

MMWR

MORBIDITY AND MORTALITY WEEKLY REPORT

- 285 ACIP: Post-Exposure Prophylaxis of Hepatitis B
- 290 National Plague Conference Report
- 295 Declining Rates of Rectal and Pharyngeal Gonorrhea Among Males—New York City
- 297 Perinatal and Maternal Mortality in a Religious Group—Indiana
- 298 Lymphocytic Choriomeningitis—Georgia

Recommendation of the Immunization Practices Advisory Committee (ACIP)

Postexposure Prophylaxis of Hepatitis B

The following statement supplements and updates certain sections of two previous statements on hepatitis B virus prophylaxis (MMWR 1981;30:423-35 and MMWR 1982;31:317-28 [1,2]). Those statements should be consulted regarding preexposure use of hepatitis B vaccine and prophylaxis of hepatitis A.

INTRODUCTION

Prophylactic treatment to prevent hepatitis B (HB) infection after exposure to hepatitis B virus (HBV) should be considered in several situations: perinatal exposure of an infant born to a hepatitis B surface antigen (HBsAg)-positive mother, accidental percutaneous or permucosal exposure to HBsAg-positive blood, or sexual exposure to an HBsAg-positive person. In each of these settings, the risk of HB infection is known to be high and justifies preventive measures. Previous recommendations for postexposure prophylaxis have relied on passive immunization with specific hepatitis B immune globulin (HBIG) (1). However, the recent demonstration of high efficacy of HB vaccine combined with HBIG in preventing chronic HB infection in infants of HBsAg-positive mothers requires the revision of recommendations for postexposure prophylaxis (3) (Table 1).

Passive immunization with HBIG alone has been partially effective in preventing clinical HB in studies of medical personnel after needlestick accidents (4) and sexual exposure to partners with acute HB (5). In addition, HBIG prophylaxis has been shown to significantly reduce the percentage of infants who become chronic HBV carriers after perinatal exposure to HBsAg-positive mothers (6). For perinatal and needlestick exposures, however, HBIG alone is only about 75% effective even when given very soon after exposure, may provide only temporary protection, and is costly (over \$150 per adult dose).

With the development of HB vaccine, the possibility arose that HB vaccine, alone or in combination with HBIG, might be useful for postexposure prophylaxis. Studies have shown that response to HB vaccine is not impaired by concurrent administration of HBIG and that the combination of HB vaccine and one dose of HBIG produces immediate and sustained high levels of protective antibody to the hepatitis B surface antigen (anti-HBs) (7). A recent study examining the efficacy of HB vaccine combined with a single dose of HBIG in preventing perinatal transmission from HBsAg carrier mothers who were also positive for hepatitis B "e" antigen (HBeAg) showed this combination to be highly effective in preventing the HBV carrier state in infants and significantly more effective than multiple doses of HBIG alone (3).

PERINATAL TRANSMISSION

Transmission from mother to infant during birth is one of the most efficient modes of HBV transmission. If the mother is positive for both HBsAg and HBeAg, about 80%-90% of infants

ACIP: Hepatitis B

will become infected. Although infection is rarely symptomatic in the acute phase, approximately 90% of these infected infants will become chronic HBV carriers. It has been estimated that 25% of these chronic carriers may die of cirrhosis or primary hepatocellular carcinoma (3). In addition, such persons are infectious, and female carriers may subsequently perpetuate the cycle of perinatal transmission. If the HBsAg-positive carrier mother is HBeAg-negative or if anti-HBe is present, transmission occurs in less than 25% and 12% of cases, respectively. Such transmission rarely leads to chronic HBV carriage; however, severe acute disease, including fatal fulminant hepatitis in the neonate, has been reported (8,9). Even if perinatal infection does not occur, the infant may be at risk of subsequent infection from other family contacts. For these reasons, prophylaxis of infants from all HBsAg-positive mothers is recommended, regardless of the mother's HBeAg or anti-HBe status.

The primary goal of postexposure prophylaxis for exposed infants is prevention of HBV carrier state. In addition, there is a need to prevent the rare occurrence of severe clinical hepatitis in some of these infants. Administration of 0.5 ml HBIG to an infant of an HBsAg, HBeAg-positive mother soon after birth and repeated at 3 months and 6 months reduces the probability of chronic infection from about 90% to about 25% (efficacy about 75%). The concurrent use of HB vaccine and various combinations of HBIG increases the efficacy to close to 90%. Since approximately 5% of perinatal infection may occur in utero, it appears likely that no form of postnatal prophylaxis will be 100% effective in this circumstance.

Concurrent HBIG and vaccine administration does not appear to interfere with vaccine efficacy. HB vaccine has been shown to be equally immunogenic in neonates, whether given in 10- μ g or 20- μ g doses. The use of HB vaccine in combination with HBIG in the perinatal setting has the advantages of increasing efficacy, eliminating the need for the second and third doses of HBIG, and providing long-term immunity to those who are not infected during the perinatal period.

TABLE 1. Hepatitis B virus postexposure recommendations

Exposure	HBIG		Vaccine	
	Dose	Recommended timing	Dose	Recommended timing
Perinatal	0.5 ml IM	Within 12 hrs of birth	0.5 ml (10 μ g) IM	Within 7 days*; repeat at 1 & 6 mos
Percutaneous	0.06 ml/kg IM or 5 ml for adults	Single dose within 24 hrs	1.0 ml (20 μ g) IM [†]	Within 7 days*; repeat at 1 & 6 mos
	or [§] 0.06 ml/kg IM or 5 ml for adults	Within 24 hours; repeat at 1 mo		
Sexual	0.06 ml/kg IM or 5 ml for adults	Within 14 days of sexual contact	¶	—

*The first dose can be given the same time as the HBIG dose but at a separate site.

[†]For persons under 10 years of age, use 0.5 ml (10 μ g).

[§]For those who choose not to receive HB vaccine.

[¶]Vaccine is recommended for homosexually active males and for regular sexual contacts of chronic HBV carriers.

*ACIP: Hepatitis B***Maternal Screening**

Since efficacy of this regimen depends on administering HBIG on the day of birth, it is vital that HBsAg-positive mothers be identified before delivery. Mothers belonging to groups known to be at high risk of HB infection (Table 2) should be tested routinely for HBsAg during a prenatal visit. If a mother belonging to a high-risk group has not been screened prenatally, HBsAg screening should be done at the time of delivery or as soon as possible thereafter.

Management of HBsAg-Positive Mothers and Their Newborns

The appropriate obstetric and pediatric staff should be notified directly of HBsAg-positive mothers, so the staff may take appropriate precautions to protect themselves and other patients from infectious material, blood, and secretions, and so the neonate may receive therapy without delay after birth.

Recent studies in Taiwan and the United States have confirmed the efficacy of the following regimen (Table 3). Other schedules have also been effective (3, 10, 11). The major consideration for all these regimens is the need to give HBIG as soon as possible after the infant has physiologically stabilized after delivery.

HBIG (0.5 ml) should be administered intramuscularly (IM) after physiologic stabilization of the infant and preferably within 12 hours of birth. HBIG efficacy decreases markedly if treatment is delayed beyond 48 hours. HB vaccine should be administered IM in three doses of 0.5 ml of vaccine (10 μ g) each. The first dose should be given within 7 days of birth and may be given concurrently with HBIG but at a separate site. The second and third doses should be given 1 month and 6 months, respectively, after the first (Table 1). HBsAg testing at 6 months may be done for counseling purposes, since HBsAg-positivity at 6 months indicates a therapeutic failure, and the third vaccine dose need not be given if HBsAg-positivity is found. If a mother's HBsAg-positive status is not discovered until after delivery, prophylaxis should still be administered if a venous (not cord) blood sample from the infant is HBsAg-negative. Testing for HBsAg and anti-HBs is recommended at 12-15 months to monitor the final success or failure of therapy. If HBsAg is found, it is likely the child is a chronic carrier. If HBsAg is not detectible, and anti-HBs is present, the child has been protected. Since maternal antibody to the core antigen (anti-HBc) may persist for more than 1 year, testing for anti-HBc may be difficult to interpret during this period. HB vaccine is an inactivated product, and it is presumed that it will not interfere with other simultaneously administered childhood vaccines (12). HBIG administered at birth should not interfere with oral polio and diphtheria-tetanus-pertussis vaccines administered at about 2 months of age (Table 3).

