CENTERS FOR DISEASE CONTROL



MORBIDITY AND MORTALITY WEEKLY REPORT

- 269 National Birthweight-Specific Infant Mortality Surveillance: Preliminary Analysis – United States, 1980
- 274 Ammonia Contamination in a Milk Processing Plant — Wisconsin
- 275 Rubella Vaccination during Pregnancy — United States, 1971-1985
- **284** HTLV-III/LAV Antibody Testing at Alternate Sites

Current Trends

National Birthweight-Specific Infant Mortality Surveillance: Preliminary Analysis — United States, 1980

Low birthweight (LBW), the greatest single problem associated with infant mortality (1), is related to many infant and maternal characteristics (2), including several that can be examined using vital records. Such records include infant's race, sex, gestational age at birth, birth order and plurality, and mother's age, educational attainment, and prenatal-care history. These maternal and infant characteristics are also related to neonatal^{*} and postneonatal[†] mortality in varying degrees, in part because of their association with LBW. To examine these associations in the population, it is necessary to link individual birth and infant death certificates. While such linkage has been conducted in various states, the last national birth-death linkage was performed for infants born during 1960 (*3*). CDC, in collaboration with all states, the National Institute of Child Health and Human Development, the National Center for Health Statistics (NCHS), and the Health Resources and Services Administration, compiled birth-death linkage bas performed for live births occurring in 1980 based on linkages performed within each state, New York City, the District of Columbia, and Puerto Rico.

Preliminary analysis from this National Infant Mortality Surveillance (NIMS) shows that neonatal mortality (NNM) ranged from 647.6/1,000 live-born infants 500-999 g[§] to 1.4/1,000 live-born infants 3,500-3,999 g (Figure 1). However, at 3,500 g or greater, NNM increased with increasing birthweight. Compared with black infants, white infants under 3,000 g had higher NNM, but whites 3,000 g or greater had lower NNM than blacks with comparable birthweight.

Postneonatal mortality (PNM) among neonatal survivors ranged from 135.2/1,000 survivors 500-999 g at birth to 1.9/1,000 survivors 4,000-4,499 g at birth (Figure 2). PNM rose with birthweight 4,500 g or greater for both blacks and whites. Black neonatal survivors experienced higher PNM in all birthweight categories.

There was a 93.9-fold relative risk (RR) of infant mortality for singleton infants under 1,500 g at birth (469.4 deaths/1,000 live births), compared with infants 2,500 g or greater (5.0 deaths/1,000 live births) (Table 1). When smaller white singleton infants were compared with larger white singleton infants, the RR was somewhat higher (108.0) than the RR (93.9) for those of all races. When smaller black singleton infants were compared with larger, the RR was lower (61.6), primarily because infant mortality among black infants did not improve as

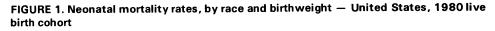
^{*}Neonatal death: death occurring from 0 days to 27 days.

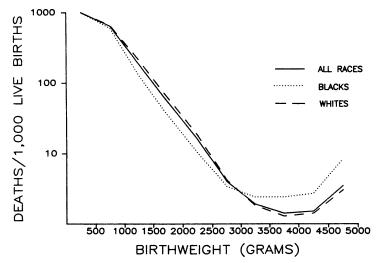
[†]Postneonatal death: death occurring from 28 days to 1 year.

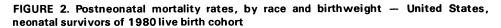
[§]By definition, all live-born infants under 500 g died during the neonatal period.

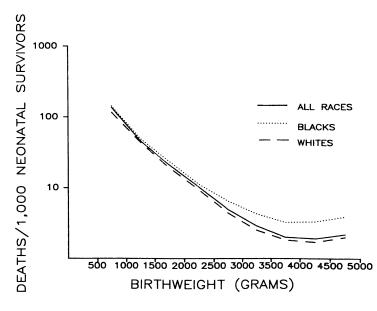
Infant Mortality Surveillance - Continued

greatly with increasing birthweight. Moreover, black infants under 2,500 g had a lower infant mortality than white infants of comparable birthweight (under 1,500 g and 1,500-2,499 g), while black infants 2,500 g or greater had a greater risk of infant mortality. The overall two-fold higher risk of infant mortality for blacks than for whites reflects their excess mortality









MMWR

Infant Mortality Surveillance – Continued

among the majority of births that occur in the larger-weight categories as well as the higher risk of LBW among black infants. For singleton black infants, 2.1% were under 1,500 g, and 9.2% weighed 1,500-2,499 g; percentages for singleton white infants were 0.7% and 4.2%, respectively.

