

Epidemiologic Notes and Reports

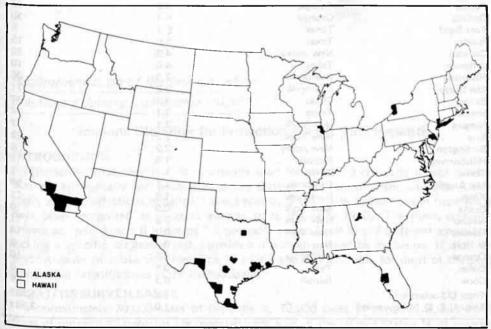
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Measles - U.S. Counties, First 26 Weeks, 1981

During the first 26 weeks (ending July 4) of 1981, 247 (7.9%) of the 3,144 counties in the United States reported measles (1). Only 33 (1.0%) of the counties reported measles for more than 5 of the first 26 weeks (Figure 1), accounting for 1,702 (72.5%) of the 2,347 cases provisionally reported during this period. These 33 counties are located in 8 states: Texas 12, New York 8, California 4, Florida 3, Georgia 2, New Jersey 2, Illinois 1, and Pennsylvania 1. The measles incidences in these counties ranged from 0.3 to 64.4 cases per 100,000 total population (all ages) (Table 1). For the 33 counties combined, the incidence was 4.3 cases per 100,000 population, compared with 0.3 case per 100,000 total population for the remaining 3,111 counties. The overall U.S.

FIGURE 1. U.S. counties that reported measles for more than 5 of the first 26 weeks of 1981*



*Ending July 4.

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Measles - Continued

incidence was 1.0 case per 100,000 total population. Eight of the 33 counties were at or below the overall U.S. incidence.

Reported by Surveillance and Assessment Br, Immunization Div, Center for Prevention Services, CDC. Editorial Note: These data show that prolonged measles transmission in the United States in 1981 has been limited to relatively few counties. During the first half of 1981 more than 92.1% (2,897) of the U.S. counties reported no measles, and another 6.8% (214) had measles transmission for short periods of time. Only 1.0% (33) of the counties had more prolonged transmission, and these accounted for almost three-fourths of the reported measles cases.

In areas with prolonged measles transmission, special emphasis should be given to

County	State	Incidencet	Cases reported
Val Verde	Texas	64.4	23
Hidalgo	Texas	54.8	153
Pasco	Florida	53.4	102
El Paso	Texas	44.9	215
Cameron	Texas	33.2	69
Philadelphia	Pennsylvania	23.6	397
Webb	Texas	14.1	14
Westchester	New York	13.4	116
Pinellas	Florida	6.5	47
Fulton	Georgia	6.5	38
DeKalb	Georgia	6.3	30
Fort Bend	Texas	6.1	8
Nueces	Texas	5.6	15
Camden	New Jersey	4.2	20
Jefferson	Texas	4.0	10
Riverside	California	3.9	26
San Diego	California	3.8	71
Bexar	Texas	3.8	37
Harris	Texas	3.1	74
Niagara	New York	3.1	- 7
Erie	New York	2.2	22
Burlington	New Jersey	2.2	8
Hillsborough	Florida	1.9	12
Travis	Texas	1.7	7
Los Angeles	California	1.1	80
Kings	New York	1.0	21
Bronx	New York	0.9	10
Nassau	New York	0.8	11
Queens	New York	0.7	14
Suffolk	New York	0.7	9
Orange	California	0.5	10
Dallas	Texas	0.5	8
Cook	Illinois	0.3	18
Total (33 counties)		4.3	1,702
Total U.S. (3,144 counties)		1.0	2,347

TABLE 1. U.S. counties that reported measles for more than 5 of the first 26 weeks,* 1981

* Ending July 4.

† Cases per 100,000 population, all ages. Based on preliminary 1980 census data.

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Measles – Continued

determining why sustained measles outbreaks have occurred. Since the fundamental strategy for measles elimination is achieving and maintaining high immunization levels against measles, an assessment of the immunization levels of school children and an evaluation of how well school laws are enforced should be given the highest priority (2). If immunization levels are low, steps should be taken to enforce laws by excluding students from school who lack documentation of measles immunity (i.e., a record of physician-diagnosed measles or of vaccination with live measles vaccine on or after the first birthday) (3-5). In addition, the county measles surveillance system and outbreak-control programs should be evaluated and improvements made, if necessary.

Most of the 33 counties wherein the majority of reported cases are concentrated have made substantial progress in recent years and are continuing to strengthen their measlescontrol programs. Stronger school immunization requirements for measles are now being enforced in several of the states in which these counties are located.

References

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- 4. Robbins KB, Brandling-Bennett AD, Hinman AR. Low measles incidence: association with enforcement of school immunization laws. Am J Public Health 1981;71:270-4.
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Recommendation of the Immunization Practices Advisory Committee (ACIP)

Immune Globulins for Protection against Viral Hepatitis

INTRODUCTION

The term "viral hepatitis" is commonly used for at least 3 clinically similar diseases that are etiologically and epidemiologically distinct (1). Two of them, hepatitis A (formerly called "infectious hepatitis") and hepatitis B (formerly called "serum hepatitis"), have been recognized as separate entities since the early 1940s. The third, currently known as "non A/non B hepatitis," is probably caused by at least 2 different agents and, lacking a specific diagnostic test, remains a disease diagnosed by exclusion. It is an important cause of acute viral hepatitis in adults and is responsible for most of the post-transfusion hepatitis cases in the United States.

HEPATITIS SURVEILLANCE

Approximately 30,000 cases of hepatitis A, 16,000 cases of hepatitis B, and 8,000 cases of unspecified hepatitis are reported each year in the United States. Most patients are young adults.

ACIP Recommendation on Viral Hepatitis - Continued

IMMUNE GLOBULINS

Immune globulins used in medical practice are sterile solutions of antibodies (immunoglobulins) from human plasma. They are prepared by cold ethanol fractionation of large plasma pools and contain 10%-18% protein. In the United States, plasma is primarily obtained from professional donors. Only plasma shown to be free of hepatitis B surface antigen (HBsAg)* is used to prepare immune globulins.

Immune globulin (IG) (formerly called "immune serum globulin," ISG, or "gamma globulin") produced in the United States contains antibodies against the hepatitis A virus (anti-HAV) and the hepatitis B surface antigen (anti-HBs). Tests of lots of IG prepared since 1977 indicate that both types of antibody have uniformly been present at stable titers.

Hepatitis B immune globulin (HBIG) is an immune globulin prepared from plasma containing extremely high titers of anti-HBs.

*Abbreviations are summarized in Table 2.

Abbreviation	Term	Comments
CAUSE AND A SUM	Hepatiti	s A
HAV	Hepatitis A virus	Etiologic agent of "infectious" hepatitis; probably an enterovirus; single serotype.
anti-HAV	Antibody to HAV	Detectable at onset of symptoms; lifetime persistence.
	Hepatiti	s B
нвv	Hepatitis B virus	Etiologic agent of "serum" or "long-incuba- tion" hepatitis; also known as Dane particle.
HBsAg	Hepatitis B surface antigen	Surface antigen(s) of HBV, detectable in large quantity in serum; several subtypes identified.
HBeAg	Hepatitis B e antigen	Soluble antigen; correlates with HBV repli- cation; high titer HBV in serum, and infec- tivity of serum.
HBcAg	Hepatitis B core antigen	No commercial test available.
Anti-HBs	Antibody to HBsAg	Indicates past infection with and immunity to HBV, passive antibody from HBIG, or immune response from HBV vaccine.
Anti-HBe	Antibody to HBeAg	Presence in serum of HBsAg carrier suggests lower titer of HBV.
Anti-HBc	Antibody to HBcAg	Indicates past infection with HBV at some undefined time.
	Non A/non B	hepatitis
NANB	Non A/non B hepatitis	Diagnosis of exclusion; at least 2 candidate viruses; epidemiology parallels that of hepa- titis B.
	Immune glo	obulins
IG	Immune globulin (previously ISG, immune serum globulin, or gamma globulin)	
HBIG	Hepatitis B immune globulin	
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TABLE 2. Hepatitis nomenclature

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ACIP Recommendation on Viral Hepatitis – Continued

Neither IG nor HBIG when properly prepared transmits hepatitis A or B.

Serious adverse effects from immune globulins administered as recommended have been exceedingly rare. Globulins are prepared for intramuscular use and should *not* be given intravenously.

Immune globulins are not contraindicated for pregnant women if needed.

HEPATITIS A

Hepatitis A is caused by the hepatitis A virus (HAV), a 27-nm RNA (ribonucleic acid) agent that is a member of the picornavirus family. The illness caused by HAV characteristically has an abrupt onset with fever, malaise, anorexia, nausea, abdominal discomfort, and jaundice. Severity is related to age. In children, most infections are asymptomatic, and illness is not accompanied by jaundice. Fatality among hospitalized patients is quite low (about 0.1%).

Hepatitis A is primarily transmitted by person-to-person contact, generally through fecal contamination. Transmission is facilitated by poor sanitation and close personal contact, including sexual exposures. Common-source infections from contaminated food and water also occur.

