENTRAL	48	-	141	-	108	383	357	34	851	2	241	256	•	31	40	
Ohio Ind.	11 4	-	2 57		83	5	129 29	17 4	130 77	-	49 74	74 17	-	1		
III.	3	-	56	-	16	203	74	9	308	-	44	17	-	26	29	
Mich.	23	-	26	-	5	29	85	4	219	2	37	47	-	4	9	
Wis.	7	-	-	•	4	146	40	-	117		37	101	-	•	2	
W.N. CENTRAL Minn.	18 6	-	11 10	-	3 1	230 39	95 20	14	205	1	128 49	138 13	-	2	2	
lowa	2	-	-	-	i		-	2	36	1	34	58	-	-	1	
Mo.	6	-	1	-	1	189	37		40	-	22	34	-	-	•	
N. Dak. S. Dak.	-	-	-	-	-	1	1	-	1	:	11 5	14 3	-	-		
Nebr.	1	-	-	-	-	-	12	-	11	-	-	1	-	-	-	
Kans.	3	-	-	-	-	1	21	12	117	-	7	15	•	2	1	
S. ATLANTIC	121	1	396	•	22	170	440 2	42	727	4	243	305 5	-	18	19 2	
Del. Md.	1 21	1	12	-	5	32 10	53	35	1 164	-	7 46	19	:	1	3	
D.C.	12	-	-	-	-	1	9	6	285	-	1	-	-	-	1	
Va. W. Va.	20 3	-	218 6	-	2	1	52 7	1	136 18	1 1	24 9	52 39	-	11	1	
N.C.	16	-	-	-	5	6	67		51	1	66	119		1	1	
S.C.	10	-	-	-	-	2	37	-	6	-	1	-	-	-	:	
Ga. Fla.	6 32	-	160	-	10	10 108	69 144	:	31 35	1	36 53	23 48	2	2 3	2 9	
E.S. CENTRAL	20	_	70	_		8	239	1	443		100	48		2	3	
Ky.	1	-	35	-	-	-	54	-	210	-	12	2	-	-	2	
Tenn.	-	-	1	-	-		130	1	215	-	29	15	-	2	1	
Ala. Miss.	10 9	-	34	-	-	4 4	40 15	N	15 N	-	55 4	24 7	:	:		
W.S. CENTRAL	82	-	14		3	448	172	22	835	30	233	304		11	11	
Ark.	4	-	-	-	1	•	20	8	133	9	34	13	-	4	2	
La. Okla.	12 10	-	- 8	:	-	4	48 19	4	301 197	-	18 62	50 163	1	1	- 5	
Tex.	56	-	6	-	2	444	85	10	204	21	119	78	•	6	4	
MOUNTAIN	43	17	135	-	33	497	75	4	208	17	796	211	•	6	25	
Mont.	5	17	23	-	31 1	128	2 8	1	2 6	2	2 330	6 71	-	-	8 1	
ldaho Wyo.	2	-	-	-		2	-	-	4	-	330	5	-		i	
Colo.	15	-	112	-	1	9	19	-	32		29	68	-	2	-	
N. Mex.	2 13	-	-	-	-	318 36	11 18	N 2	N 139	1 14	53 352	13 38	:	-	- 5	
Ariz. Utah	4	-	-	-	-	1	15	-	7		27	10	-	3	10	
Nev.	2	-	-	-	-	3	2	1	18	-	1	-	-	1	-	
PACIFIC	358	8	670	-	47	945	685	9	552	10	505	635	1	96	215	
Wash.	24 16	:	7	-	2	44 102	63 43	2 N	59 N	2 2	113 50	97 71	-	÷	2	
Oreg. Calif.	304	8	653	-	37	794	555	7	451	4	275	225	1	68	139	
Alaska	3	-	1	•	-	1	.7	-	13	-	7	6	-	-	2	
Hawaii	11	-	3	-	8	4	17	-	18	2	60	236	-	28	70	
Guam	2	:	- 226	-	1	2 771	12	-	3 10	•	- 15	- 20	-	1 3	1	
P.R. V.I.	-		- 220	-	-	-	-	-	33	:	15	20	-	3	3	
Amer. Samoa	-	-	-	-	-	1	3	-	3	-	-	-	-	-	-	
C.N.M.I.	1	-	-	-	-	-	1	-	2	-	-	-	-	-	-	

TABLE III. (Cont'd.) Cases of specified notifiable diseases, United States, weeks ending December 3, 1988 and December 5, 1987 (48th Week)

For measles only, imported cases includes both out-of-state and international importations.