For multiple-born infants (e.g., twins, triplets), birthweight-specific infant mortality also declined with increasing birthweight (Table 1); black multiple-born infants experienced lower infant mortality than whites, only at birthweight under 1,500 g. Within races, multiple-born infants under 2,500 g had lower infant mortality than singleton infants of comparable birthweight (under 1,500 g and 1,500-2,499 g), but multiple-born infants 2,500 g or greater had higher infant mortality. Among black multiple-born infants, 16.3% were under 1,500 g, and 45.5% were 1,500-2,499 g; among whites, 9.4% were under 1,500 g, and 39.8% weighed 1,500-2,499 g. Overall, multiple-born infants experienced higher infant mortality than singleton infants because of a greater risk of both LBW and death among heavier infants.

Infant mortality was higher among singleton males than among singleton females (Table 2). Among white singletons, infant mortality improved with longer gestations to 42 weeks and was lowest for second-born infants; for blacks, infant mortality was lowest for third-born infants and decreased with increasing gestation to 40 weeks. Infant mortality was lowest for black infants born to mothers 25-34 years of age; for white infants, infant mortality was lowest among those born to mothers 30-34 years of age. For all races combined, infant mortality was lowest among infants born to college graduates and women who received prenatal care beginning in the first trimester.

Reported by all state health depts; the health depts of New York City, the District of Columbia, and Puerto Rico; Demographic and Behavioral Sciences Br, Center for Population Research, National Institute of Child Health and Human Development, National Institutes of Health; Div of Maternal and Child Health, Health Resources and Svcs Administration; National Center for Health Statistics; Pregnancy Epidemiology Br, Research and Statistics Br, Div of Reproductive Health, Center for Health Promotion and Education, CDC.

Editorial Note: The reduction of infant mortality is a major health objective in the United States (1). Success in improving survival of LBW infants since 1960 has not been paralleled by a decline in the incidence of LBW (2,4-8). Lower infant mortality rates in other industrialized nations point to further improvements that can be achieved in the United States (8-10), especially for black infants who continue to suffer neonatal and postneonatal mortality rates approximately twice as high as those for white infants (11, 12).

	< 1,	500	1,500	2,499	≥ 2,	500	Tota	al *
Plurality/race	No.	Rate	No.	Rate	No.	Rate	No.	Rate
Singletons								
Blacks	5,152	443.2	1,512	29.5	3,609	7.2	10,602	18.4
Whites	9,568	475.3	4,411	38.5	11,764	4.4	26,478	9.3
All [†]	15,302	469.4	6,207	36.3	16,133	5.0	38,887	11.0
Multiple-borns								
Blacks	937	417.1	144	23.2	63	12.0	1,185	83.2
Whites	2,261	469.1	405	20.0	195	7.5	2,997	55.8
All [†]	3,312	456.7	571	21.0	264	8.2	4,330	61.9

TABLE 1. Infant deaths and infant mortality rate per 1,000 live births, by birthweight, race, and plurality, for infants born during 1980 — U.S. National Infant Mortality Surveillance

*Total number includes infant deaths of unknown birthweight; for calculation of rates, numbers of unknown birthweights were redistributed among known birthweights.

[†]All races, including unknown race and infants of other races.

Infant Mortality Surveillance - Continued

Analysis of infant mortality by birthweight provides a powerful tool at both national and state levels for identifying problems in maternal and infant care and for developing intervention plans. The NIMS project represents an intermediate step towards the routine annual collection of linked birth and death information as proposed by NCHS. This project required major efforts from all states and other vital registration reporting areas to provide the first national data on birthweight-specific infant mortality since 1960.