TABLE 2. Women for whom prenatal HBsAg screening is recommended

-
1. Women of Asian, Pacific Island, or Alaskan Eskimo descent, whether immigrant or U.S.-born.
 2. Women born in Haiti or Sub-Saharan Africa.
- and
- Women with histories of:
3. Acute or chronic liver disease.
 4. Work or treatment in a hemodialysis unit.
 5. Work or residence in an institution for the mentally retarded.
 6. Rejection as a blood donor.
 7. Blood transfusion on repeated occasions.
 8. Frequent occupational exposure to blood in medico-dental settings.
 9. Household contact with an HBV carrier or hemodialysis patient.
 10. Multiple episodes of venereal disease.
 11. Percutaneous use of illicit drugs.
-

ACIP: Hepatitis B

ACUTE EXPOSURE TO BLOOD CONTAINING HBsAg

There are no prospective studies directly testing the efficacy of a combination of HBIG and HB vaccine in preventing clinical HB following percutaneous or mucous-membrane exposure to HBV. However, since health-care workers at risk to such accidents are candidates for HB vaccine and since combined HBIG plus vaccine is more effective than HBIG alone in perinatal exposures, it is reasonable to recommend both HB vaccine and HBIG after such exposure. This combination will provide prolonged immunity to subsequent exposures and may also increase efficacy in preventing HB in such postexposure situations. In addition, because the second dose of HBIG is not considered necessary if the vaccine is used, the cost of combination treatment is usually less than that of two HBIG doses alone. If exposure to blood occurs in situations where the HBsAg status of the blood is unknown, refer to "Immune Globulins for Protection against Viral Hepatitis" (1). If HBsAg testing reveals the source of the blood to be positive, the following treatment schedule should be instituted as soon as possible.

For percutaneous (needlestick), ocular, or mucous-membrane exposure to blood known to contain HBsAg and for human bites from HBsAg carriers that penetrate the skin, a single dose of HBIG (0.06 ml/kg or 5.0 ml for adults) should be given as soon as possible after exposure and within 24 hours if possible. HB vaccine 1 ml (20 µg) should be given IM at a separate site as soon as possible, but within 7 days of exposure, with the second and third doses given 1 month and 6 months, respectively, after the first (Table 1). If HBIG is unavailable, immunoglobulin (IG [formerly ISG or "gamma globulin"]) may be given in an equivalent dosage (0.06 ml/kg or 5.0 ml for adults). If an individual has received at least two doses of HB vaccine before an accidental exposure, no treatment is necessary if serologic tests show adequate levels (> 10 S/N by RIA) of anti-HBs. For persons who choose not to receive HB vaccine, the previously recommended two-dose HBIG regimen may be used (1).

HBIG FOR SEXUAL CONTACTS OF PERSONS WITH ACUTE HBV INFECTION

Sexual contacts of persons with acute HB infection are at increased risk of acquiring HB infection. Two published studies have assessed the value of postexposure prophylaxis for regular sexual contacts of persons with acute HB infection. One showed that HBIG was significantly more effective than IG that contained no measureable anti-HBs in preventing both HB infec-

TABLE 3. Routine pediatric vaccination schedule and HBV prophylaxis for infants of HBsAg-positive mothers

Age (months)	Hepatitis B prevention schedule	HBV marker screening	Routine pediatric schedule
Birth	HBIG*		
1	HB vaccine†		
2	HB vaccine		DPT§, Polio
4			DPT, Polio
6	HB vaccine	HBsAg test¶ **	DPT
12-15		HBsAg** & anti-HBs†† test	
15			MMR§§
18			DPT, Polio

*Hepatitis B immune globulin 0.5 ml IM within 12 hours of birth.

†HB vaccine 0.5 ml IM within 7 days of birth.

§Diphtheria-tetanus-pertussis.

¶Optional. If positive, indicates infection, and a third HB vaccine dose need not be given.

**HBsAg-positive indicates therapeutic failure.

††Anti-HBs-positive indicates therapeutic success.

§§Measles-mumps-rubella.

ACIP: Hepatitis B

tion and clinical illness (5). The second study, however, showed comparable disease rates in persons receiving HBIG and IG containing the increased levels of anti-HBs found in currently available lots (13). Because data are limited, the period after sexual exposure during which HBIG is effective is unknown, but extrapolation from other settings makes it unlikely that this period would exceed 14 days. The value of HB vaccine alone in this setting is unknown. However, since about 90% of persons with acute HB infections become HBsAg-negative within 15 weeks of diagnosis, the potential for repeated exposure is usually self-limited. HB vaccine is not routinely recommended for such exposures.

Prescreening sexual partners for susceptibility before HBIG treatment is recommended if it does not delay HBIG administration beyond 14 days after last exposure. In one study, 27% of regular sexual partners (heterosexual) were positive for HBsAg or anti-HBs at the time they presented for evaluation (5). Among homosexually active males, over 50% have markers indicating prior infection, and 5%-6% are HBsAg positive (2). Testing for anti-HBc is the most efficient prescreening test to use in this population group.

A single dose of HBIG (0.06 ml/kg or 5 ml for adults) is recommended for susceptible individuals who have had sexual contact with an HBsAg-positive persons if HBIG can be given within 14 days of the last sexual contact, and for persons who will continue to have sexual contact with an individual with acute HB before loss of HBsAg in that individual (Table 1). In exposures between heterosexuals, a second HBIG dose should be given if the index patient remains HBsAg-positive 3 months after detection. If the index patient is a known HBV carrier or remains HBsAg-positive for 6 months, HB vaccine should be offered to regular sexual contacts. For exposures among homosexual men, the HB vaccine series should be initiated at the time HBIG is given following a sexual exposure, since HB vaccine is recommended for all susceptible homosexual men (2). Additional doses of HBIG are unnecessary if vaccine is given. Because current lots of IG contain anti-HBs, it remains an important alternative to HBIG when HBIG is unavailable.

References

1. ACIP. Immune globulins for protection against viral hepatitis. *MMWR* 1981;30:423-8, 433-5.
2. ACIP. Inactivated hepatitis B virus vaccine. *MMWR* 1982;31:317-22, 327-8.
3. Beasley RP, Hwang L-Y, Lee GC, et al. Prevention of perinatally transmitted hepatitis B virus infections with hepatitis B immune globulin and hepatitis B vaccine. *Lancet* 1983;ii:1099-102.
4. Seeff LB, Wright EC, Zimmerman HJ, et al. Type B hepatitis after needle-stick exposure: prevention with hepatitis B immune globulin. Final report of the Veterans Administration Cooperative Study. *Ann Intern Med* 1978;88:285-93.
5. Redeker AG, Mosley JW, Gocke DJ, McKee AP, Pollack W. Hepatitis B immune globulin as a prophylactic measure for spouses exposed to acute type B hepatitis. *N Engl J Med* 1975;293:1055-9.
6. Beasley RP, Hwang LY, Stevens CE, et al. Efficacy of hepatitis B immune globulin for prevention of perinatal transmission of the hepatitis B virus carrier state: final report of a randomized double-blind, placebo-controlled trial. *Hepatology* 1983;3:135-41.
7. Szmuness W, Stevens CE, Oleszko WR, Goodman A. Passive-active immunisation against hepatitis B: immunogenicity studies in adult Americans. *Lancet* 1981;i:575-7.
8. Sinatra FR, Shah P, Weissman JY, Thomas DW, Merritt RJ, Tong MJ. Perinatal transmitted acute icteric hepatitis B in infants born to hepatitis B surface antigen-positive and anti-hepatitis B positive carrier mothers. *Pediatrics* 1982;70:557-9.
9. Delaplane D, Yogev R, Crussi F, Schulman ST. Fatal hepatitis B in early infancy: the importance of identifying HBsAg-positive pregnant women and providing immunoprophylaxis to their newborns. *Pediatrics* 1983;72:176-80.
10. Stevens CE, Toy P, Tong MJ, et al. Perinatal hepatitis B virus treatment in the U.S.: prevention by passive-active immunization. Submitted for publication.
11. Wong VCW, Ip MMH, Reesink HW, et al. Prevention of the HBsAg carrier status in newborn infants in mothers who are chronic carriers of HBsAg and HBeAg by administration of hepatitis B vaccine and hepatitis B immune globulin: double-blind randomized placebo-controlled study. *Lancet* 1984;i:921-6.