The incubation period of hepatitis A is 15-50 days (average 28-30). HAV has consistently been demonstrated in stools of infected persons, with the highest concentrations of virus being excreted late in the incubation and early in the prodromal phase of illness. Virus excretion diminishes rapidly once jaundice appears. Greatest infectivity is during the 2-week period immediately before the onset of jaundice. Viremia is of short duration. A chronic carrier state with HAV in blood or feces has not been demonstrated. Although theoretically possible, transmission of HAV by blood transfusion or percutaneous routes appears to be extremely rare.

Specific tests are available to differentiate anti-HAV of the IgM class, which appears in the acute phase of illness, from anti-HAV of the IgG class, which appears in convalescence (4-6 weeks after onset) and largely replaces IgM-class antibody. The diagnosis of acute hepatitis A is therefore confirmed by finding IgM-class anti-HAV as the predominant specific antibody in serum collected during the acute phase of disease. IgG-class anti-HAV, which replaces IgM-class antibody, remains detectable in serum for years and apparently confers life-long protection against reinfection.

Sero-epidemiologic studies show that hepatitis A is still a common infection in the United States. More than half the population over age 40 have serologic evidence of past infection.

IG AND HEPATITIS A

Numerous field studies conducted in the past 4 decades confirm that IG given before exposure or during the incubation period of hepatitis A is protective (2-4). Its prophylactic value is greatest (80%-90%) when given early in the incubation period and declines thereafter (4). In view of the need to give IG as soon as possible after exposure to HAV, and recognizing its intrinsic safety and the time required for—and cost of—antibody testing, routine serologic screening for anti-HAV before giving IG is not encouraged. Giving IG more than 2 weeks after exposure is not indicated.

RECOMMENDATIONS FOR IMMUNE GLOBULIN PROPHYLAXIS FOR HEPA-TITIS A

Specific recommendations for IG prophylaxis for hepatitis A depend on the nature of the HAV exposure:

ACIP Recommendation on Viral Hepatitis - Continued

Post-Exposure Prophylaxis

Person-to-person contact:

Close personal contact. IG is recommended for all household and sexual (heterosexual or homosexual) contacts of persons with hepatitis A.

Day-care centers: Day-care centers with children in diapers can be important locales for HAV transmission (5,6). If epidemiologic evidence shows that HAV transmission is occurring in a day-care center that cares for children in diapers, IG should be administered to staff, attendees, and to all members of households whose diapered children attend. Careful handwashing after changing diapers is important.

Schools and preschools: Contact at school is usually not an important means of transmitting hepatitis A. Routine administration of IG is not indicated for pupils and teachers in contact with a patient. However, when epidemiologic study clearly shows the existence of a school- or classroom-centered outbreak, it is reasonable to give IG to those who have close personal contact with patients.

Institutions for custodial care: Living conditions in some institutions, such as prisons and facilities for the developmentally disabled, favor transmission of hepatitis A. When outbreaks occur, giving IG to residents and staff who have close contact with patients with hepatitis A may effectively reduce the spread of disease. Depending on the epidemiologic circumstances, prophylaxis can be limited in extent or can involve the entire institution.

Hospitals: Routine IG prophylaxis for hospital personnel is not indicated. Rather, sound hygienic practices should be emphasized. Intensive continuing staff education should point out the risk of exposure to hepatitis A and emphasize precautions regarding close contact with patients with hepatitis or with infective materials (7).

Offices and factories: Routine IG administration is not indicated under the usual office or factory conditions for persons exposed to a fellow worker with hepatitis A. Common-source exposure:

IG might be effective in preventing foodborne or waterborne hepatitis A if exposure is recognized in time. However, IG is not recommended for persons exposed to a common source of potential hepatitis infection once cases have begun to occur.

If a foodhandler is diagnosed as having hepatitis A, common-source transmission is possible. IG should be administered to other kitchen employees and may be considered for patrons if 1) the infected person is directly involved in handling foods that are not to be cooked or cooked foods before they are eaten, 2) the hygienic practices of the food-handler are deficient, and 3) consumers can be identified and treated within 2 weeks of exposure.

For postexposure IG prophylaxis, a single intramuscular dose of 0.02 ml/kg* is recommended.

Pre-Exposure Prophylaxis:

Travelers to foreign countries: The risk of hepatitis A for Americans traveling abroad appears to be small. It varies with living conditions, the prevalence of hepatitis A in the areas visited, and especially the length of stay (8,9). As with any enteric infection, the best way to prevent hepatitis A is to avoid potentially contaminated water and food.

Travelers who follow the usual tourist routes may be at no greater risk of getting hepatitis A than they would be in the United States. IG is not recommended for them. However, travelers to high-risk areas outside ordinary tourist routes may be at increased risk.

*Milliliters/kilogram of body weight.

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For such travelers, at risk for up to 2.3 months, a single IG dose of 0.02 ml/kg is recommended. For more prolonged travel, 0.06 ml/kg should be given every 5 months.

HEPATITIS B

Hepatitis B is caused by the hepatitis B virus (HBV), a 42-nm, double-shelled DNA (deoxyribonucleic acid) virus. Several well defined antigen-antibody systems have been associated with HBV infection. HBsAg, formerly called "Australia antigen" or "hepatitis associated antigen," is an antigen found on the surface of the virus and on accompanying 22-nm spherical and tubular forms. HBsAg can be identified in serum 30-60 days after exposure to HBV and persists for variable periods. The various subtypes of HBsAg provide useful epidemiologic markers. Antibody against HBsAg, i.e., anti-HBs, develops after a typically resolved infection and is responsible for long-term immunity.

The frequency of chronically carrying HBsAg apparently relates both to age and immunologic competency. As many as 10% of HBV infections result in chronic carriage of HBsAg. The carrier state can be completely asymptomatic or, less commonly, associated with active liver disease. HBsAg carriers play an important role in the continuing transmission of hepatitis B, even though they show varying degrees of infectivity.

The hepatitis B e antigen (HBeAg) and antibody (anti-HBe) are distinct from HBsAg and anti-HBs. The potential infectivity of a carrier is higher if the HBeAg is present and lower if anti-HBe is present.

Routes of transmission of HBV include 1) direct percutaneous inoculation of infective serum or plasma by needle or transfusion of infective blood or blood products; 2) indirect percutaneous introduction of infective serum or plasma, such as through minute skin cuts or abrasions; 3) absorption of infective serum or plasma through mucosal surfaces, such as those of the mouth or eye; 4) absorption of other potentially infective secretions such as saliva or semen through mucosal surfaces, as might occur following sexual (heterosexual or homosexual) contact; and 5) transfer of infective serum or plasma via inanimate environmental surfaces or, possibly, vectors. Experimental data indicate that fecal transmission of HBV does not occur and that airborne spread is not epidemiologically important.

The onset of hepatitis B is generally insidious. Clinical symptoms and signs include various combinations of anorexia, malaise, nausea, vomiting, abdominal pain, and jaundice. Arthralgias and arthritis can also occur. Overall fatality rates for hospitalized patients generally do not exceed 1%. The incubation period of hepatitis B is long-45-160 days (average 60-90). Cirrhosis and primary heptatocellular carcinoma are closely associated with chronic HBV infection.

IMMUNE GLOBULINS AND HEPATITIS B

IG and HBIG contain different amounts of anti-HBs. IG is prepared from plasma that is not preselected for anti-HBs content. Since 1977, all lots tested have contained anti-HBs at a titer of at least 1:100 by radioimmunoassay (RIA). HBIG, on the other hand, is prepared from plasma preselected for high titer anti-HBs. In the United States, HBIG has an anti-HBs titer of >1:100,000 by RIA. (Currently, the price of a dose of HBIG is more than 20 times that of IG.)

Recent studies have shown that immune globulins can prevent up to 75% of hepatitis B cases in certain settings (10,11). What has been difficult to determine is the concentration of antibody that would be effective under various conditions of exposure, because studies differed in experimental design and in the immune globulins tested (12-19).

The studies generally indicated that: 1) HBIG is effective when given after percutaneous

ACIP Recommendation on Viral Hepatitis - Continued

(needle stick) or mucous membrane exposure to blood containing HBsAg; 2) IG appears to have some effect in preventing clinical hepatitis; and 3) an immune globulin is most effective if given immediately after exposure.

It can be agreed further that HBIG is preferable to IG when there is bona fide percutaneous or mucous membrane exposure to blood known to contain HBsAg. However, because IG does contain anti-HBs, it remains an important alternative to HBIG whenever HBIG is unavailable, its cost is prohibitive, or a truly significant exposure to HBV may not have occurred.

Post-Exposure Prophylaxis:

Acute exposure to blood that might contain HBsAg: Percutaneous (needle stick) or mucous membrane exposure to blood that might contain HBsAg calls for a prompt decision about giving an immune globulin. In deciding whether to give a globulin and, if so, whether it should be IG or HBIG, one must recognize that the need is relative and depends on the kind of exposure. In the hospital, risk of clinical hepatitis B following exposure to blood *known* to contain HBsAg is approximately 1 in 20. If the blood is of *unknown* HBsAg status, the risk is 100 times lower, only about 1 in 2,000. This latter risk increases, however, in direct proportion to the likelihood that the blood is HBsAg-positive.