Characteristic	Blacks	Whites	All races
Infant's		· · · ·	
Sex			
Male	20.1	10.4	12.1
Female	16.8	8.2	9.7
Gestation (wks.)			
17-27	456.1	529.0	509.3
28-31	100.0	136.7	125.0
32-35	24.2	29.4	28.3
36	12.1	14.0	13.9
37	10.9	9.3	9.9
38-39	7.2	4.8	5.3
40	8.4	4.5	5.1
41	7.9	4.1	4.7
42-45	10.0	5.6	6.3
Live-birth order			
First	18.5	9.5	10.9
Second	17.7	8.7	10.2
Third	16.6	9.2	10.7
Fourth	17.5	9.7	11.7
Fifth	20.9	11.5	14.0
Sixth or higher	20.0	11.8	14.4
Mother's			
Age (yrs.)			
10-14	35.5	25.0	31.5
15-19	20.8	13.6	15.8
20-24	18.1	9.4	11.1
25-29	16.5	8.0	9.2
30-34	16.5	7.8	9.0
35-39	18.7	9.3	10.8
40-44	19.1	14.1	15.1
45-49	35.0	21.1	22.6
Education (yrs.)			22.0
0-8	25.3	15.1	17.2
9-11	22.0	13.7	16.3
12	17.5	8.9	10.5
13-15	15.4	7.4	8.8
≥ 16	13.2	6.7	0.0 7.3
Month prenatal		0.1	7.5
care began			
1-3	16.7	8.5	9.7
4-6	17.1	11.0	12.6
7-9	16.2	10.8	12.6
None	67.1	38.3	48.7

TABLE 2. Infant mortality rate* per 1,000 live births, by race and selected characteristics, for singleton infants born during 1980 — U.S. National Infant Mortality Surveillance

*Rates were based on birthweights that included redistributed unknown birthweights.

MMWR

Infant Mortality Surveillance – Continued

With the exception of infant's sex and race, information on the relationship between these infant and maternal characteristics and the risk of infant death can be obtained only through linkage of the birth certificate and the death certificate with information on cause and age at death. This preliminary analysis has revealed gradients in infant mortality for all reported characteristics, including at least a twofold gradient by maternal age and education for both blacks and whites. These data confirm the findings of previous studies that indicate a crossover in NNM between blacks and whites, with black infants experiencing lower NNM at lower birthweights and white infants experiencing lower NNM at higher birthweights (12, 13). This crossover is also evident by reported gestational age. For birthweight under 3,000 g, black infants have been reported to experience lower NNM for all combinations of birthweight and gestational age in individual states (12, 13).

These data will be disseminated through detailed tabulations in forthcoming reports that will include numbers and rates for NNM and PNM divided into 500-g intervals (under 4,500 g). In addition to the characteristics for singleton births listed in Table 2 (i.e., infant's sex, gestation, and live-birth order; and mother's age, education, and month prenatal care began), data will be available for type of delivery, number of other previous terminations, and number of prenatal-care visits. All tables for singleton infants will be reported separately for blacks, whites, and all races. For multiple gestations, data include plurality, gestational age, type of delivery, race, and birthweight. For all singleton deaths, data will also be tabulated by underlying cause, birthweight, race, and age at death. Preliminary analysis by cause of death has previously been reported (14).