ACIP: Hepatitis B

12. Chiron JP, Coursaget P, Yvonnet B, et al. Simultaneous administration of hepatitis B and diphtheria/tetanus/polio vaccines [Letter]. *Lancet* 1984; i:623-4.
13. Perrillo, RP, Campbell CR, Strang S, Bodicky CJ, Costigan DJ. Immune globulin and hepatitis B immune globulin: prophylactic measures for intimate contacts exposed to acute type B hepatitis. *Arch Intern Med* 1984; 144:81-5.

Current Trends**National Plague Conference Report**

In response to the increased number of human plague cases in the western United States, an Ad Hoc National Plague Prevention and Control Committee held its first meeting February 22, and February 23, 1984, in Santa Fe, New Mexico. Sponsored by the New Mexico Health and Environment Department and CDC, the committee consisted of representatives from four

*(Continued on page 295)***TABLE I. Summary—cases specified notifiable diseases, United States**

Disease	21st Week Ending			Cumulative, 21st Week Ending		
	May 26, 1984	May 28, 1983	Median 1979-1983	May 26, 1984	May 28, 1983	Median 1979-1983
Acquired Immunodeficiency Syndrome (AIDS)	93	N	N	1,537	N	N
Aseptic meningitis	62	91	77	1,504	1,668	1,447
Encephalitis: Primary (arthropod-borne & unsp.)	12	22	12	327	368	306
Post-infectious	3	5	4	27	43	43
Gonorrhea: Civilian	13,553	15,385	17,373	315,681	357,325	378,065
Military	289	406	361	7,885	9,814	11,001
Hepatitis: Type A	248	361	439	8,575	9,247	10,199
Type B	298	409	357	9,307	8,982	7,799
Non A, Non B	41	61	N	1,412	1,351	N
Unspecified	87	121	188	2,387	2,925	4,071
Legionellosis	11	20	N	210	280	N
Leprosy	3	4	6	85	109	85
Malaria	10	7	20	289	266	343
Measles: Total*	45	70	171	1,327	817	1,680
Indigenous	44	63	N	1,185	675	N
Imported	1	7	N	142	142	N
Meningococcal infections: Total	53	50	57	1,356	1,382	1,384
Civilian	53	48	57	1,352	1,367	1,374
Military	-	2	1	4	15	10
Mumps	25	48	216	1,486	1,766	3,261
Pertussis	13	36	29	807	719	442
Rubella (German measles)	16	15	76	333	488	1,364
Syphilis (Primary & Secondary): Civilian	452	611	578	11,046	13,183	12,133
Military	7	14	9	141	196	150
Toxic Shock syndrome	5	7	N	160	172	N
Tuberculosis	376	476	505	8,366	8,899	10,358
Tularemia	4	7	5	35	71	57
Typhoid fever	3	7	11	124	136	149
Typhus fever, tick-borne (RMSF)	13	13	32	68	101	143
Rabies, animal	63	135	131	1,879	2,696	2,642

TABLE II. Notifiable diseases of low frequency, United States

	Cum. 1984		Cum. 1984
Anthrax	-	Plague	6
Botulism: Foodborne	6	Poliomyelitis: Total	1
Infant (Oreg. 1)	42	Paralytic	1
Other	2	Psittacosis (Mass. 1, N.Y. City 1, Hawaii 1)	30
Brucellosis (Va. 1)	43	Rabies, human	-
Cholera	-	Tetanus (La. 1)	12
Congenital rubella syndrome	3	Trichinosis	19
Diphtheria	-	Typhus fever, flea-borne (endemic, murine)	6
Leptospirosis	8		

*One of the 44 reported cases for this week was imported from a foreign country or can be directly traceable to a known internationally imported case within two generations.

**TABLE III. Cases of specified notifiable diseases, United States, weeks ending
May 26, 1984 and May 28, 1983 (21st Week)**

Reporting Area	AIDS Cum. 1984	Aseptic Menin- gitis 1984	Encephalitis		Gonorrhea (Civilian)		Hepatitis (Viral), by type				Legionel- losis 1984	Leprosy Cum. 1984
			Primary Cum. 1984	Post-in- fectious Cum. 1984	Cum. 1984	Cum. 1983	A 1984	B 1984	NA,NB 1984	Unspeci- fied 1984		
UNITED STATES	1,537	62	327	27	315,681	357,325	248	298	41	87	11	85
NEW ENGLAND	54	1	23	-	9,459	8,952	2	10	-	22	1	5
Maine	-	-	-	-	345	476	-	-	-	-	-	-
N.H.	1	-	4	-	245	247	-	-	-	1	1	-
Vt.	-	-	2	-	157	161	-	-	-	-	-	-
Mass.	31	1	12	-	3,736	4,000	2	10	-	21	-	4
R.I.	4	-	-	-	614	498	-	-	-	-	-	1
Conn.	18	-	5	-	4,362	3,570	-	-	-	-	-	-
MID ATLANTIC	726	14	45	2	44,515	45,700	70	62	5	8	-	10
Upstate N.Y.	58	1	17	2	6,872	6,903	12	10	1	2	-	2
N.Y. City	538	1	-	-	18,998	19,074	39	18	-	3	-	8
N.J.	96	8	15	-	7,201	8,570	14	27	3	3	-	-
Pa.	34	4	13	-	11,444	11,153	5	7	1	-	-	-
E.N. CENTRAL	69	7	70	8	40,179	51,226	16	31	5	4	3	5
Ohio	9	5	28	4	11,619	13,376	6	12	-	2	2	2
Ind.	9	-	12	-	4,776	5,821	-	4	-	-	-	-
Ill.	39	-	10	3	6,268	14,245	1	-	-	-	-	1
Mich.	9	2	17	-	12,514	13,456	9	15	5	2	1	2
Wis.	3	-	3	1	5,002	4,328	-	-	-	-	-	-
W.N. CENTRAL	11	1	10	-	15,256	16,765	3	3	1	1	-	-
Minn.	3	-	3	-	2,154	2,409	-	-	-	-	-	-
Iowa	-	-	4	-	1,772	1,838	2	1	-	1	-	-
Mo.	6	1	1	-	7,204	8,157	1	2	1	-	-	-
N. Dak.	-	-	-	-	160	160	-	-	-	-	-	-
S. Dak.	-	-	-	-	414	467	-	-	-	-	-	-
Nebr.	1	-	1	-	1,104	971	-	-	-	-	-	-
Kans.	1	-	1	-	2,448	2,763	-	-	-	-	-	-
S. ATLANTIC	184	14	67	8	79,651	91,162	23	102	15	9	-	5
Del.	3	-	1	-	1,422	1,673	-	-	1	1	-	-
Md.	16	6	16	-	9,504	11,460	3	11	-	1	-	-
D.C.	22	1	-	-	5,946	6,310	-	-	-	-	-	1
Va.	14	2	16	4	7,704	7,709	2	9	-	1	-	3
W. Va.	3	-	4	-	1,020	956	2	-	-	-	-	-
N.C.	4	-	13	3	13,175	13,329	-	6	2	2	-	-
S.C.	3	-	2	-	7,747	8,660	-	18	1	-	-	-
Ga.	20	1	2	-	13,255	19,741	2	38	1	-	-	-
Fla.	99	4	13	1	19,878	21,324	14	20	10	4	-	1
E.S. CENTRAL	12	12	15	1	27,477	29,867	9	26	2	2	6	-
Ky.	7	-	2	-	3,320	3,598	2	2	-	-	-	-
Tenn.	2	5	2	-	11,261	12,155	3	7	2	2	-	-
Ala.	2	7	10	1	8,879	9,171	3	17	-	-	6	-
Miss.	1	-	1	-	4,017	4,943	1	-	-	-	-	-
W.S. CENTRAL	64	9	21	2	44,632	50,218	68	37	5	38	1	3
Ark.	-	-	-	1	3,959	3,778	3	1	1	3	-	-
La.	8	3	2	-	9,880	8,817	-	2	-	1	-	-
Okla.	4	-	5	1	4,741	5,934	1	6	1	1	1	-
Tex.	52	6	14	-	26,052	31,689	64	28	3	33	-	3
MOUNTAIN	21	3	10	2	10,291	11,030	44	15	5	2	-	7
Mont.	-	1	-	-	467	475	-	-	-	-	-	-
Idaho	-	-	-	-	485	514	9	5	-	-	-	-
Wyo.	1	-	-	-	318	277	-	-	-	-	-	-
Colo.	12	-	6	-	2,957	3,100	21	1	2	1	-	-
N. Mex.	-	-	-	-	1,161	1,353	8	1	2	1	-	-
Ariz.	6	-	1	-	2,765	3,061	-	-	-	-	-	5
Utah	1	2	3	2	534	542	4	1	1	-	-	1
Nev.	1	-	-	-	1,604	1,708	2	7	-	-	-	1
PACIFIC	396	1	66	4	44,221	52,405	13	12	3	1	-	50
Wash.	19	-	2	-	3,111	3,952	1	1	-	-	-	3
Oreag.	3	-	-	-	2,790	2,853	12	7	3	-	-	1
Calif.	370	U	62	4	36,369	43,497	U	U	U	U	U	31
Alaska	-	-	-	-	1,172	1,241	-	2	-	1	-	-
Hawaii	4	1	2	-	779	1,062	-	2	-	-	-	13
Guam	-	U	-	-	78	74	U	U	U	U	U	-
P.R.	24	1	-	1	1,321	1,268	1	8	-	3	-	-
V.I.	-	U	-	-	163	110	U	U	U	U	U	-
Pac. Trust Terr.	-	U	-	-	-	-	U	U	U	U	U	-