Reporting Area		(Civilian) Secondary)	Toxic- shock Syndrome	Tuber	ulosis	Tula- remia	Typhoid Fever	Typhus Fever (Tick-borne) (RMSF)	Rabies, Animal
	Cum. 1988	Cum. 1987	Cum. 1988	Cum. 1988	Cum. 1987	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988
UNITED STATES	37,175	32,694	317	19,472	19,757	170	361	605	3,958
NEW ENGLAND	1,121	586	24	506	589	4	35	12	15
Maine N.H.	12 6	1 3	4 5	20	22	-	-	-	1
Vt.	3	3	2	11 4	18 15		1	-	5
Mass.	412	282	10	298	324	3	21	7	
R.I. Conn.	31	12	-	39	58	-	6	2	-
	657	284	3	134	152	1	7	3	9
MID. ATLANTIC Upstate N.Y.	8,875 548	6,029 240	48 22	4,034	3,605 474		70	19	459
N.Y. City	6,119	4,466	6	503 2,218	1.778		15 42	11 6	43
N.J.	918	666	3	668	641	-	11	-	14
Pa.	1,290	657	17	645	712	-	2	2	402
E.N. CENTRAL	1,092	808	46	2,139	2,181	1	34	34	143
Ohio Ind.	104 49	101 56	31	412	389	-	8	22	5
III.	483	408	1	222 929	228 987		2 18	27	29 31
Mich.	423	188	13	480	487	1	4	2	35
Wis.	33	55	•	96	90	-	2	1	43
W.N. CENTRAL	222	171	44	485	567	77	4	91	429
Minn.	18	20	5	85	112	3	2	2	125
lowa Mo.	23 146	26 78	7 12	53 234	38 308	- 47	2	-	13
N. Dak.	1	1	3	15	13	1	2	55	21 102
S. Dak.	-	11	4	33	24	16	-	7	112
Nebr. Kans.	28 6	15	4	14	25	3	-	1	19
	•	20	9	51	47	7	-	26	37
S. ATLANTIC Del.	13,363	11,187	19	4,144	4,215	5	43	198	1,372
Md.	96 651	66 579	1 3	38 390	39 361	2	3	1 22	57
D.C.	652	383	-	177	145	-	3	-	307 13
Va.	402	306	-	378	402	2	12	17	341
W. Va. N.C.	37 765	13 670	- 9	68 479	96 534	:	1	2	96
S.C.	698	668	3	479	534 430	-	2	107 23	8 121
Ga.	2,389	1,556	-	681	760	1	8	23	283
Fla.	7,673	6,946	3	1,495	1,448	-	14	3	146
E.S. CENTRAL	1,859	1,764	24	1,592	1,785	11	3	91	277
Ky. Tenn.	63 796	23	10	343	407	5	1	30	113
Ala.	533	699 465	10 3	476 481	544 509	5	1	39 10	69
Miss.	467	577	1	292	325	1	1	10	88 7
W.S. CENTRAL	4,018	4,089	29	2,468	2,305	53	8	144	, 508
Ark.	237	233	2	284	277	34	-	31	508 84
La.	803	759	-	311	285	-	4	2	10
Okla. Tex.	137 2,841	169 2,928	9 18	229 1,644	224 1,519	16 3	4	93	33
MOUNTAIN	-							18	381
Mont.	788 3	659 9	35	539 31	599 16	11	11 1	12	350
Idaho	4	5	5	19	29	-	-	6 2	196 11
Wyo.	.1	3	-	5	2	2	-	3	38
Colo. N. Mex.	99 47	115 54	3 2	74 90	146 94	5 2	3	1	28
Ariz.	157	284	16	232	255	1	1 6	-	11
Utah	16	23	9	29	25	i		-	41 9
Nev.	461	166	-	59	32	-	-	-	16
PACIFIC	5,837	7,401	48	3,565	3,911	8	153	4	405
Wash. Oreg.	196	153	8	212	227	1	14	1	-
Calif.	284 5,314	280 6,950	1 38	135 3,018	121 3,323	1 4	7 126	1	-
Alaska	15	0,350	-	3,018	58	2	126	2	384 21
Hawaii	28	14	1	156	182	-	5	-	-
Guam	3	2	-	30	26	-	-		_
P.R.	625	832	-	219	277	-	5	•	66
V.I. Amer. Samoa	2	9	-	6 4	2 10	-	:	-	-
C.N.M.I.	1			24	10	-	1	-	