(Continued on page 433)

10 M	34th WE	EK ENDING		CUMULATIVE, FIRST 34 WEEKS					
DISEASE	August 29 1981	August 23 1980	MEDIAN 1976-1980	August 29 1981	August 23 1980	MEDIAN 1976-1980			
Aseptic meningitis	405	323	323	4,499	3,530	2,985			
Brucellosis	3	4	4	94	129	129			
Chickenpox	271	293	264	166,151	156,857	156 851			
Diphtheria	-	-	1	3	2	56			
Encephalitis: Primary (arthropod-borne & unspec.)	45	40	53	693	552	554			
Post-infectious	5	2	3	58	146	152			
Hepatitis, Viral: Type B	360	364	304	13,282	11,254	9,833			
Туре А	379	490	5 80	16,341	17,931	19,148			
Type unspecified	222	229	188	7,204	7,367	5,763			
Malaria	13	54	15	920	1.351	436			
Measles (rubeola)	13	25	82	2,624	12.747	23,453			
Meningococcal infections: Total	33	30	30	2,485	1,899	1,736			
Civilian	33	30	30	2,476	1,885	1.714			
Military	-	al secol 1-	-	9	14	17			
Mumps	34	34	106	3,043	6,963	13,211			
Pertussis	26	50	50	741	1.024	903			
Rubelia (German measles)	7	28	49	1,702	3,179	10,562			
Tetanus	2	1	2	39	54	45			
Tuberculosis	561	576	576	17,574	17,655	19,081			
Tularemia	6	6	4	150	134	102			
Typhoid fever	12	8	8	329	295	295			
Typhus fever, tick-borne (Rky. Mt. spotted)	40	38	45	937	846	715			
Venereal diseases:	1.000								
Gonorrhea: Civilian	18,690	21.472	21.739	648,263	637,399	638,780			
Military	309	588	588	18,711	17.723	17.723			
Syphilis, primary & secondary: Civilian	571	534	467	19,562	17.015	15,697			
Military	2	15	8	238	217	193			
Rabies in animals	156	134	82	4,810	4,456	2,055			

TABLE II. Notifiable diseases of low frequency, United States

	CUM. 1981		CUM, 1981
Anthrax	-	Poliomyelitis: Total	3
Botulism (Calif. 1)	38	Paralytic	3
Cholera	3	Psittacosis (N.J. 1)	77
Concenital rubella syndrome	1 7	Rabies in man	1
Leprosy (III, 1, Calif, 5, Hawaii 5)	174	Trichinosis (Ohio 1)	105
Leptospirosis	26	Typhus fever, flea borne (endemic, murine) (Tex. 2)	35
Plague	• •	in both served interconnect different	IT THERE

All delayed reports and corrections will be included in the following week's cumulative totals.

TABLE III. Cases of specified notifiable diseases, United States, weeks ending August 29, 1981 and August 23, 1980 (34th week)