N: Not notifiable

U: Unavailable

TABLE III. (Cont.'d). Cases of specified notifiable diseases, United States, weeks ending

May 26, 1984 and May 28, 1983 (21st Week)

Reporting Area	Malaria	Measles (Rubeola)					Meningococcal Infections	Mumps		Pertussis			Rubella		
		Indigenous		Imported*		Total		1984	Cum. 1984	1984	Cum. 1984	Cum. 1983	1984	Cum. 1984	Cum. 1983
		1984	Cum. 1984	1984	Cum. 1984	Cum. 1983									
UNITED STATES	269	44	1,185	1	142	817	1,356	25	1,486	13	807	719	16	333	488
NEW ENGLAND	20	-	84	-	7	6	84	-	45	1	11	24	-	27	7
Maine	-	-	-	-	-	-	1	-	13	-	-	-	-	1	-
N.H.	-	-	26	-	3	2	5	-	5	-	2	4	-	-	2
Vt.	1	-	2	-	2	-	21	-	3	1	8	3	-	-	3
Mass.	12	-	49	-	-	2	29	-	13	-	-	14	-	26	2
R.I.	2	-	-	-	-	-	8	-	4	-	1	3	-	-	-
Conn.	5	-	7	-	2	2	20	-	7	-	-	-	-	-	-
MID ATLANTIC	41	6	64	-	13	26	218	5	195	1	56	210	13	99	37
Upstate N.Y.	12	-	13	-	3	2	75	3	40	1	37	60	12	80	17
N.Y. City	9	6	49	-	3	20	30	-	7	-	2	26	1	17	6
N.J.	13	-	2	-	3	1	49	1	119	-	3	11	-	2	3
Pa.	7	-	-	-	4	3	64	1	29	-	14	113	-	-	11
E.N. CENTRAL	22	19	383	1	63	448	210	9	556	1	222	177	-	46	82
Ohio	6	-	1	1†	2	23	79	-	215	-	37	50	-	2	1
Ind.	-	-	2	-	1	306	28	-	29	-	152	13	-	1	13
Ill.	6	-	114	-	1	114	38	3	132	-	11	88	-	22	35
Mich.	4	19	265	-	54	5	41	6	140	1	12	11	-	14	12
Wis.	6	-	1	-	5	-	24	-	40	-	10	15	-	7	21
W.N. CENTRAL	6	-	1	-	1	1	87	3	75	1	73	47	3	22	29
Minn.	-	-	1	-	1	1	15	-	4	-	10	17	-	2	5
Iowa	1	-	-	-	-	-	16	2	16	-	3	4	-	-	-
Mo.	4	-	-	-	-	-	24	-	6	1	12	8	-	-	-
N. Dak.	-	-	-	-	-	-	1	-	1	-	-	1	-	3	-
S. Dak.	-	-	-	-	-	-	5	-	-	-	1	2	-	-	-
Nebr.	-	-	-	-	-	-	7	1	2	-	2	-	-	-	-
Kans.	1	-	-	-	-	-	19	-	46	-	45	15	3	17	24
S. ATLANTIC	48	1	9	-	12	163	306	2	114	2	54	104	-	17	63
Del.	3	-	-	-	-	-	3	-	2	-	-	-	-	-	-
Md.	12	1	4	-	5	2	24	-	22	-	3	23	-	1	1
D.C.	-	-	-	-	-	-	4	-	-	-	-	-	-	-	-
Va.	9	-	1	-	1	21	34	-	8	-	7	36	-	-	1
W. Va.	-	-	-	-	-	-	4	1	23	-	6	3	-	-	-
N.C.	4	-	-	-	-	-	41	1	14	-	17	5	-	-	6
S.C.	1	-	-	-	-	4	29	-	1	-	1	6	-	-	-
Ga.	4	-	-	-	-	6	65	-	16	-	2	21	-	2	8
Fla.	15	-	4	-	6	130	102	-	28	2	18	10	-	14	47
E.S. CENTRAL	2	-	1	-	2	5	53	-	30	1	5	5	-	5	7
Ky.	-	-	1	-	-	1	4	-	6	-	1	2	-	1	6
Tenn.	-	-	-	-	2	-	19	-	10	-	2	2	-	-	-
Ala.	2	-	-	-	-	4	21	-	4	-	-	-	-	1	1
Miss.	-	-	-	-	-	-	9	-	10	1	2	1	-	3	-
W.S. CENTRAL	23	18	284	-	14	57	160	5	86	5	201	58	-	13	77
Ark.	-	-	-	-	-	10	24	-	4	-	11	4	-	3	-
La.	4	-	-	-	-	12	35	-	-	-	3	2	-	-	9
Okla.	3	-	6	-	-	1	22	N	N	5	178	34	-	-	-
Tex.	16	18	278	-	14	34	79	5	82	-	9	18	-	10	68
MOUNTAIN	11	-	74	-	10	2	48	-	165	-	57	71	-	10	15
Mont.	-	-	-	-	-	-	†	-	3	-	16	1	-	-	2
Idaho	2	-	-	-	-	-	5	-	7	-	1	2	-	1	5
Wyo.	-	-	-	-	-	-	2	-	1	-	3	4	-	2	1
Colo.	1	-	-	-	-	2	18	-	11	-	20	43	-	2	-
N. Mex.	-	-	51	-	8	-	7	N	N	-	5	6	-	-	-
Ariz.	6	-	-	-	-	-	11	-	137	-	8	9	-	-	4
Utah	2	-	23	-	2	-	4	-	5	-	2	6	-	5	2
Nev.	-	-	-	-	-	-	-	-	1	-	2	-	-	-	1
PACIFIC	96	-	285	-	20	109	190	1	220	1	128	23	-	94	171
Wash.	3	-	80	-	4	23	4	-	21	-	15	2	-	1	6
Oreg.	2	-	-	-	-	7	31	N	N	-	9	5	-	-	9
Calif.	88	U	205	U	18	97	129	U	185	U	41	16	U	91	156
Alaska	-	-	-	-	-	-	6	-	4	-	-	-	-	-	-
Hawaii	3	-	-	-	2	1	1	1	10	1	63	-	-	2	-
Guam	-	U	79	U	2	2	1	U	3	U	-	-	U	1	-
P.R.	2	-	-	-	-	73	4	4	72	-	-	7	-	3	3
V.I.	-	U	-	U	-	5	-	U	3	U	-	-	U	-	1
Pac. Trust Terr.	-	U	-	U	-	-	-	U	-	U	-	-	U	-	-