TABLE III. (Cont'd.) Cases of specified notifiable diseases, United States, weeks ending December 3, 1988 and December 5, 1987 (48th Week)

U: Unavailable

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	r –	All Cau	uses, B	y Age	Years)		P&I**		T	All Cau	uses, B	y Age	Years)		P&I**
Reporting Area	All Ages	≥65	45-64	25-44	1-24	<1	Total	Beporting Area	All Ages	≥65	45-64	25-44	1-24	<1	Total
NEW ENGLAND	754	499	145	60	25	25	62	S. ATLANTIC	1,260	762	274	135	51	37	58
Boston, Mass.	187	108	42	19	7	11	29 2	Atlanta, Ga.	169	94	39	26	7	3 7	5
Bridgeport, Conn. Cambridge, Mass.	56 27	40 22	8 3	6 2	1	1	5	Baltimore, Md. Charlotte, N.C.	165 102	89 53	45 32	20 13	4 3	í	5 8
Fall River, Mass.	32	24	4	2	2	-	2	Jacksonville, Fla.	152	94	24	20	8	6	5
Hartford, Conn.	64	33	16	9	4	2	1	Miami, Fla.	97	51	24	16	5	1	-
Lowell, Mass. Lynn, Mass.	26 22	17 19	8 1	2	-	1	2	Norfolk, Va.	87 84	57 54	18 14	6 10	3 5	3 1	9 7
New Bedford, Mass.	33	26	5	1	1	-	-	Richmond, Va. Savannah, Ga.	47	35	8	2	2		2
New Haven, Conn.	66	42	8	5	6	5	2	St. Petersburg, Fla.	113	95	10	1	4	3	9
Providence, R.I.	83 5	60 4	20 1	3	-	:	3	Tampa, Fla.	72	45 86	14 39	4 16	4	4 8	3 5
Somerville, Mass. Springfield, Mass.	46	25	11	4	2	4	5	Washington, D.C. Wilmington, Del.	153 19	00 9	39	10	2	° -	
Waterbury, Conn.	41	31	8	2	-	-	4	E.S. CENTRAL	688	451	148	50	14	25	40
Worcester, Mass.	66	48	10	5	2	1	7	Birmingham, Ala.	116	79	22	6	1	- 25	2
MID. ATLANTIC	2,785	1,827	542	276	78	61	168	Chattanooga, Tenn.	52	36	11	3	2	-	3
Albany, N.Y.	58 24	43 19	8	1	3 1	3	3	Knoxville, Tenn.	60	39	10	8 4	1	3 1	5 3
Allentown, Pa. Buffalo, N.Y.§	106	79	19	5	2	1	8	Louisville, Ky. Memphis, Tenn.	77 168	50 114	21 34	12	4	4	15
Camden, N.J.	44	25	11	1	6	1	-	Mobile, Ala.	39	24	8	4	ź	1	1
Elizabeth, N.J.	22	16	1	1	3	1	6	Montgomery, Ala.	31	22	7	1	:	1	1
Erie, Pa.† Jersey City, N.J.§	47 59	36 40	7 11	2 6	2 1	1	4	Nashville, Tenn.	145	87	35	12	4	7	10
N.Y. City, N.Y.	1,449	958	260	17Ž	33	26	76	W.S. CENTRAL	1,845	1,165	410	165	65	40	68 3
Newark, N.J.	106	45	32	18	8	3	4	Austin, Tex. Baton Rouge, La.	64 20	45 13	14	2	2 1	1	1
Paterson, N.J. Philadelphia, Pa.	32 306	11 170	9 82	8 33	2 11	2 9	1 21	Corpus Christi, Tex.§		37	10	ĩ	-	-	1
Pittsburgh, Pa.†	88	61	20	3	2	2	-	Dallas, Tex.	165	97	43	11	7	7	5
Reading, Pa.	37	24	8	4	1	-	4	El Paso, Tex. Fort Worth, Tex	103 95	64 64	28 18	8 5	1 6	2	11 4
Rochester, N.Y.	130	104		3	-	3	19	Houston, Tex.§	734	436	169	89	24	16	18