REPORTING AREA UNITED STATES 41 NEW ENGLAND Maine REI. Conn. MID. ATLANTIC Upstate N.Y. N.Y. City Pa. E.N. CENTRAL Ohio III. Mich. Wis. W.N. CENTRAL Minn. S. Dak. S. Dak. S. Dak. S. Dak. S. Dak. S. CATLANTIC Del. Md. D.C. Va. W. Va. S.C. S.C. S.C. S.C. S.C. S.C. S.C. S.	MENINA GITIS 1981 405 24 - - 7 7 9 6 6 5 9 21 16 16 75 37 16 16 1 30 - 6 20 - - 4 70 - 8 - - 4 70 - 8 -	CEL- LOSIS 1991 3 - - - - - - - - - - - - - - - - - -	CHICKEN- PDX 1981 271 70 2 - - - - - - - - - - - - - - - - - -	DIPH1 1981	CUM. 1981 3 - - - - - - - - - - - - - - - - - -	Pr 1981 45 - - - - - - - - - - - - -	imary 1980 40 4 - 1 - 2 - 1 6 3 - - 3 15 2 8 5 - - 2 - - - 2 - - - - - - - - - - - - -	Post-in- fections 1981 5 2 2 	B 1991 360 22 - - 3 4 3 12 64 14 20 30 NA 35 9 8 4 11 3 9 2	A 1981 379 12 1 1 1 2 - 7 31 12 9 10 NA 65 5 14 4 9 23 4 9 1	Unspecified 1981 222 11 - - 10 - 1 14 3 4 7 NA 22 10 1 1 4 - - 3 4 - - - - - - - - - - - - -	MA 1981 13 3 - - 2 1 3 - - 1 - - - 1 - - - 1 - - - - - - - - - - - - -	CUM 1981 1981 920 48 8 3 3 3 2 8 8 2 11 111 30 35 35 11 11 44 7 7 6 14
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Aass. Ll. conn. MID. ATLANTIC Jystate N.Y. Jystate N.Y. J.J. CITY Ja. N. CENTRAL White Mich. Minn. Owa Ac. J. Dak. Jebr. Jebr. Jebr. J. Dak. Jebr. J. Dak. Jebr. Ja. J.C. Ja. Ja. <td>7 9 6 21 10 16 75 37 21 1 1 1 30 - 6 20 - - 4 70 - 8 -</td> <td></td> <td>63 32 55 27 NN - 70 8 11 15 1 35 15 1 - 4 10 -</td> <td></td> <td></td> <td>- - 2 2 - 4 13 10 - - 2 1 14 13 1 -</td> <td>- 1 3 - 3 15 2 8 5 - - 2</td> <td></td> <td>4 3 12 64 14 20 30 NA 35 9 8 4 11 3 9</td> <td>2 - 7 31 12 9 10 NA 65 15 14 9 23 4 9</td> <td>10 1 14 3 4 7 NA 22 10 7 1 4 - 3</td> <td>2 1 3 - 1</td> <td>28 2 11 30 35 11 44 7 6 14 14</td>	7 9 6 21 10 16 75 37 21 1 1 1 30 - 6 20 - - 4 70 - 8 -		63 32 55 27 NN - 70 8 11 15 1 35 15 1 - 4 10 -			- - 2 2 - 4 13 10 - - 2 1 14 13 1 -	- 1 3 - 3 15 2 8 5 - - 2		4 3 12 64 14 20 30 NA 35 9 8 4 11 3 9	2 - 7 31 12 9 10 NA 65 15 14 9 23 4 9	10 1 14 3 4 7 NA 22 10 7 1 4 - 3	2 1 3 - 1	28 2 11 30 35 11 44 7 6 14 14
X.I. Jonn. AID. ATLANTIC AID. ATLANTIC Diate N.Y. V.Y. City Ja. N. CENTRAL Dhio II. Mich. Vis. V.N. CENTRAL Mich. Vis. Jak. Vebr. Cans. V. Va. J.G. J. J.A. V. Va. J.G. J.C. J.G. J.C. J.G. J.C. J.G. J.C. J.G.	9 6 9 21 19 16 75 37 21 - 16 1 30 - 6 20 - - 4 70 - 8 -		3 2 5 27 NN - 70 8 11 15 15 15 1 - 4 10			- - 2 2 - 4 13 10 - 2 1 14 13 1 -	- 1 3 - 3 15 2 8 5 - - 2	2	3 12 64 14 20 30 NA 35 9 8 4 11 3 9 2	- 7 31 12 9 10 NA 65 15 14 9 23 4 9	- 1 14 3 4 7 NA 22 10 7 1 1 4 - 3	1 3 2 1	2 11 30 35 11 44 7 6 14 14
MID. ATLANTIC ATLANTIC Jpstate N.Y. IV. CITY I.J. a. M. CENTRAL Model II. Model M. CENTRAL Model Model A. Joak. Jebr. Loak. Jebr. Joak. Jebr. J. ATLANTIC Model J.C. Ja. J.C. Ja. J.C. Ja. Ja. S. CENTRAL	65 9 21 19 16 75 37 21 - 16 1 30 - 6 20 - - 4 70 - 8 -		32 5 27 NN 70 8 11 15 1 35 15 - 1 4 10			8 2 2 - 4 13 10 - 2 1 1 14 13 1 -	6 3 - 3 15 2 8 5 - - 2		64 14 20 30 NA 35 9 8 4 11 3 9 2	31 12 9 10 NA 65 15 14 9 23 4	14 3 4 7 NA 22 10 7 1 4 -	3	111 30 35 11 44 7 6 14
Jostate N.Y. JAY. Giry JJ. a. N. CENTRAL Mio Min. Min. CENTRAL Min. Ova Mo. Loak.	9 21 19 16 75 37 21 - 16 1 30 - 6 20 - - 4 70 - 8 -		5 27 NN 70 8 11 15 15 15 15 15 15 - 4 10 -			2 2 - 4 13 10 - 2 1 14 13 1 -	3 - - 3 15 2 8 5 - - 2		14 20 30 NA 35 9 8 4 11 3 9 2	12 9 10 NA 65 15 14 9 23 4 9	3 4 7 NA 22 10 7 1 4 -	- 2 1	30 35 35 11 44 7 6 14 14
I.Y. CITY N.J. Ta. N.CENTRAL Dhio I. Mich. Vis. V.N. CENTRAL Minn. Vis. V.N. CENTRAL Vis. S. Dak. S. Dak. S. Dak. S. Dak. S. Dak. S. Dak. S. Dak. S. Dak. S. CENTRAL S. CENTRAL	21 19 16 75 37 21 		27 NN 70 8 11 15 15 15 15 15 15 4 10			2 4 13 10 - 2 1 14 13 1 -	- 3 15 2 8 5 - -		20 30 NA 35 9 8 4 11 3 9 2	9 10 NA 65 15 14 9 23 4 9	4 7 NA 22 10 7 1 4 -	2 1	35 35 11 44 7 6 14 17
N.J. a. S.N. CENTRAL bio nd. Min. Mich. Vis. V.N. CENTRAL Min. Owa Ao. J. Dak. Dak. bak.	19 16 75 37 21 - 16 1 30 - 6 20 - - 4 70 - 8 - 8	1	NN 70 8 11 15 15 15 15 15 15 - 1 4 10 -			- 4 13 10 - 2 1 14 13 1 -	- 3 15 2 8 5 - - 2		30 NA 35 9 8 4 11 3 9 2	10 NA 65 15 14 9 23 4	7 NA 22 10 7 1 4 -		35 11 44 7 6 14 17
N. CENTRAL Dhio nd. Mich. Wis. N. N. CENTRAL Minn. owa Mo. N. Dak. S. CENTRAL	75 37 21 16 1 30 6 20 - 4 70 8 	1	8 11 15 15 - 15 - 1 - 4 10 -			13 10 - 2 1 14 13 1 -	15 2 8 5 - - 2	-	35 9 8 4 11 3 9 2	65 15 14 9 23 4	22 10 7 1 4 -		44 7 6 14 17
Thio	37 21 16 1 30 - 6 20 - - 4 70 - 8 -		8 11 15 15 - 15 - 1 - 4 10 -			10 2 1 14 13 1 	2 8 5 - 2	-	9 8 4 11 3 9 2	15 14 9 23 4	10 7 1 4 -		7 6 14 17
nd	21 16 1 30 - 6 20 - - 4 70 8 -	1	11 15 1 35 15 - 1 4 10 -			- 2 1 14 13 1	8 5 2	-	8 4 11 3 9 2	14 9 23 4	7 1 4 - 3	-	6 14 17
II. Mich. Mirk. Mirk. Mirk. Mirk. Mirk. Mirk. Mirk. Mirk. Sowa Mirk. S. Dak. Mirk.	- 16 1 30 - 6 20 - - 4 70 - 8		15 1 35 15 - 1 4 10 -		-	- 2 1 14 13 1 -	5 - - 2	3	4 11 3 9 2	9 23 4 9	1 4 - 3	Ξ	14
Vis. Vis. CENTRAL dinn. owa do. J. Dak. J. Dak. S. Dak. Vebr. S. Dak. Vebr. S. Dak. S. Dak. Vebr. S. Central	1 30 6 20 - - 4 70 8 -	1	35 15 1 4 10	-	-	1 14 13 1		-	3 9 2	4	-	-	-
V.N. CENTRAL Minn. Gwa Ao. J. Dak. Dak. Dak. Dak. Dak. Dak. Dak. Ad. Ad. Ad. Ad. Ad. Ad. Ad. Ad	30 6 20 - - 4 70 8		15 			14 13 1		-	9 2	9		-	-
Minn. wwa Mo. N. Dak. S. Dak. S. Dak. S. Dak. S. Dak. Mo. S. ATLANTIC Del. Mo. J.C.	- 20 - - 4 70 8		1		Ē	13			2			1	
Owa Mo. S. Dak. S. Dak. Sens. S. ATLANTIC Md. J.C. J.C. J.C. J.C. J.C. J.C. J.C. J.	20 - - 4 70 8		10	Ē	Ē	1	-		£		1	ī	25
N. Dak. S. Dak. Vebr. Cans. S. ATLANTIC Del. Md. D.C. Va. V.C. S.C. S.C. S.CENTRAL	- - - - - - - - - - -	1	10	Ξ	-	-	_	-	ī	-	-	-	- 3
5. Dak. Vebr. 24 ans. 3. ATLANTIC 24. 0. C. 25. 3. C. 3. C. 5. CENTRAL	- - - - - - - -	1	10	1	2	-	1	-	5	4	1		2
Vebr. & ATLANTIC >a. ATLANTIC >bel. Md. 0.C. ya. V.Va. V.C. 3a. =1a. E.S. CENTRAL	4 70 8	2		-				-	-	ī	-	-	1
Cans. 2. ATLANTIC Del. dd. 0. C. 2. C. 4. C. 5. C. 5. CENTRAL	70 8	-					ī		ī		-	Ξ	1
Ъе!, Иd, J.C. Va,	8			-	-	-		-	-	3	1	-	7
Md. J.C. J.C. J.C. J.C. Ja. -S. CENTRAL	-		28	4.4	1	4	4	1	113	51	49	1	- 11
D.C. /a. /v. Va. /.C. /d. /a. .S. CENTRAL	-	-	6	2	1.2	1	1.1	2	19	25	1	1	25
V. Va. N.C. Sa. Sa. Sa. CENTRAL		-	-	-	-	-	1		2	ĩ	-	1	
N.C. S.C. Ga. Fla.	33	-	2	-	-	4	-	-	21	4	13	-	20
S.C. Sa. Fla. E.S. CENTRAL	3	2	NN	-	12	12	2	1	2	2	1	-	3
S. CENTRAL	-	-	-	1			-	-	21	í	2 - E	_	1
S. CENTRAL	2	1		1		12	Ξ.	ī	13 33	13	14	1	
E.S. CENTRAL (23		20		1		1						37
ΥΥ .	40	-	6	-	-	1	1	-	14	16 1	3	1	10
Tenn.	23	-	NN			12.		-	10	12	2	- 2	-
Ala.	10	-	-	-	-	1	-	-	-	-	1	-	9
Miss.	3	-	-	-	-	-	-	-	3	3	-	-	1
N.S. CENTRAL	13	-	21	-		3	-	-	32	55	42	-	65
Ark. _a.	3	1	1 NN	1.1	- 2	1		-	2	6 25	8	- 21	5
Okla.	í	-	-	12	-	-			2	-1	ĩ	-	ē
lex.	9	-	20	-	1.5	2	-	-	12	23	20	-	51
	15 2	Ξ	1	-	1	1	3	-	15	37 2	33	2	29
daho	-	-	-	-	1	-	-		_	7	-	-	2
Yyo.	-	-	-	-		-	-	-		4	1	-	
Colo. N. Mex.	7 =	2	4	-	1	1.1	-	-	4	6 3	3	1	13
N. Mex. Ariz.	2	-	NN	- 2	2	ī	- E -	- 2 -	7	5	17	- 1	
Jtah	-	-	-	-	-	_	-	-	2	2	9	-	
lev.	1	-	-	-	-	-	-	-	2	8	3	-	3
ACIFIC	73	-	25	-	1	1	6	2	56	103	45	5	47
Nash. Dreg.	10	1	10	12	-	1.2	6	-	11	14	1	- 2	26
Calif.	54	-	2	10.1	-	1		2	38	85	41	5	431
Alaska	2	-	5	-	1	-	-	-	-	-	-	-	1
lawaii	7	-	7	1.	-			-	4	4	-	-	
Juam (NA L	NA	NA	NA		NA	-	-	NA	NA	NA	NA	,
P.R. I	NA	NA	NA	NA	-	NA	-	-	NA	NA	NA	NA	ę
V.I. Pac. Trust Terr.	NA	NA NA	NA NA	NA	1.2	NA NA	-	- C -	NA NA	NA NA	NA NA	NA NA	-12

NN: Not notifiable. NA: Not available.

All delayed reports and corrections will be included in the following week's cumulative totals.

MMWB

TABLE III (Cont.'d). Cases of specified notifiable diseases, United States, weeks ending August 29, 1981 and August 23, 1980 (34th week)