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

N: Not notifiable U: Unavailable †International §Out-of-state

TABLE III. (Cont.'d). Cases of specified notifiable diseases, United States, weeks ending

May 26, 1984 and May 28, 1983 (21st Week)

Reporting Area	Syphilis (Civilian) (Primary & Secondary)		Toxic- shock Syndrome	Tuberculosis		Tula- remia	Typhoid Fever	Typhus Fever (Tick-borne) (RMSF)	Rabies, Animal
	Cum. 1984	Cum. 1983	1984	Cum. 1984	Cum. 1983	Cum. 1984	Cum. 1984	Cum. 1984	Cum. 1984
UNITED STATES	11,046	13,183	5	8,366	8,899	35	124	68 + 14	1,879
NEW ENGLAND	231	292	1	238	241	1	4	-	11
Maine	2	8	-	12	15	-	-	-	7
N.H.	3	10	1	17	20	-	-	-	-
Vt.	1	1	-	3	1	-	-	-	-
Mass.	138	184	-	128	131	1	3	-	3
R.I.	8	10	-	18	18	-	-	-	-
Conn.	79	79	-	60	56	-	1	-	1
MID ATLANTIC	1,549	1,705	-	1,573	1,618	-	18	1	119
Upstate N.Y.	112	148	-	260	269	-	7	1	4
N.Y. City	946	977	-	637	678	-	4	-	-
N.J.	288	345	-	335	332	-	3	-	2
Pa.	203	235	-	341	339	-	4	-	113
E.N. CENTRAL	439	734	1	1,129	1,140	-	18	2	77
Ohio	110	189	1	233	187	-	3	2	6
Ind.	62	66	-	115	90	-	2	-	7
Ill.	60	351	-	456	502	-	8	-	40
Mich.	173	96	-	259	303	-	2	-	5
Wis.	34	32	-	66	58	-	3	-	19
W.N. CENTRAL	180	159	-	225	300	10	5	4	306
Minn.	49	68	-	34	52	-	2	-	29
Iowa	10	4	-	30	31	-	-	-	63
Mo.	92	59	-	107	161	10	2	2	32
N. Dak.	2	1	-	5	2	-	-	-	60
S. Dak.	2	3	-	6	21	-	-	-	72
Nebr.	9	10	-	13	8	-	-	-	19
Kans.	16	14	-	30	25	-	1	2	31
S. ATLANTIC	3,354	3,377	1	1,788	1,747	3	14	21	540
Del.	10	15	-	21	14	-	-	-	-
Md.	224	210	1	231	122	-	-	-	288
D.C.	129	145	-	58	76	-	5	-	-
Va.	180	246	-	164	169	-	4	4	114
W. Va.	8	13	-	64	66	-	-	1	14
N.C.	343	311	-	282	230	1	1	7	8
S.C.	326	216	-	206	158	-	1	7	19
Ga.	486	615	-	230	333	2	-	1	58
Fla.	1,648	1,606	-	532	579	-	3	1	39
E.S. CENTRAL	705	896	-	759	856	-	3	7	103
Ky.	45	53	-	173	220	-	1	2	26
Tenn.	194	254	-	242	259	-	2	3	47
Ala.	253	362	-	247	216	-	-	2	30
Miss.	213	227	-	97	161	-	-	-	-
W.S. CENTRAL	2,655	3,470	2	910	1,030	11	7	30	432
Ark.	81	89	2	101	112	7	-	5	50
La.	509	730	-	123	175	3	1	1	19
Okla.	76	101	-	97	125	1	1	13	52
Tex.	1,989	2,550	-	589	618	-	5	11	311
MOUNTAIN	273	307	-	196	249	6	5	2	68
Mont.	1	4	-	10	22	-	1	2	43
Idaho	11	6	-	9	14	2	-	-	-
Wyo.	2	6	-	-	4	-	-	-	-
Colo.	60	66	-	20	22	1	1	-	-
N. Mex.	39	105	-	45	47	-	2	-	9
Ariz.	110	68	-	83	110	1	-	-	16
Utah	8	9	-	15	18	2	-	-	-
Nev.	42	43	-	14	12	-	1	-	-
PACIFIC	1,660	2,243	-	1,548	1,718	4	50	1	223
Wash.	48	71	-	77	90	-	1	-	1
Oreg.	50	39	-	64	77	2	1	1	-
Calif.	1,527	2,097	U	1,298	1,412	2	44	-	216
Alaska	3	7	-	22	25	-	1	-	6
Hawaii	32	29	-	87	114	-	3	-	-
Guam	-	-	U	5	3	-	-	-	-
P.R.	333	377	-	167	212	-	3	-	25
V.I.	6	8	U	2	1	-	-	-	-
Pac. Trust Terr.	-	-	U	-	-	-	-	-	-

U. Unavailable

TABLE IV. Deaths in 121 U.S. cities,* week ending
May 25, 1984 (21st Week Ending)