Schenectady, N.Y. Scranton, Pa.†	20 35	15 29		1	-	:	8	Little Rock, Ark.	77	45	22	4	5	1	-
Syracuse, N.Y.	117	77	24	9	2	5	5	New Orleans, La.	134	87	26	15	4	2	1
Trenton, N.J.	46	28	11	3	1	3	4	San Antonio, Tex. Shreveport, La.	177 93	116 62	29 20	18 5	9 3	5 3	6 8
Utica, N.Y.	20 39	15 32	4	1	-	1	3	Tulsa, Okla.	135	99	27	5	3	ĭ	10
Yonkers, N.Y.		1,647	555	185	65	97	114	MOUNTAIN	802	501	190	52	29	30	41
E.N. CENTRAL Akron, Ohio	2,551 84	1,647		3		3/	2	Albuquerque, N. Mex	<. 98	62	18	13	2	3	-
Canton, Ohio	36	29	5	2	-	-	3	Colo. Springs, Colo.	42 144	25 90	10 31	4	1 6	2 4	3 6
Chicago, III.§	564	362		45 4	10	22	16	Denver, Colo. Las Vegas, Nev.	129	90 75	43	13 7	2	2	8
Cincinnati, Ohio	147 184	102 100	27 46	23	8 4	6 11	12	Ogden, Utah	32	25	4	2	1	-	5
Cleveland, Ohio Columbus, Ohio	127	73		11	3	. 8	1	Phoenix, Ariz.	167	97	42	4	10	14	8
Dayton, Ohio	136	98		9	4		5	Pueblo, Colo. Salt Lake City, Utah	31 38	22 17	8 15	1	2	3	1
Detroit, Mich.	348	181 61		37 5	21 1	12 6	9 1	Tucson, Ariz.	121	88	19	ż	5	2	ž
Evansville, Ind.	79 87	61		7	i	2	4	PACIFIC	2,088	1,375	404	179	74	49	118
Fort Wayne, Ind. Gary, Ind.	13	7	3	3	-	-	1	Berkeley, Calif.	18	15	2	1	-	-	3
Grand Rapids, Mich.	54	37		3 13	1	2 11	2	Fresno, Calif.	116	72	21	9	7	7	8
Indianapolis, Ind.	163 43	100 29		3	5	2	3	Glendale, Calif. Honolulu, Hawaii	21 74	18 41	2 25	1	3	2	8
Madison, Wis. Milwaukee, Wis.	160	115		5	4	4	11	Long Beach, Calif.	103	70	16	12	3	2	9
Peoria, III.	58	39		2	1	4	7	Los Angeles Calif.	492	321	89	52	19	4	16
Rockford, III.	53	39 37		4	-	1	6 4	Oakland, Calif.	73 35	47 28	17 2	4	2 4	3	5 3
South Bend, Ind.	53 118	85		4	2	i	12	Pasadena, Calif. Portland, Oreg.	124	87	23	10	2	2	5
Toledo, Ohio Youngstown, Ohio	44	32		-	•	1	-	Sacramento, Calif.	184	125	34	14	6	5	19
•	1,012	717		70	28	18	41	San Diego, Calif.	144	84	35	9	10	6	10
W.N. CENTRAL Des Moines, Iowa	104	80	15	5	3	1	9	San Francisco, Calif. San Jose, Calif.	192 183	115 123	42 35	29 14	2 7	4	5 14
Duluth, Minn.	44	32		3 2	2	1	i	San Jose, Calif. Seattle, Wash.	234	159	43	15	8	9	4
Kansas City, Kans.	39 110	23 77	20	9	4	-	8	Spokane, Wash.	60	43	12	3	1	1	6
Kansas City, Mo.	57	45	8	3	1	-	1	Tacoma, Wash.§	35	27	6	2	-	-	3
Lincoln, Nebr. Minneapolis, Minn.	236	162		16	6	9	16	TOTAL 1	3,785**	8,944	2,847	1,172	429	382	710
Omaha, Nebr.	114	78 107		10 12	4	2	3	1							
St. Louis, Mo.	159 89	71		5	1	2	1	1							
St. Paul, Minn. Wishita, Kana	60	42		5	3	2	ż								
Wichita, Kans.								I				nonula			