REPORTING AREA	N	EASLES (R	UBEOLA)	MENIN	GOCOCCAL I TOTAL	NFECTIONS		MUMPS	PERTUSSIS	RU	BELLA	TETANUS
REPORTING AREA	1981	CUM. 1981	CUM. 1980	1981	CUM. 1981	CUM. 1980	1981	CUM. 1981	1981	1981	CUM. 1981	CUM. 1981
UNITED STATES	13	2,624	12.747	33	2,485	1,899	34	3,043	26	7	1,702	39
NEW ENGLAND		76	669	5	176	110	3	142	2	-	104	2
Maine		5	33	1	20	5		29	ī	-	33	-
N.H.		6	331	3	20	6	1	18	-		35	
Vt. Mass.	1	1 56	226 55		6 55	13	ī	33	ī	- 21	24	- 22
R.I.		-	2	1	15	38	- ÷ -	20	1	-		-
Conn.	-	8	22	-	60	41	1	36	-	- 1	12	2
MID. ATLANTIC	3	795 210	3.748	7	346	329	- 3	543	2	1.2	202	2
N.Y. City	2	72	1,170	3	59	80	1 2	103	2	- 21	49	i
N.J.		55	825	2	79	73		83	2		46	-
Pa.	1.70	458	1,073	2	97	68	-	285	-	-	11	-
E.N. CENTRAL Ohio	12	78	2,393	1.5	291	240	6	844	2	12	348	7
Unio Ind.	- 2	15	373	-	108	72	2	135	2		3 123	1 2
111.	020	23	332		72	66	3	171	-	- 2	83	-
Mich.	- 1	30	235	-	67	53	1	298	-	-	34	3
Wis.		2	1,363	-	4	13	-	146	et -		105	1
W.N. CENTRAL	25	6	1,327		106	74	2	166	T	1	75	3
Minn. Iowa	- 21	2	1.093	-	37	18	2	43	-	- 21	6	2
Mo.	- 21	1	- 64	- 21	33	- 33		15	1 2 2		2	1
N. Dak.	-	301 E.	-	1. 2.	ĩ	1				1.4		1
S. Dak.	-	-	-	-	- 4	4		1	1		-	-
Nebr. Kans.	- 2.5	1	83 67	1.21	13		- 1	3 96	1	1	62	
S. ATLANTIC	3	358	1.862	15	561	454	6	433	3	1	134	
Del.		-	3		4	2	-	9	- 14 me	-	1	-
Md.	1	5	71	-	40	45	1	82	1	-	1	-
D.C. Va.		1	298	1	3	1	- 2	2	1.1	1	7	
W. Va.	1	7	298	6	71 23	44	3	116	<u> </u>	- 2	22	120
N.C.		4	128	2	82	86	ĩ	15	- in the second	-	- 5	2
S.C.		2	159	1	71	- 53	-	10	-	-	8	2
Ga. Fla	ī	109 222	799 395	1 4	93 174	73	ī	33 91	3	1	35 55	1 3
E.S. CENTRAL		4	328	- 12	178	171		75	1		30	2
Ky.	12		53	-	48	53	1	37			19	-
Tenn.	-	2	169	-	50	45	-	20	- 10	1	10	
Ala.	-	2	22	-	57	46	× -	15			1	2
Miss.	-		84		23	27		3	1	- 1	-	-
N.S. CENTRAL	- 2 -	926	936	2	407	198	1	173	1	2	150	7
Ark. La.	- 21	1 2	16		22	17	-	1		- 2	2 9	1
Okia.	1.44	6	771	1	34	17	-	3.4	_		-	î
Tex.	- 4	917	138	1	252	92	- 1	168	1	2	139	3
MOUNTAIN		33	458	-	102	69		109		-	80	2
Mont. Idaho	-	ī	2		63	3	112	9	C 2 1		43	-
Nyo.	12.1		100	-	1	2		1		- 1	7	- 2
Colo.		9	24	-	35	17	-	42	7 5	-	27	-
N. Mex.	-		11	-	6	8	- - 1	100	1	- 1	5	-
Ariz. Jtah	- 2		366	- 21	19	12	1	24	1	-	19	1
Vev.	- 1	10		-	27	21	6 E I	13	-	- 2	10	-
ACIFIC	3	348	1,026		318	254	12	558	7	4	579	6
Wash.	-	3	174	-	59	47	1	138	i	- 1	94	- 3
Dreg.		110		-	47	44	1	62	4	7	48	-
Calif. Alaska	3	339	841 5	1	201	155	10	331	2	4	426	6
lawaii	-	2	6	-	4		-	20		-	10	- 77
Comment.						1.1.12						
Guam P.R.	NA NA	262	119	1	10	1 9	NA	109	NA	NA NA	1 3	3
/.1.	NA	25	6	-	1	1	NA	5	NA	NA	1	-
ac. Trust Terr.	NA	1	6		-	-	NA	9	NA	NA	1	

All delayed reports and corrections will be included in the following week's cumulative totals.

TABLE III (Cont.'d). Cases of specified notifiable diseases, United States, weeks ending August 29, 1981 and August 23, 1980 (34th week)

Later Vil	TUBE	RCULOSIS	TULA		HOID	(Tick-	S FEVER barne)			EAL DISEASES (T			RABIES (in
REPORTING AREA		CUM.	REMIA CUM.		VER CUM.	+	USF) CUM.		GONORRHEA	CUM.		CUM.	& Sec.) CUM.	Animals CUM.
0.0	1981	1981	1981	1981	1981	1981	1981	1981	1981	1980	1981	1981	1980	1981
UNITED STATES	561	17,574	150	12	329	40	937	18.690	648,263	637.399	571	19,562	17,015	4,81
NEW ENGLAND	15	496	1	1	13	-	8	320	15,960	15,946	3	388	341	24
Maine	- 4	33	-	-	1	-		22	617	913	1	3	4	13
N.H. Vt.	17.	13	-		-	-	-	16	563	570	-	11	1	4
Mass.	1	16			- 7		5	2	266	364	NA	13	5	
R.I.	10	293 29	1.2				1	NA 55	6,461 903	6,620 1,017	2	256 23	197	
Conn.	-	112	1	1	5	12	2	225	6,950	6,462	-	82	113	-
MID. ATLANTIC	62	2,762	10	1	53	3	37	2,233	77,537	67,815	81	2,925	2,417	63
Upstate N.Y.	10	501	10		- îî	ĩ	13	449	13.114	12,459	15	264	201	4
N.Y. City	32	1,067	-	1	28	-	3	1.095	32,105	25,841	36	1,750	1.586	100
N.J. Pa.	28	572	-	-	10	-	9	199	14,539	12, 362	13	404	293	12
Pa.	12	622	-	-	4	2	12	490	17,779	17,153	15	507	337	4
E.N. CENTRAL	101	2.302	1	1	23	-	44	2,682	96,515	98,284	50	1,339	1,580	662
Ohio	14	459	-	1	- 4	-	36	555	32.047	25,690	-	193	250	51
Ind. III.	16	230	112	- 21			2	238	8,412	10.156	22	153	129	72
Mich.	32	883	1		11	-	5	378	25,970 21,249	30,760 22,319	24	697	886	448
Wis.	5	125	1		2			351	8,837	9,359	1	64	60	81
W.N. CENTRAL	24	635				-								
Minn,	- 1	115	21	1.2	12		38	1,298	31,225	29,340 4,908	11	403	75	2,015
lowa	i.	69	-	-	3	_	÷	130	3,363	3,234	1	16	14	634
Ma.	6	277	17	- 1	2	-	20	590	14.527	12,736	7	216	102	176
N. Dak.	-	23	-	-	12.1	-		5	407	421	10-		3	313
S. Dak.	-	- 44	-	-	1	-	-	39	856	899	-	2	2	236
Nebr.	-	19	3		2	-	3	37	2,359	2, 302	-	5	6	150
Kans.	8	88	1		2	-	9	279	4,847	4,840	1	19	6	151
S ATLANTIC	108	3,850	12	3	47	29	538	4.615	160.443	159,649	183	5,214	4.041	337
Md.	12	54	1	- 7	4.7	- 7	2	68	2,554	2.207		8	10	1
D.C.	12	387	1.5	1	14	1	49	423	18,381 9,436	17.161	13	385	285	24
Va.	8	395	2		- î	9	95	515	14.767	14,012	15	461	367	66
W. Va.	2	122	-	1	ŝ		5	65	2,419	2,139		16	15	16
N.C.	17	681	2	-	í	9	226	820	24,942	22.648	12	397	280	
S.C.	6	354	3			5	89	457	15.664	15,204	6	336	230	21
Ga.	30	638	- 4	-	4	5	64	1,123	33,103	30,940	37	1,332	1,151	145
Fla.	25	974	1.7	1	21	- 1	8	933	39,177	44,179	85	1,856	1,405	58
E.S. CENTRAL	48	1,545	6	-	7	5	99	1,580	53.900	52,186	29	1,294	1,402	305
Ky.	6	393	2	-		-	2	174	6,709	7,695	3	63	100	96
Tenn. Ala.	16	514	4	-	3	4	64	627	20,486	18,702	12	490	591	157
Miss.	9	409	1		2	1	14	477 302	16,264	15,500	7	363 378	297	56
WEGENERAL														
W.S. CENTRAL Ark.	59	1.992	68 39		48	2	142	2,360	85,709 6,354	81,998	125	4.702	3,390	817
La,	20	362	2	1	2	1	32	348	14,566	14,845	45	1,107	619	21
Okla.	13	241	15		ŝ	1	61	385	9,335	6.141	4	110	66	159
Tex.	21	1.180	12	3	38	-	29	1,431	55,454	52,662	73	3,393	2,397	518
MOUNTAIN	21	505	26	-	21	1	26	798	25.401	24,807	37	515	392	157
Mont.		27	5	-	-4		12	26	919	938	-	11	ī	84
Idaho	-	6	4	-	-	-	5	29	1,127	1.088	-	17	15	1
Wya.	-	7	1	-	-	1	6	11	575	717	1	8	8	13
Colo. N. Mex.	3	53	6	-	6	-	-	213	6,884	6,691	4	153	107	19
Ariz,	5	97	1			-	12.5	88	2,729	3.012	1	93	64	21
Utah	6	236		1	10	-	ī	182	7,630	6,750 1,214	18	123	129	13
Nev.	2	39	ĩ	-	1	-	2	188	4,324	4,397	10	90	57	1
PACIFIC	103	3,487	5	2	105			2,804	101,573	107,374	52	2.782	3,242	422
Wash.	103	256	ĩ	1	105		ĩ	219	8.211	9,082	22	21/82	166	11
Oreg.	i.	129	-	-	- 4	-		193	6.059	7,186	-	61	69	1
Calif.	86	2,965	4	2	97	-		2.305	82.768	86,366	52	2,572	2,891	390
Alaska Hawaii	-	44	-	-	-	-	-	87	2,563	2,582	-	9	1	14
	2	93		-	1	-	-	NA	1.972	2,158	NA	46	109	
				1.0			100							- 24
Guam	B4 4													
Guam P.R.	NA	219	-	NA NA	-	NA NA		NA	2-055	89	NA	430	281	
Guam P.R. V.I. Pac. Trust Terr.	NA NA NA	219 1	Ē	NA NA	4	NA NA		NA	2.055 131	1,723	NA NA	430	381	53

NA: Not available. All delayed reports and corrections will be included in the following week's cumulative totals.