Reporting Area	All Causes, By Age (Years)						P&I** Total	Reporting Area	All Causes, By Age (Years)						P&I** Total
	All Ages	≥65	45-64	25-44	1-24	<1			All Ages	≥65	45-64	25-44	1-24	<1	
NEW ENGLAND	668	451	144	34	15	23	47	S. ATLANTIC	1,096	676	267	86	31	36	46
Boston, Mass.	205	122	54	10	6	12	15	Atlanta, Ga.	151	77	46	15	5	8	5
Bridgport, Conn.	41	27	10	3	-	1	2	Baltimore, Md.	149	99	32	9	4	5	2
Cambridge, Mass.	21	15	5	1	-	1	2	Charlotte, N.C.	72	52	15	4	1	-	5
Fall River, Mass.	29	20	6	1	-	1	1	Jacksonville, Fla.	83	49	25	5	2	2	9
Hartford, Conn.	83	59	18	4	-	2	5	Miami, Fla.	84	53	18	11	4	1	-
Lowell, Mass.	25	18	5	1	-	1	-	Norfolk, Va.	49	31	7	3	4	4	1
Lynn, Mass.	20	11	8	1	-	-	1	Richmond, Va.	71	38	23	3	2	5	3
New Bedford, Mass.	21	18	3	-	-	-	-	Savannah, Ga.	36	24	7	1	3	1	2
New Haven, Conn.	43	25	7	5	2	4	1	St. Petersburg, Fla.	103	81	18	3	-	1	5
Providence, R.I.	53	41	8	2	-	2	5	Tampa, Fla.	64	40	14	7	1	2	5
Somerville, Mass.	8	5	2	1	-	-	-	Washington, D.C.	183	102	46	20	8	7	9
Springfield, Mass.	44	34	7	-	2	1	6	Wilmington, Del.	51	30	16	5	-	-	-
Waterbury, Conn.	25	19	3	1	2	-	2	E.S. CENTRAL	786	475	206	54	29	22	39
Worcester, Mass.	50	37	8	4	1	-	6	Birmingham, Ala.	147	91	29	12	6	9	5
MID. ATLANTIC	2,496	1,683	513	198	51	51	117	Chattanooga, Tenn.	60	36	17	4	1	2	4
Albany, N.Y.	54	34	10	1	3	6	1	Knoxville, Tenn.	80	39	27	7	2	5	5
Allentown, Pa.	20	16	4	-	-	-	-	Louisville, Ky.	106	66	24	8	7	1	4
Buffalo, N.Y.	132	94	32	2	1	3	8	Memphis, Tenn.	163	105	38	15	4	1	10
Camden, N.J.	45	28	13	1	-	3	2	Mobile, Ala.	69	41	21	4	2	1	4
Elizabeth, N.J.	22	11	6	5	-	-	5	Montgomery, Ala.	46	22	20	1	3	-	-
Erie, Pa.†	54	45	8	1	-	-	1	Nashville, Tenn.	115	75	30	3	4	3	-
Jersey City, N.J.	56	40	11	3	1	1	-	W.S. CENTRAL	1,209	719	287	113	50	40	60
N.Y. City, N.Y.	1,354	913	266	125	24	26	56	Austin, Tex.	55	35	14	4	1	1	9
Newark, N.J.	49	28	8	2	3	4	4	Baton Rouge, La.	16	12	2	1	1	-	1
Paterson, N.J.	36	28	4	2	2	-	5	Corpus Christi, Tex.	48	36	9	2	-	1	-
Philadelphia, Pa.†	192	91	56	32	8	5	8	Dallas, Tex.	184	98	46	21	13	6	2
Pittsburgh, Pa.†	59	38	17	2	2	-	4	El Paso, Tex.	59	39	8	8	2	2	3
Reading, Pa.	27	23	3	1	-	-	-	Fort Worth, Tex.	90	59	22	3	2	4	4
Rochester, N.Y.	140	110	24	3	3	-	16	Houston, Tex.	295	153	79	37	19	7	13
Schenectady, N.Y.	31	24	3	4	-	-	1	Little Rock, Ark.	63	32	21	4	1	5	8
Scranton, Pa.†	40	28	8	2	2	-	1	New Orleans, La.	117	66	35	12	2	2	1
Syracuse, N.Y.	98	70	20	4	2	2	3	San Antonio, Tex.	158	101	29	12	5	11	9
Trenton, N.J.	29	18	8	2	1	-	-	Shreveport, La.	34	29	4	1	-	-	-
Utica, N.Y.	28	22	6	-	-	-	1	Tulsa, Okla.	90	59	18	8	4	1	10
Yonkers, N.Y.	30	22	6	-	-	2	1	MOUNTAIN	712	444	159	51	30	28	31
E.N. CENTRAL	2,255	1,594	413	110	73	55	86	Albuquerque, N.Mex.	85	51	18	9	4	3	4
Akron, Ohio	72	53	11	4	2	2	-	Colorado Springs, Colo.	32	21	6	1	2	2	1
Canton, Ohio	36	27	9	-	-	-	4	Denver, Colo.	121	78	28	6	3	6	3
Chicago, Ill. §	508	448	8	9	16	17	14	Las Vegas, Nev.	76	39	23	9	2	3	8
Cincinnati, Ohio	105	70	23	4	6	2	11	Ogden, Utah	38	29	6	2	-	1	3
Cleveland, Ohio	171	95	45	16	7	8	4	Phoenix, Ariz.	147	94	33	13	4	3	1
Columbus, Ohio	128	61	43	8	4	2	1	Pueblo, Colo.	41	29	7	1	2	2	5
Dayton, Ohio	107	63	34	5	3	2	4	Salt Lake City, Utah	62	35	9	6	6	6	-
Detroit, Mich.	253	167	52	24	8	2	9	Tucson, Ariz.	110	68	29	4	7	2	6
Evansville, Ind.	38	32	6	-	-	-	1	PACIFIC	1,950	1,454	277	105	57	42	88
Fort Wayne, Ind.	52	39	10	3	-	-	4	Berkeley, Calif.	24	17	5	-	-	2	1
Gary, Ind.	20	5	12	2	1	-	-	Fresno, Calif.	93	58	20	7	7	1	8
Grand Rapids, Mich.	64	45	15	3	-	-	1	Glendale, Calif. §	27	27	10	-	-	-	-
Indianapolis, Ind.	169	108	36	8	8	9	2	Honolulu, Hawaii	47	34	10	3	-	-	2
Madison, Wis.	44	25	7	4	1	1	6	Long Beach, Calif.	76	49	18	4	3	2	1
Meiwa, Ill.	55	36	12	7	5	3	4	Los Angeles, Calif. §	590	535	3	9	17	12	-
Rockford, Ill.	57	42	12	3	3	1	5	Oakland, Calif.	71	51	8	9	2	-	2
South Bend, Ind.	62	44	15	-	2	1	3	Pasadena, Calif.	35	27	6	2	-	-	3
Toledo, Ohio	91	63	21	3	2	2	5	Portland, Ore.	132	93	24	9	3	3	5
Youngstown, Ohio	72	52	15	3	1	1	-	Sacramento, Calif.	134	80	38	9	5	2	19
W.N. CENTRAL	735	523	131	30	23	28	38	San Diego, Calif.	146	89	32	14	5	6	16
Des Moines, Iowa	67	45	12	3	5	2	6	San Francisco, Calif.	174	109	42	16	3	4	5
Duluth, Minn.	21	16	2	3	-	-	-	San Jose, Calif.	142	94	29	9	7	3	13
Kansas City, Kans.	30	21	7	-	2	-	1	Seattle, Wash.	153	114	21	11	2	5	4
Kansas City, Mo.	122	80	27	6	2	7	8	Spokane, Wash.	56	40	10	3	2	1	3
Lincoln, Nebr.	36	27	7	1	1	-	1	Tacoma, Wash.	50	37	11	-	1	1	6
Minneapolis, Minn.	74	49	10	5	2	8	4	TOTAL	11,907 ^{††}	8,019	2,397	781	359	325	552
Omaha, Nebr.	90	60	20	3	3	4	5								
St. Louis, Mo.	160	122	21	7	5	5	6								
St. Paul, Minn.	76	61	12	2	1	-	1								
Wichita, Kans.	59	42	13	-	2	2	6								

* 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

† Because 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.

†† Total includes unknown ages.

§ Data not available. Figures are estimates based on average of past 4 weeks.

Plague — Continued

state health departments, the Indian Health Service, CDC, Pan American Health Organization/World Health Organization, and one city health department. The agenda for the conference was: (1) develop standards for epidemiologic and environmental investigation of human plague cases in the United States; (2) assess and improve current surveillance and control strategies; and (3) establish short- and long-term research objectives aimed at preventing human plague cases.

The committee perceived a need to standardize investigation and reporting of all U.S. human plague cases. Subcommittees were appointed to develop surveillance forms for cooperative reporting of plague cases and field investigations by state and local health departments. Data submitted on these forms will be analyzed by CDC and used for ongoing plague research.

The committee concluded that current surveillance and control strategies appeared to be effective when systematically and intensively applied and that more effective and/or less costly surveillance and control strategies needed to be developed and defined.

The committee listed five priority areas for plague research directed toward preventing human plague cases and deaths. They are: (1) studies on the basic biology of flea vectors and plague ecology, with emphasis on interactions among vector, pathogen, and weather that may prolong survival of infective fleas and increase human transmission potential; (2) documentation of the role of pets in transporting or transmitting plague to humans; (3) studies to delineate plague risk factors among humans; (4) evaluation and improvement of existing plague control strategies directed at preventing human cases; and (5) development of improved and simplified laboratory diagnostic tests.

The committee recognized that human plague, despite its severe morbidity and moderate-to-high mortality, remains a disease of low, if increasing, incidence and that cost-benefit ratios of plague programs should be carefully considered. Although plague in nature is widespread throughout the western United States, definite geographic areas are identified as high-risk areas to humans. The committee views selective and increased commitment of both federal and state funds for research and the development of sound and cost-effective surveillance and control methods in recognized human high-risk areas as an important first step toward reducing human plague morbidity and mortality.