TABLE IV. Deaths in 121 U.S. cities,* week ending December 3, 1988 (48th Week)

*Mortality data in this table are voluntarily reported from 121 cities in the United states, most of which have populations of 100,000 or more. A death is reported by the place of its occurrence and by the week that the death certificate was filed. Fetal deaths are not

more. A deam is here a structure included. **Pneumonia and influenza. *Because of changes in reporting methods in these 3 Pennsylvania cities, these numbers are partial counts for the current week. Complete counts will be available in 4 to 6 weeks. Complete counts will be available. Figures are estimates based on average of past available 4 weeks.

Screening Tests - Continued

USE OF HTLV-I SCREENING TESTS IN BLOOD BANKS

The FDA recommends that whole blood and cellular components donated for transfusion be screened for HTLV-I antibody using a licensed EIA screening test. The FDA further recommends that units that are repeatably reactive by EIA be quarantined, then destroyed, unless otherwise stipulated by the FDA. Source plasma (obtained from plasma donors) intended for use in further manufacturing need not be screened for HTLV-I antibody.

DONOR DEFERRAL AND NOTIFICATION

FDA recommends permanent deferral of donors whose sera are repeatably reactive in EIA and confirmed as positive for HTLV-I antibody by additional, more specific testing. Such donors should be notified and counseled accordingly.

Donors whose serum specimens are repeatably reactive in the EIA but not confirmed as positive for HTLV-I antibody need not be notified on the first occasion. Although the donated units must be destroyed, the donors remain eligible for future donation. If, however, the donors test repeatably reactive in the EIA on a subsequent donation, they should be deferred indefinitely as donors and notified and counseled accordingly.

GUIDELINES FOR COUNSELING

Counseling should be considered a routine adjunct depending on the results of HTLV-I testing. Given some of the uncertainties related to testing, e.g., the inability to distinguish between antibodies to HTLV-I and HTLV-II, and the low probability that disease will occur in seropositive persons, every effort should be made to minimize the anxiety provoked by a repeatably reactive screening test, particularly one that is not confirmed as HTLV-I-seropositive by additional testing.

Persons confirmed as seropositive for HTLV-I should be notified that they have antibody to HTLV-I and are likely infected with HTLV-I or HTLV-II. They should be given information concerning disease associations and possible modes of transmission. In addition, they should be advised that they have been permanently deferred as blood donors and should neither give blood for transfusion nor share needles that have been used for percutaneous injection or infusions with other persons. Breastfeeding of infants should be discouraged. The paucity of data concerning sexual transmission of HTLV-I/HTLV-II does not permit a firm recommendation concerning sex practices; specific recommendations, such as the use of condoms to reduce the potential risk of sexual transmission, should be developed in consultation with a health-care professional.