TABLE IV. Deaths in 121 U.S. cities,* week ending August 29, 1981 (34th week)

		ALL CA	USES, BY	AGE (YE	ARS)					ALL C	AUSES, BY	AGE (YE	ARS)		
REPORTING AREA	ALL AGES	≥65	45-64	25-44	1-24	<1	P&1** TOTAL	REPORTING AREA	ALL AGES	≥65	45-64	25 44	1-24	<1	P 8 I* TOTA
NEW ENGLAND	587	411	129	17	14	16	40	S. ATLANTIC	1,165	762	277	53	43	46	34
Boston, Mass.	180	111	47	6	6	10	20	Atlanta, Ga.	139	82	32	13	7	5	2
Bridgeport, Conn.	52	28 19	12		1	1	5	Baltimore, Md. Charlotte, N.C.	165	99	37	17	8	4	3
Cambridge, Mass. Fall River, Mass.	22	15	é	-	1	-	-	Jacksonville. Fla.	77	46	21 2C	2 5	4	4	5
Hartford, Conn.	40	30	9	-	ī	-	-	Miami, Fla.§	106	87	20	ร์	5	5	í
Lowell, Mass.	22	16	5	1	-	-	-	Norfolk, Va.	54	28	16	6	2	2	1
Lynn, Mass.	17	10	6	1		-	1	Richmond, Va.	67	28	25	1	2	5	6
New Bedford, Mass New Haven, Conn.	. 1E 41	13	15	1	1	2	1	Savannah, Ga. St. Petersburg, Fla.	47	28	13	2	3	1	25
Providence, R.1. §	60	56	12	2	-	2	4	Tampa, Fla.	65	29	14	ŝ	1	2	4
Somerville, Mass.	é	5	-	1		-	-	Washington, D.C.	197	91	72	21	6	- 7	3
Springfield, Mass.	32	24	4	2	1	1	3	Wilmington, Del.	57	29	17	4	2	5	-
Waterbury, Conn. Worcester, Mass.	26	18	7	1	ī	1	3								
WORCESTER, Mass.		20	,	_	•	-	-	E.S. CENTRAL	615	365	169	52	17	12	22
								Birmingham, Ala.	87	54	24	4	2	3	3
MID. ATLANTIC		1.652	526	167	67	52	73	Chattanooga, Tenn.	57	28	17	7	5	-	3
Albany, N.Y.	49	37	?	1	-	4	1	Knoxville, Tenn.	51	37	12	1	1		7
Allentown, Pa. Buffalo, N.Y.	20	12	32	3 8	4	5	9	Louisville, Ky. Memphis, Tenn.	92 130	53 73	25 40	8 12	- 4	2	4
Camden, N.J.	37	22	ŝ	5	- 201	ĩ	2	Mobile, Ala.	55	36	12	12	_	-	2
Elizabeth, N.J.	27	15	é	1	1	-	4	Montgomery, Ala.	45	26	12	i	-	-	1
Erie, Pa.†	40	32	4	3	1	-	1	Nashville, Tenn.	SE	58	27	£	2	5	6
Jersey City, N.J. N.Y. City, N.Y.	41 1,288	27 879	1C 267	2 50	35	17	1 29								
Newark, N.J.	62	25	14	e	1	4	1	W.S. CENTRAL	1,063	610	267	53	63	30	37
Paterson, N.J.	26	14	é	4	i	i		Austin, Tex.	49	34	Ĩ.e	3	3	ĩ	3
Philadelphia, Pa.t	320	190	εe	22	8	12	13	Baton Rouge, La.	35	21	10		3	1	-
Pittsburgh, Pa. † Reading, Pa.	63	43	12	6	2	2	-	Corpus Christi, Tex.	28	20	. 4	2	1	1	ī
Rochester, N.Y.	100	69	23	4	ź	2	6	Dallas, Tex.	177	104	46	10	14	3	2
Schenectady, N.Y.	16	12	- 3	i	-		-	El Paso, Tex. Fort Worth, Tex.	74	46	22	5	1	- 2	7
Scranton, Pa.1	21	13	6	1	1	-	1	Houston, Tex.	153	68	45	22	12	6	2
Syracuse, N.Y.	81	55	18	2	3	3	-	Little Rock, Ark.	62	27	23	8	Э	1	3
Trenton, N.J. Utica, N.Y.	49 20	37	5 1	2	- 2 - 1	1	5	New Orleans, La.	104	63	21	10	4	6	10
Yonkers, N.Y.	17	14	ź	-	-	-	-	San Antonio, Tex. Shreveport, La. Tulsa, Okla.	58	1C2 32 57	4C 17 17	13 6 5	5	8	3
	2, 181	1, 319	532	153	78	99	55								
E.N. CENTRAL Akron, Ohio	77	55	11	4	2	5	~~	MOUNTAIN	537	338	115	35	36	13	20
Canton, Ohio	22	17	4	i	-	-	2	Albuquerque, N. Mex		30	lé	3	4	13	5
Chicago, III.	535	286	134	54	28	37	15	Colo. Springs, Colo.	28	19	7	1	- i		1
Cincinnati, Ohio	172	56	47	16	6	7	8	Denver, Colo.	113	73	20	7	9	- 4	1
Cleveland, Ohio	169 87	57	51 18	12	3	9	3	Las Vegas, Nev.	55	26	14	8	5	2	12
Columbus, Ohio Dayton, Ohio	97	68	18	é	î	2	-	Ogden, Utah Phoenix, Ariz.	11 120	7	1 28	1	6	2	ĩ
Detroit, Mich.	240	139	62	21	10	8	2	Pueblo, Colo.	15	16	2	-	ĭ	-	1
Evansville, Ind.	42	28	12	1	1	1	-	Salt Lake City, Utah	40	22	8	4	5	1	
Fort Wayne, Ind.	40 1 E	28	ç	ī	1	2	5	Tucson, Ariz.	98	69	19	3	5	2	8
Gary, Ind. Grand Rapids, Mich		40	10	4	-	2	2								
Indianapolis, Ind.	158	51	47	4	9	7	3	PACIFIC	1,663	1,089	373	55	61	35	69
Madison, Wis.	36	20	11	1	2	2	2	Berkeley, Calif.	24	17	4	1	1	1	-
Milwaukee, Wis.	141	103	30	1	5	2	-	Fresno, Calif.	56	37	15	1	2	3	5
Peoria, III. Rockford, III.	27	22	4	2	1 2	2	1	Glendale, Calif.	32	25	, é	-	1		
South Bend, Ind.	45	33	12	- <u>5</u> =	1	-	3	Honolulu, Hawaii Long Beach, Calif.	70	39 58	15	8	3	5	4
Toledo, Ohio	123	78	22	12	3	8	8	Los Angeles, Calif.	464	313	105	28	12		14
Youngstown, Ohio	50	32	14	3	1	-	-	Oakland, Calif. Pasadena, Calif.	77 15	55 15	14	é 1	2	1	1
W.N. CENTRAL	715	461	166	42	20	26	27	Portland, Oreg.	157	103	35	10	6	3	1
Des Moines, Iowa	88	50	25	ΞÊ.	4	1		Sacramento, Calif. San Diego, Calif.	125	36	40	é	1	2	- 4
Duluth, Minn.	37	29	é	1	i		5	San Francisco, Calif.	137	74	38	15	5	5	3
Kansas City, Kans.	32	20	7	4	ī	-	3	San Jose, Calif.	145	57	26	5	13	- 4	13
Kansas City, Mo.	115	79	28	?	2	3	6	Seattle, Wash.	125	50	28	8	6	3	5
Lincoln, Nebr. Minneapolis, Minn.	28	21	5 14	1	3	1 5	2	Spokane, Wash.	43	32	é	3	-	2	4
Minneapolis, Minn. Omaha, Nebr.	77	54	14	3	3	3	2	Tacoma, Wash.	35	22	8	1	2	2	- 6
St. Louis, Mo.	135	15	37	8	3	8	5		1.20						
St. Paul, Minn.	ÉÉ	43	13	4	2	- 4	- 1	TOTAL	10.951	6.947	2.554	751	999	333	377
Wichita, Kans.	45	26	17	-	1	ı	1								

*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

tBecause of changes in reporting methods in these 4 Pennsylvania cities, these numbers are partial counts for the current week. Complete counts will be available in 4 to 6 weeks.

ttTotal includes unknown ages.

§Data not available this week. Figures are estimates based on average percent of regional totals.

ACIP Recommendation on Viral Hepatitis – Continued

Recommendations on prophylaxis can, thus, be categorized as to 1) whether the source of blood is known or unknown and 2) whether the HBsAg status of the source blood is known or unknown. The following outline and summary table (Table 3) are based on these categories. Management of each exposure must be individualized in view of the number of contributing factors. Furthermore, it is important to emphasize that for greatest effectiveness, globulin should be given promptly (its value beyond 7 days of exposure is unclear). A. Source known, HBsAg status positive.

HBIG (0.06 ml/kg) should be given immediately, ideally within 24 hours of exposure. A second identical dose should be given 1 month later. (If HBIG is not available, IG should be used in the same dose and schedule.)