Copies of the committee report may be obtained from Allan M. Barnes, Ph.D., Plague Branch, Division of Vector-Borne Viral Diseases, Center for Infectious Diseases, CDC, P.O. Box 2087, Fort Collins, Colorado 80522.

Reported by J Doll, PhD, Arizona Dept of Health Svcs; SB Werner, MD, B Nelson, PhD, California Dept of Health Svcs; J Emerson, DVM, Colorado Dept of Health; J Mann, MD, O Rollag, DVM, H Hull, MD, J Thompson, N Weber, New Mexico State Health and Environment Dept, T Brown, Environmental Improvement Div, C Montman, Albuquerque Environmental Health, G Heck, B Tempest, R Leach, R Grinnell, Indian Health Svc, New Mexico; J Rust, PhD, Pan American Health Organization/World Health Organization; Plague Br, Div of Vector-Borne Viral Diseases, Center for Infectious Diseases, CDC.

Declining Rates of Rectal and Pharyngeal Gonorrhea Among Males—New York City

The rates of rectal and pharyngeal gonorrhea for New York City males aged 15-44 years* has declined from 129 per 100,000 males in that age group in 1980 to 74/100,000 in 1983—the lowest level in the past 7 years. This decrease is most evident in the area with the highest rates—Manhattan—where reported rectal and pharyngeal gonorrhea rates declined

*1980 Census data.

Rectal and Pharyngeal Gonorrhea — Continued

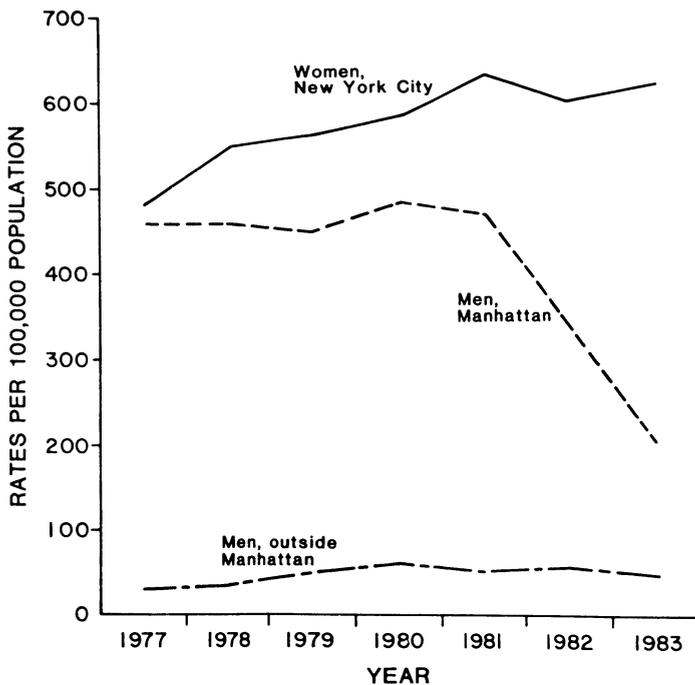
from 485/100,000 in 1980 to 201/100,000 in 1983—a 59% decrease (Figure 1). In other areas of New York City, the rates of rectal and pharyngeal gonorrhea have declined slightly since 1980, but the initial rates outside Manhattan were much lower. Gonorrhea rates for females 15-44 years old have risen over the same period from 587/100,000 females in that age group to 624/100,000 in 1983 (Figure 1).

The majority of New York City rectal and pharyngeal gonorrhea was reported from one New York City Department of Health sexually transmitted disease (STD) clinic in Manhattan, whose patients are primarily homosexual males. At this clinic, culture testing for pharyngeal and rectal gonorrhea is provided to all males identified as being at risk for contracting gonorrhea due to same-sex contact. Based on analyses of second- and fourth-quarter data from each year, the percentage of positive rectal cultures declined from 30.3 in 1980 to 16.5 in 1983, and the percentage of positive pharyngeal cultures declined from 6.8% in 1980 to 2.4% in 1983 (Table 4). First clinic visits by males decreased by 4.3% from 18,434 in fiscal year 1980 to 17,635 in fiscal year 1983.

Reported by S Schultz, MD, S Friedman, MD, A Kristal, DrPH, DJ Sencer, MD, New York City Dept of Health; Div of Sexually Transmitted Diseases, Center for Prevention Svcs, AIDS Activity, Center for Infectious Diseases, CDC.

Editorial Note: Since 1980, reported pharyngeal and rectal gonorrhea rates among New York City males 15-44 years old have shown consistent annual decreases, while the reported rates of gonorrhea for females in the same age group have increased during the same period.

FIGURE 1. Reported rates of rectal and pharyngeal gonorrhea among males 15-44 years old and rates (all sites) of gonorrhea among women 15-44 years old — New York City, 1977-1983



Rectal and Pharyngeal Gonorrhea — Continued

In Manhattan, the greatest decreases in male pharyngeal and rectal gonorrhea rates occurred in 1982 and 1983.

The percent decreases in infection were substantially greater than either the percent decreases in clinic attendance or total cultures taken. Hence, it is unlikely that changes in testing or clinic attendance account for a large portion of the declines. A similar decrease in gonorrhea incidence has been reported among homosexual males attending a public clinic in Denver, Colorado (1).

The major gonorrhea decreases in 1982 and 1983 coincide with the period of heightened awareness and concern about the incidence of acquired immunodeficiency syndrome (AIDS) among homosexual males. U.S. Public Health Service recommendations stress the importance of reducing the numbers of sexual partners for preventing AIDS among homosexual males (2). Similar recommendations have been developed and widely distributed by the American Association of Physicians for Human Rights and many local groups concerned with the health of homosexual males. Recently, a reduction of the number of sexual partners among homosexual males has been documented in Madison, Wisconsin (3). The substantial and persistent declines in gonorrhea among homosexual males in New York City suggest that prevention efforts have succeeded in reducing the incidence of this short-incubation-period sexually transmitted infection. Further sustained efforts should help in reducing the incidence of AIDS among homosexual males.

References

1. Judson FN. Fear of AIDS and gonorrhea rates in homosexual men [Letter]. *Lancet* 1983;II:159-60.
2. CDC. Prevention of acquired immune deficiency syndrome (AIDS): report of inter-agency recommendations. *MMWR* 1983;32:101-3.
3. Golubjatnikov R, Pfister J, Tillotson T. Homosexual promiscuity and the fear of AIDS [Letter]. *Lancet* 1983;II:681.

TABLE 4. Results of rectal and pharyngeal cultures on males at a sexually transmitted diseases clinic — New York City, combined second and fourth quarters, 1979-1983

Year	Rectal			Pharyngeal		
	Total no. of cultures	No. of positive cultures	Percent positive	Total no. of cultures	No. of positive cultures	Percent positive
1979	3,850	940	24.4	3,384	184	5.4
1980	3,388	1,025	30.3	2,755	188	6.8
1981	4,078	1,062	26.0	3,717	163	4.4
1982	4,324	930	21.5	4,361	217	5.0
1983	3,202	529	16.5	3,359	81	2.4

*Epidemiologic Notes and Reports***Perinatal and Maternal Mortality in a Religious Group — Indiana**

In May 1983, the Indiana State Board of Health received reports of apparently excessive perinatal and maternal mortality among members of a religious group in the northeastern part of the state. Approximately 40 deaths among Indiana residents were reported to have occurred over the past 8 years in this group—a fundamentalist church that avoids all medical attention in favor of spiritual healing. Pregnant members receive no prenatal care; they labor and deliver at home without medical assistance. To assess the effect of this group's practices on

Perinatal and Maternal Mortality – Continued

reproductive outcomes, the Indiana State Board of Health, in cooperation with CDC, investigated perinatal and maternal mortality rates for church members from 1975 to 1982 (7).^{*} Because most members in Indiana reside in Elkhart or Kosciusko Counties, the investigation focused in these counties.