Persons whose serum specimens are repeatably reactive on more than one occasion in the EIA but not confirmed as positive for HTLV-I antibody in more specific testing should be informed that they have inconclusive test results that do not necessarily imply infection with HTLV-I or HTLV-II. Nevertheless, they should be notified that they have been deferred indefinitely as donors and should not donate blood for transfusion. Periodic follow-up of such donors with EIA, more specific serologic tests, and possibly sophisticated techniques such as virus isolation and/or PCR may provide more reliable information regarding the presence of viral infection. *Reported by: Public Health Service Working Group.*[†]

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Screening Tests – Continued

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Progress in Chronic Disease Prevention

Years of Potential Life Lost due to Cancer – United States, 1968–1985

Although the incidence of cancer is relatively low in persons <65 years of age (82.9 deaths per 100,000 persons in this age group in 1985), it is the second leading cause of years of potential life lost (YPLL) for this age group, exceeded only by injuries. In 1985, 1,952,171 YPLL were attributable to cancer*. This is essentially unchanged (a 0.7% increase) from the YPLL for 1984. In 1985, white males contributed 43.1% of the cancer-attributable YPLL; white females, 40.1%; black males, 7.9%; and black females, 7.0%. Males and females of other races account for the remaining 1.9%.

Between 1968 and 1985, cancer mortality rates in persons <55 years of age declined 23% from 43 to 35 deaths per 100,000 persons in this age group, while rates in those \geq 55 years of age increased 17% from 775 to 905 per 100,000 persons (1). During this period, total YPLL remained relatively constant, with an average annual decline of <1%. However, the age-adjusted rate of cancer-attributable YPLL for the total population steadily decreased from 1968 to 1985 (Table 1), reflecting the overall decline in cancer mortality in younger persons.

Rates of cancer-attributable YPLL in 1985 were age-adjusted by race-gender groups: the highest rate occurred for black males (1208.1 per 100,000), followed by white males (949.4), black females (876.7), and white females (840.5). The rates in all four major race-gender groups also declined differentially (Figure 1). The average annual decline between 1968 and 1985 was approximately twice as great for black females (a decline of 18.9 per 100,000 per year) as for black males (9.4), white females (9.6), or white males (9.9).

Reported by: Div of Chronic Disease Control and Community Intervention, Center for Chronic Disease Prevention and Health Promotion, CDC.

Editorial Note: As life expectancy increases and all causes of death in earlier years of life decrease, mortality patterns and public health priorities may change. The patterns for cancer mortality and YPLL illustrate the complex shifts that may alter perception of the importance of cancer in young persons. Declines in mortality from infectious diseases

^{*}This report examines cancer mortality and YPLL for all mentions of cancer on death certificates, using multiple cause of death tapes from the National Center for Health Statistics. Cancer is selected as the underlying cause of death on 88% of death certificates mentioning cancer as a cause of death. Cancer-attributable YPLL is computed using differences between age at death from cancer and 65 years.

YPLL - Continued

and major chronic conditions such as cardiovascular disease in younger persons have increased the relative public health burden of cancer mortality.

YPLL reflects both the rate of disease and the size of the population at risk. Although the rate of cancer in younger persons, particularly those <55 years, is decreasing, some of the largest population increases by age group occur for persons 30–50 years of age, the result of higher birth rates during 1946–1964. Because cancer rates have decreased while the size of the population at risk has increased, virtually no change has occurred in the annual total number of cancer-attributable YPLL from 1968 to 1985. Thus, cancer-attributable YPLL has produced a constant disease burden.