B. Source known, HBsAg status unknown.

Two decisions are involved here: whether to test for HBsAg and which immune globulin to give. Because these decisions relate both to the relative probability that the source will be HBsAg-positive and to the inherent delay in testing, the following operational guidelines are suggested:

1. High risk that the source is HBsAg-positive-such as associated with patients with acute, unconfirmed viral hepatitis; patients institutionalized with Down syndrome; patients on hemodialysis; persons of Asian origin; male homosexuals; users of illicit, intravenous drugs.

If HBsAg test results can be known within 7 days of the exposure, IG (0.06 ml/kg)

Exposure	HBsAg Testing	Recommended prophylaxis
HBsAg positive		HBIG (0.06 mI/kg) immediately and 1 month later
HBsAg status unknown Source known:		
High Risk†	Yes‡	IG (0.06 ml/kg) immediately, and if
		—TEST POSITIVE— HBIG (0.06 mI/kg) immediately and
		1 month later or if
		-TEST NEGATIVE- Nothing
Low Riskt	Νο	Nothing or IG (0.06 ml/kg)
HBsAg status unknown		
Source unknown	No	Nothing or IG (0.06 ml/kg)

TABLE 3. Summary of postexposure prophylaxis of acute exposures to HBV*

Important details are in the text.

[†] Characterized in text.

[‡] If results can be known within 7 days of exposure.

ACIP Recommendation on Viral Hepatitis – Continued

should be given immediately, certainly within 24 hours. If test results are positive, HBIG (0.06 ml/kg) should be given at that time and again 1 month later.

If HBsAg test results cannot be known within 7 days of the exposure, the decision to use IG or HBIG must be based on the clinical and epidemiologic characteristics of exposure and the availability of globulin, remembering the importance of characterizing the source and giving globulin as soon after exposure as possible.

2. Low risk that the source is HBsAg-positive—such as associated with the average hospital patient.

Prophylaxis is optional; HBsAg testing is not recommended. If an immune globulin is to be used, IG (0.06 ml/kg) should be given promptly, certainly within 24 hours. No further action is necessary.

C. Source unknown, HBsAg status unknown.

Prophylaxis is optional. If an immune globulin is to be used, IG (0.06 ml/kg) should be given promptly, certainly within 24 hours. No further action is necessary.

Exposure of the newborn: Infants born to HBsAg-positive mothers (especially mothers who are HBeAg positive) are at risk of being infected with HBV and becoming chronic carriers. Recent studies have shown that the carrier state can be prevented in about 75% of such infections if newborns are given HBIG immediately after birth (20). (IG was not included in the protocol.)

All infants born to HBsAg-positive mothers should be given HBIG, *total dose* 0.5 ml intramuscularly, as soon after birth as possible (no later than 24 hours). The same dose (0.5 ml) should be repeated 3 months and 6 months later.

Sexual contact with persons with hepatitis B: In only 1 study has there been any evaluation of the value of immune globulin for sexual contacts of patients with acute hepatitis B (21). Although results suggest protection with HBIG, additional studies comparing IG, HBIG, and placebo groups are needed before specific recommendations can be made.

Pre-Exposure Prophylaxis:

Staff and patients of hemodialysis units: Routine passive immunization against hepatitis B is not recommended for staff and patients of hemodialysis units. Instead, precautions such as serologic screening of patients and staff, segregation of carriers, and environmental hygiene should be encouraged. In the rare event that such measures fail to interrupt transmission, prophylaxis with an immune globulin may be considered. Because carefully controlled studies have failed to demonstrate an advantage of HBIG over IG in this setting, IG (0.05-0.07 ml/kg) every 4 months is recommended for patients and staff (22).

Staff and patients of institutions for custodial care of the developmentally disabled: HBV is commonly endemic in institutions for the developmentally disabled, but passive immunization is not routinely recommended for staff or clients unless it is shown that hepatitis B cannot be controlled by environmental measures alone. Then IG may be administered in the same dose and at the same intervals as for patients and staffs of hemodialysis units.

HEPATITIS NON A/NON B AND HEPATITIS-NONSPECIFIC

Without accurate tests for diagnosing non A/non B viral hepatitis, the value of prophylaxis with immune globulins cannot be determined. No specific recommendation can be

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ACIP Recommendation on Viral Hepatitis – Continued

made, but as with hepatitis that cannot be specifically diagnosed (hepatitis-nonspecific), it is reasonable to apply the recommendations for prophylaxis against hepatitis A.

References

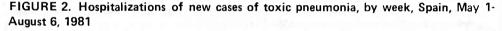
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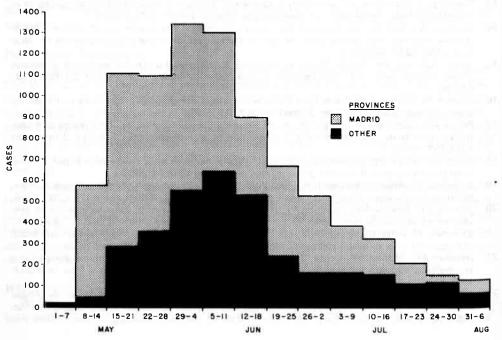
International Notes

Follow-Up on Toxic Pneumonia - Spain

From May 1 to August 6, 1981, 12,147 persons with "toxic pneumonia" were hospitalized in Spain, mostly in the province of Madrid and the provinces of Valladolid, León, Palencia, Avila, and Segovia (Figure 2) (1). The illness was initially called atypical pneumonia because clinical and X-ray findings were compatible with that diagnosis. Most commonly reported symptoms included fever, muscle pains, a variable rash, pruritis, and marked eosinophilia (>1,500 eosinophils/mm³ for 67% of patients in 1 survey). Convalescence was protracted, marked at times by severe myalgia; 10%-25% of patients required rehospitalization. Mortality has remained <1% for hospitalized patients.

Initial microbiologic, serologic, and pathologic studies, including those for trichinosis, did not incriminate food in this outbreak. Subsequently, a survey of hospitalized patients showed a very high frequency of use of illicit, bargain, unlabeled oil that had been sold from house to house as olive oil. Other hospital surveys confirmed this finding, and a case-control study was undertaken in the town of Navas del Marqués (province of Avila) on June 11. All the families of patients gave a history of consuming this oil, compared with 12 of 54 size-matched families ($X^2 = 40.55$, p<0.001) and 16 of 54 randomly selected families ($X^2 = 31.94$, p<0.001). In families of patients, there was a statistically significant direct relationship between personal daily consumption of oil and illness ($X^2 = 13.47$, p=0.001). There was no significant association between consumption of





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Toxic Pneumonia – Continued

fried foods and illness; however, the consumption of salads (dressed with raw oil) was associated with illness ($X^2 = 5.30$, p<0.05).

A resurvey of the same families on July 9 to compare families of patients with families who had also consumed the implicated oil but had remained well showed that the attack rate for families of patients had increased in the interim (from 51% to 62% of family members). During efforts to define a particular source of the incriminated oil, investigators learned that 24 (75%) of 32 families of patients had bought oil from a particular salesman in April and May, while only 5 (20%) of 25 control families reported having the same supplier ($X^2 = 16.34$, p<0.001). Again, a significant association was found between illness and the consumption of vegetables with raw oil. This time there was no statistically significant association between estimated weekly consumption of oil and an increase in the number of cases; however, there was a direct relationship between the former and continuing illness ($X^2 = 7.52$, p<0.01). No association was found between illness and doing the cooking or being in the kitchen when food was being cooked, but, as in the previous survey, nearly all families in both groups used olive oil when frying food.

Samples of the implicated oil taken from the homes of patients were analyzed at the Spanish National Center for Foods and Nutrition and other specialized laboratories in Madrid (Central Customs Laboratory, National Institute of Toxicology) and Seville (Institute for the Study of Fats and Derivatives). The oil was found to be a mixture of rape-seed oil, liquified pork fat, and a small amount of low-quality olive oil-possibly that obtained from the final pressing of olives including seeds (called "orujo"). The mixture contained abnormally high levels of aniline, anilide-oil complexes, and azoben-zene. Animal toxicity experiments and further analyses of the oil for toxic substances are in progress. Spanish authorities are continuing their efforts to determine how this product was contaminated with rape-seed oil.

Both the sale without label and the adulteration of olive oil with rape-seed oil are prohibited by Spanish law. Legal and administrative measures have been taken to stop the distribution of this product. The public has been advised repeatedly to discard the implicated oil, and the number of hospitalizations for toxic pneumonia has since decreased. On June 29 and 30, consumers were requested to exchange their remaining supplies of the implicated oil for oil provided by municipal authorities in an operation organized by the Ministry of Health. Most of the new hospitalizations since July 1 have been of patients who became ill before June 11 and a small proportion of consumers who have continued to use the oil.

Reported by L Valenciano, MD, Director General of Public Health, Ministry of Labor, Health and Social Security, Madrid; Center for Environmental Health, Center for Infectious Diseases, Epidemiology Program Office, CDC.

Editorial Note: Spanish health officials are not aware of a similar outbreak of clinical illness associated with contaminated oil in their country.

Foodborne disease outbreaks are, of course, common, and there have been previous reports of illness associated with the use of cooking oil (2). Usually such outbreaks have resulted from replacing cooking oil with a cheaper but toxic substitute such as fuel oil Or from cooking oil that was contaminated with toxic substances such as polychlorinated biphenyls.