Each of the 40 deaths among Indiana members that occurred from 1975 through 1982 was confirmed, including 21 perinatal deaths, seven infant and child deaths, six maternal deaths, and six nonmaternal adult deaths. Of these, 17 perinatal and three maternal deaths occurred among residents of Elkhart or Kosciusko Counties. Reported fetal deaths accounted for 11 of these 17 perinatal deaths.

To estimate 1979-1982 live births in Elkhart and Kosciusko Counties, the Board of Health screened 1975-1982 birth records in these counties for births that were not attended by physicians and that occurred outside hospitals to women who had not received prenatal care. Using these criteria, 344 live births were identified for calculating the perinatal and maternal mortality rates among church members.

The estimated 1975-1982 perinatal mortality rate for members residing in Elkhart or Kosciusko Counties was 48/1,000 live births and fetal deaths, compared with 18/1,000 for all other Indiana residents. The estimated 1975-1982 maternal mortality rate for members residing in Elkhart or Kosciusko Counties was 872/100,000 live births, compared with 9/100,000 for other Indiana residents.

Reported by C Spence, MD, TS Danielson, MD, Div of Maternal and Child Health, Indiana State Board of Health; Pregnancy Epidemiology Br, Div of Reproductive Health, Center for Health Promotion and Education, CDC.

Editorial Note: This report highlights the increased risk of perinatal and maternal mortality for women receiving no obstetric care. Because the denominators may have included non-member births, the rates may underestimate perinatal and maternal mortality among religious-group members. Ongoing surveillance and investigation of maternal deaths by the Indiana Maternal Mortality Committee (2) improved the likelihood that all Indiana pregnancy-associated deaths during the years examined in this investigation were identified.

Other religious groups may also encourage members to avoid medical care. By advising lay people of the risks associated with lack of obstetric care, public health officials and health-care providers may increase the safety of childbirth in their communities.

References

1. Spence C, Danielson TS, Kaunitz AM. The Faith Assembly: a study of perinatal and maternal mortality. *Indiana Medicine* 1984 (March);180-3.
2. Ragan WD. Maternal mortality in Indiana: a report of maternal deaths in 1979. *J Indiana State Med Assoc* 1981;74:565.

^{*}The perinatal mortality rate is defined as the number of fetal deaths (≥ 20 weeks' gestation) plus the number of neonatal deaths (infants who die ≤ 28 days after birth) per 1,000 live births plus fetal deaths. The maternal mortality rate is defined as the number of maternal deaths (pregnancy-related deaths that occur during or up to 1 year after termination of pregnancy) per 100,000 live births.

Lymphocytic Choriomeningitis — Georgia

On December 31, 1983, a 58-year-old woman from Winder, Georgia, was admitted to a hospital in Cincinnati, Ohio, with a 1-week history of malaise, diffuse myalgias, fever, and chills; a 1-day history of vomiting, severe headache, stiff neck, and photophobia; and a his-

Lymphocytic Choriomeningitis — Continued

tory of exposure to mice in her home. Examination revealed a lethargic but arousable patient with a temperature of 38.5 C (101.3 F), and nuchal rigidity. The cerebrospinal fluid (CSF) contained 930 white blood cells, with 69% lymphocytes. Lymphocytic choriomeningitis (LCM) virus was isolated from the CSF. The patient's recovery was uncomplicated, and she was discharged January 7, 1984.

On January 13, the Georgia Department of Human Resources and CDC visited the patient's home. Blood specimens were obtained from two other household residents, and rodent traps were set inside and outside the house. By the following morning, seven grey house mice (*Mus musculus*) had been caught, six within the house and one in an adjacent wooded area.

The two household residents had no detectable LCM antibodies by indirect fluorescent antibody (IFA) testing. However, all six mice trapped in the house had evidence of LCM virus infection; four mice had IFA antibodies, and two were viremic and had virus antigen in the liver, as detected by direct FA staining of liver-touch impressions. On February 23, the patient's neighborhood was investigated. Blood specimens were obtained from 13 persons in six nearby residences; traps were set in five of those residences. Five mice were trapped in two of the houses. None of the specimens from humans or mice showed evidence of LCM virus.

Reported by W Bullock, MD, M Meier, MD, University of Cincinnati Medical Center, Ohio; B Willingham, Barrow County Health Dept, Winder, RK Sikes, DVM, State Epidemiologist, Georgia Dept of Human Resources; Special Pathogens Br, Viral and Rickettsial Zoonoses Br, Epidemiology Office, Div of Viral Diseases, Center for Infectious Diseases, CDC.

Editorial Note: LCM virus is a mouse-borne arenavirus that can cause three different forms of human illness: aseptic meningitis, encephalitis, and an influenza-like illness. Inapparent infections may also occur (1). Diagnosis requires the isolation of LCM virus from CSF or blood using an appropriate cell-culture system, or demonstration of either a fourfold rise in antibody titer between acute- and convalescent-phase serum specimens or an IFA titer 1:128 or higher in a single specimen in which immunoglobulin M (IgM) anti-LCM antibody is present.

This investigation points out that LCM should be considered in sporadic cases of aseptic meningitis, especially in the winter. Few estimates exist of the exact incidence of LCM; however, in one investigation, LCM was responsible for 8% of 1,568 cases of clinically diagnosed aseptic meningitis in the United States (2). The present report emphasizes the previously described association of sporadic cases with infected mice (3), and the previously described observation that the virus can remain localized to a single household.

In contrast, outbreaks of LCM in the general population have generally been traced to contact with Syrian golden hamsters (*Mesocricetus auratus*). In the United States (4) and Germany (1), these have resulted from exposure in the home to pet hamsters obtained from breeders with infected stock. Outbreaks have also occurred among laboratory workers following the introduction of LCM virus into hamster colonies through infected cell lines (5).

References

1. Ackermann WS, Blumenthal W, Helm EB, Keller K, Baldus O. Syrische Goldhamster als Ubertrager von Lymphozytärer Choriomeningitis. *Deutsch Med Woch* 1972;45:1725-31.
2. Meyer HM Jr, Johnson RT, Crawford IP, Dascomb HE, Rogers NG. Central nervous system syndromes of "viral" etiology. *Am J Med* 1960;29:334-47.
3. Farmer TW, Janeway CA. Infections with virus of lymphocytic choriomeningitis. *Medicine* 1942;21:1-63.
4. Biggar RJ, Woodall JP, Walter PD, Haughie GE. Lymphocytic choriomeningitis outbreak associated with pet hamsters: fifty-seven cases from New York State. *JAMA* 1975;232:494-500.
5. Lewis AM Jr, Rowe WP, Turner HC, Huebner RJ. Lymphocytic-choriomeningitis virus in hamster tumor: spread to hamsters and humans. *Science* 1965;150:363-4.

The *Morbidity and Mortality Weekly Report* is prepared by the Centers for Disease Control, Atlanta, Georgia, and available on a paid subscription basis from the Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402, (202) 783-3238.

The data in this report are provisional, based on weekly reports to CDC by state health departments. The reporting week concludes at close of business on Friday; compiled data on a national basis are officially released to the public on the succeeding Friday.

The editor welcomes accounts of interesting cases, outbreaks, environmental hazards, or other public health problems of current interest to health officials. Such reports and any other matters pertaining to editorial or other textual considerations should be addressed to: ATTN: Editor, *Morbidity and Mortality Weekly Report*, Centers for Disease Control, Atlanta, Georgia 30333.

Director, Centers for Disease Control
James O. Mason, M.D., Dr.P.H.
Director, Epidemiology Program Office
Carl W. Tyler, Jr., M.D.

Editor
Michael B. Gregg, M.D.
Assistant Editor
Karen L. Foster, M.A.

DEPARTMENT OF
HEALTH & HUMAN SERVICES
Public Health Service
Centers for Disease Control
Atlanta GA 30333

Official Business
Penalty for Private Use \$300



Postage and Fees Paid
U.S. Dept. of H.H.S.
HHS 396

S *HCRH NEWV75 8129
OR VERNE F NEWHOUSE
VIROLOGY DIVISION
CID
7-814

X