Age-adjusted YPLL rates (Table 1) show an overall downward trend, however, reflecting diminishing cancer mortality rates in persons <55 years of age. This downward trend, which occurred in each of the four largest race-gender groups (Figure 1), is most prominent for black women, whose decrease is twice that of the other groups. This decrease does not appear to be attributable to greater population growth among black women, since their growth rate is identical to that of black men, whose decline in age-adjusted YPLL was the smallest of the four groups. Instead, the differential decline in age-adjusted YPLL appears to be related to a complex interaction between cancer incidence, mortality, and survival; this interaction may vary by tumor sites for different segments of the population. Since the number of cancer

Year	YPLL*	Age-adjusted rate [†]
1968	1,958,714	1,073.6
1969	1,953,236	1,062.0
1970	1,970,612	1,065.0
1971	1,960,541	1,048.1
1972	1,952,028	1,033.6
1973	1,956,137	1,027.5
1974	1,952,977	1,017.5
1975	1,919,724	993.6
1976	1,920,902	986.3
1977	1,930,981	983.4
1978	1,921,543	969.9
1979	1,906,684	953.8
1980	1,932,719	959.0
1981	1,906,013	939.0
1982	1,924,103	939.6
1983	1,921,549	929.3
1984	1,938,046	928.0
1985	1,952,171	925.9

TABLE 1. Cancer-attributable years of potential life lost before age 65 years (YPLL) and age-adjusted YPLL rates – United States, 1968–1985

*Calculated by multiplying the age-specific disease rate by population by the number of years between death and age 65. Deaths that occur after age 65 do not contribute to the total. *Rates are per 100,000 persons <65 years and are computed by applying age-specific rates for YPLL (i.e., YPLL/population for each age stratum) to a standard population. Here, the U.S. population structure under age 65 for 1980 was the standard.

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YPLL – Continued

cases occurring in certain population groups may be small, data are insufficient to address such interactions (1,2).

Although the decline in age-adjusted rates for cancer-attributable YPLL is encouraging, understanding the basis and public health implications of this decline requires further investigation. These efforts may need to focus on cancer incidence, mortality, and survival among younger population subgroups and on the relative impact of these measurements and programs designed to affect them (3).

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- National Cancer Institute. Cancer incidence and mortality in the United States, surveillance, epidemiology and end results. Bethesda, Maryland: US Department of Health and Human Services, Public Health Service, National Institutes of Health; NIH publication no. 85-1837.
- US Department of Health and Human Services. Report of the Secretary's Task Force on Black and Minority Health. Washington, DC: US Department of Health and Human Services, 1985.

1400 1200 1000 800 Rate 600 Black Male Black Female 400 White Male 200 White Female 0 1976 1978 1982 1984 1968 1970 1972 1974 1980 Year

FIGURE 1. Age-adjusted rate of years of potential life lost due to cancer per 100,000 persons by race-gender group – United States, 1968–1985

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Dental Caries and Periodontal Disease among Mexican-American Children from Five Southwestern States, 1982–1983.

Rabies Surveillance, United States, 1987 (Vol. 37, No. SS-4, September 1988).

Clarification: Vol. 37, No. 47

p. 718 In the article, "HIV-Related Beliefs, Knowledge, and Behaviors among High School Students," the second sentence of the second full paragraph should be clarified to read: "Because response rates of schools from some sites were less than 100%, results from these sites cannot be generalized...."

Errata: Vol. 37, No. 42

- p. 654 In the article, "Human Plague United States, 1988," the vaccine dose given to the patient in case 7 is incorrect. The dose given in the first sentence under case 7 should be 1.0 mL.
- p. 656 Reference 3 should be: ACIP. Plague vaccine. MMWR 1982;31:301-4.

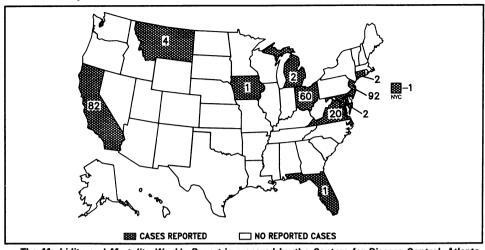


FIGURE I. Reported measles cases - United States, Weeks 44-47, 1988

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