In this outbreak, the specific agent has not been identified, although there is strong epidemiologic evidence of an association between illness and consumption of the lowcost, contaminated "olive oil." The identified contaminants in the oil-rape-seed oil and

Toxic Pneumonia – Continued

pork fat-have not been associated previously with clinical illness. Rape-seed oil, extracted from the seed of *Brassica napus*, a member of the cabbage family, is commonly used in preparing food, making soap, and producing high-temperature lubricating oils. The seed does contain glucosinolates that, in some animals, may be converted to toxic thiocyanates and isothiocyanates. However, the clinical syndrome of this current illness does not resemble cyanate compound toxicity. The significance of pork fat in the olive oil, other than being an indication of the oil's quality, is unclear.

The other contaminants—aniline, azobenzene, and anilide-oil complexes—were present in low concentration, i.e., <100 parts/million, levels not usually associated with clinical illness. How the oil was contaminated with aniline and azobenzene is unclear, but it has been reported that an aniline extraction technique is sometimes used to remove unpalatable flavor from rape-seed oil. The anilide-oil complexes may represent compounds resulting from the mixture of aniline and oil.

Some rape-seed oil contains high concentrations of erucic acid. Such oil has produced necrosis of the myocardium, anemia, and stunted growth in animals—effects quite different from those observed in the outbreak reported here (i.e., a total of 14 oil samples contained $\leq 1\%$ erucic acid, a concentration compatible with edible rape-seed oil).

The importance of cooking or frying the oil is not clear. Heat might be a factor in creating toxic compounds or might lead to the inhalation of toxins, but the surveys failed to establish a differential in attack rates between exposure to cooked and uncooked oil.

The high levels of circulating eosinophils suggest an allergic response, but no allergen has been identified in studies thus far. Animal studies are continuing in an effort to identify a toxin and/or an allergen.

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Current Trends

Surveillance of Childhood Lead Poisoning - United States

During the second quarter of fiscal year 1981, 59 childhood lead-poisoning prevention programs reported that 127,600 children were screened and 3,785 were identified with lead toxicity (Table 4). For the first 6 months of the year, 253,837 children were screened, 10,492 were found to have lead toxicity, and 10,789 were referred to care for iron deficiency. In each of the first 2 quarters, almost 20,000 urgent-, high-, and moderate-risk children were under pediatric management for lead toxicity.

As of March 31, 1981, 6,050 urgent- and high-risk children were under clinical management for lead toxicity. Of these children, 5,076 had an environmental investigation performed in their behalf; 4,621 had their probable source of lead exposure identified, and 3,056 had the source of exposure reduced.

Reported by the Environmental Health Services Div, Center for Environmental Health, CDC.

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Childhood Lead Poisoning - Continued

TABLE 4. Results of screening in childhood lead-poisoning control programs, United States, second quarter fiscal year 1981 (January 1-March 31, 1981)

			132 101	Number of a	hildren	weak shaked	Number	of dwellin children w	gs related
		· · · · · ·	With I	ad toxicity*	Der Tribtan	Life and		ead toxicit	
0	1 . A . A	Requirin	g pediatric i	management	Receiving	Identified	Contractor	Found	
Programs	Screened	Total	Class II	Classes III & IV	pediatric management†	with iron deficiency	Inspected	with	Reduce
ridgeport, Conn.	1,144	22	12	10	127	16	25	23	6
Vaterbury, Conn.	720	8	7	1	152	51	35	27	16
oston Mass	4,636	89	61	28	865	180	107	103	49
awrence, Mass.	1 867	71	66	5	282	13	57	52	39
orcester, Mass	1,587	23	18	5	121	9	30	30	17
hode Island State	1,806	64	26	38	510	131	71	58	45
EGION I TOTAL	11,760	277	190	87	2,057	400	325	293	172
Cumulative FY 81	23,399	794	522	272		762	645	594	369
tlantic City, N.J.	310	23	11	12	36	2	11	10	10
amden, N.J.	865	16	2	9	296	29	38	20	42
ast Orange, N.J.	881	28	20	8	164	103	19	15	15
rsey City, N.J.	1.047	105	59	46	204	47	45	36	34
ong Branch, N.J.	276	5	4	1	40	i g	16	10	9
ewark, N.J.	1,955	259	189	70	850	142	74	74	75
aterson, N.J.	1,269	75	57	18	699	115	59	47	68
ainfield, N.J.	793	31	25	6	102	25	25	20	4
rie Co., N.Y.	1 995	92	76	16	214	17	94	28	8
onroe Co., N.Y.	1,475	56	43	13	283	23	32	27	88
ew York City	29,307	973	721	252	2,401	2,551	175	91	131
nondaga Co., N.Y.	1,775		17	252	2,401	119	47	42	20
estchester Co., N.Y.	1.452	22	26	9	288	108	38	32	27
EGION II TOTAL	43,400	1,720	1,255	465	5,866	3,290	673	452	531
Cumulative FY 81	85,939	4,389	3,084	1,305	5,000	5,911	1,892	1,326	1,126
					222		26	1,320	7,120
elaware State	1,048	25 22	21	4	233 244	64	26	38	21
ashington, D.C.	2,565 6,041	87	15 57		244 641	169 27	82	38 69	49
altimore, Md.				30				0	49
llentown-Bethlehem, Pa.	784	4	2	2	25 144	133	22	17	13
hester, Pa.		11				13			
niladelphia, Pa.	5,814	713	480	233	2,597	79	143	136	112
ilkes-Barre, Pa.	484	12	9	3	90	41	37	26	20
ork, Pa.	284	11	9	2	32	14	11	11	6
vnchburg, Va.	280	1	1	0	61	7	4	2	2
ewport News, Va.	606	5	3	2	32	38	25	14	25
orfolk, Va.	1,005	8	6	2	239	23	21	12	9
ortsmouth, Va.	703	12	6	6	110	18			29
ichmond, Va.	1,276	16	9		165	28	35	29	
EGION III TOTAL	21,477	927	627	300	4,613	654	473	371	300
Cumulative FY 81	38,444	2,267	1,470	797		1,262	1,038	825	585
ugusta, Ga.	822	8	6	2	89	29	11 =	10	7
ouisville, Ky.	2,650	32	26	6	342	75	49	47	26
abarrus Co., N.C.	190	2	1	-1	15	7	6	6	3
outh Carolina State	7,366	61	38	23	316	13	47	35	29
EGION IV TOTAL	11,028	103	71	32	762	124	113	98	65
Cumulative FY 81	20,290	237	155	82		298	287	242	205
hicago, III.	NA	1				1			
. (other local programs)‡	1,206	23	11	12	36	0	15	7	1
ankakee, III.	540	12	8	4	39	130	6	6	0
adison Co., III.	549	17	12	5	59	27	10	6	0
ockford, III.	470	2	2	0	90	16	6	4	23
aukegan Lake Co., III.	948	10	5	5	28	36	20	19	7
t. Wayne, Ind.	175	6	2	4	63	1	13	3	0
etroit, Mich.	4,489	109	66	43	433	27	165	112	178
ayne Co., Mich.	354	12	9	3	79	8	14	11	1
kron, Ohio	836	10	10	0	149	62	9	5	8
incinnati, Ohio	2,343	38 .	23	15	328	99	92	8	10
eveland, Ohio	3,630	120	89	31	677	345	88	29	26
eloit, Wis.	141	3	3	0	21	10	3	2	1
ilwaukee, Wis. EGION V TOTAL	1,926	82	53	29	360	51	106	91	66
EGION V TOTAL	17,607	444	293	151	2,362	812	547	303	321
Cumulative FY 81	47,464	1,989	1,286	703	ALC: DOLLARS	1,714	1,991	959	1,144
rkansas State	3,144	29	16	13	147	93	42	27	51
puisiana State	8,707	5	2	3	19	NA	5	5	0
ew Orleans, La.	3,196	44	30	14	610	109	80	56	72
ouston, Tex.	1,404	7	6	1	163	7	7	0	0
EGION VI TOTAL	16,451	85	54	31	939	209	134	88	123
Cumulative FY 81	25,651	194	115	79	and the second second	412	272	189	276
edar Rapids-Linn Co., Iowa	633	9	7	2	69	10	12	12	11
avenport-Scott Co., Iowa	442	2	1	1	51	9	7	7	4
Louis, Mo.	2,610	191	124	67	2,651	131	524	347	276
pringfield, Mo.‡	256	9	4	5	9	15	14	6	0
maha-Douglas Co., Neb.	675	16	13	3	143	13	77	66	40
EGION VII TOTAL	4,616	227	149	78	2,923	178	634	438	331
Cumulative FY 81	9,454	615	386	229	10-1	222	1,317	889	962
os Angeles, Calif.	1,261	2	Ō	2	54	101	27	4	2
EGION IX TOTAL	1,261	ź	ŏ	2	54	101	27	4	2
Cumulative FY 81	3,196	7	1	6		208	89	20	13
		· ·			10.05				
S. TOTAL	127,600	3,785	2,639	1,146	19,576	5,768	2,926	2,047	1,845

"Screening Class II and Classes III & IV defined in CDC statement, "Preventing Lead Poisoning in Young Children," April 1978. 1Not cumulative

FReporting program not receiving lead-poisoning prevention grant support NA - Not available

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