References

- 1. U.S. Public Health Service. Pregnancy and infant health. In: Promoting health/preventing disease: objectives for the nation. Washington, D.C.: U.S. Department of Health and Human Services, 1980: 15-20.
- 2. Institute of Medicine. Preventing low birthweight. Washington, D.C.: National Academy Press, 1985.
- Armstrong RJ. A study of infant mortality from linked records by birth weight, period of gestation, and other variables, United States. Hyattsville, Maryland; National Center for Health Statistics, 1972; DHEW publication no. (HSM) 72-1055 (Vital and health statistics; series 20; no. 12).
- 4. Lee KS, Paneth N, Gartner LM, Pearlman MA, Gruss L. Neonatal mortality: an analysis of the recent improvement in the United States. Am J Public Health 1980;70:15-21.
- Kleinman JC. Trends and variation in birth weight. Health United States 1981. Hyattsville, Maryland: U.S. Public Health Service, 1981; DHHS publication no. (PHS) 82-1232.
- Williams RL, Chen PM. Identifying the sources of the recent decline in perinatal mortality rates in California. N Engl J Med 1982;306:207-14.
- 7. David RJ, Siegel E. Decline in neonatal mortality, 1968-1977: better babies or better care? Pediatrics 1983;71:531-40.
- Guyer B, Wallach LA, Rosen SL. Birth-weight-standardized neonatal mortality rates and the prevention of low birth weight: how does Massachusetts compare with Sweden? N Engl J Med 1982; 306:1230-3.
- 9. Ericson D, Bjerkedal T. Fetal and infant mortality in Norway and the United States. JAMA 1982; 247:987-91.
- National Center for Health Statistics. Proceedings of the International Collaborative Effort on Perinatal and Infant Mortality, Volume I. Hyattsville, Maryland: National Center for Health Statistics, 1985; DHHS publication no. (PHS) 85-1252.
- 11. Kleinman JC, Kessel SS. The recent decline in infant mortality. Health United States 1980. Hyattsville, Maryland: U.S. Public Health Service, 1980; DHHS publication no. (PHS) 81-1232.
- 12. Binkin NJ, Williams RL, Hogue CJR, Chen PM. Reducing black neonatal mortality: will improvement in birth weight be enough? JAMA 1985;253:327-5.
- 13. Alexander GR, Tompkins ME, Altekruse JM, Hornung CA. Racial differences in the relation of birth weight and gestational age to neonatal mortality. Public Health Rep 1985;100:539-47.
- 14. CDC. Years of potential life lost attributable to low birthweight—United States, 1980 birth cohort. MMWR 1986;35:188-90, 195.

Epidemiologic Notes and Reports

Ammonia Contamination in a Milk Processing Plant — Wisconsin

On October 30, 1985, the Wisconsin Division of Health was informed by the state poison control center of two elementary schoolchildren who presented with severe burning of the mouth and throat, as well as nausea. The symptoms developed within 1 hour of drinking milk packaged in half-pint containers with an expiration date of 11/9 from a Wisconsin milk processor. An investigation into the source of the milk determined that, 5 days previously, the milk processor had noted an ammonia leak in one of its cooling chambers, where approximately 250,000 half-pint milk containers with an expiration date of 11/9 were stored. The liquid ammonia, used to cool the tanks and stored under pressure, had sprayed about the storage tank for an undetermined number of hours. On discovery of the leak, the milk processors destroyed those cartons with obvious external damage to the paper and polyethylene containers. After tasting and smelling approximately 75 of the remaining 250,000 cartons, they determined the milk was safe and began distributing the product throughout the state.

Thirty milk containers with expiration date 11/9 were retrieved from the index elementary school. An analysis of these 30 containers by the Wisconsin Department of Agriculture identified seven (23%) that were contaminated with ammonia at levels ranging from 530 ppm to 1,524 ppm (normal = less than 15 ppm). The pH levels of these contaminated samples ranged from 9.1 to 10.0 (normal milk pH = 6.7-6.9).

On the basis of the initial reports of adverse symptoms associated with ingestion of the implicated milk, a case definition was established: the development of symptoms of irritation of the gastrointestinal tract, including the mouth, throat, or stomach, with onset within 1 hour of ingesting milk with expiration date 11/9 from the implicated processing plant.

Over the next 24 hours, 268 schools that had received milk with expiration date 11/9 from the implicated plant were contacted and instructed to withdraw these milk products from their schools. Additionally, each school was requested to inform the Wisconsin Division of Health if any child developed symptoms consistent with the case definition.

This surveillance effort identified approximately 520 cartons of milk ingested before notification. Twenty children fulfilling the case definition were identified (attack rate 3.9%). None required hospitalization, and no deaths occurred. Schools were instructed to return the unused cartons to the milk processer, where they were destroyed. This is the first reported incident of acute ammonia poisoning associated with contaminated milk.

Reported by M Ziarnik, W Otto, T Sieger, MS, C Gannon, H Anderson, MD, Section of Environmental and Chronic Disease Epidemiology, Wisconsin Div of Health; Div of Field Svcs, Epidemiology Program Office, Div of Environmental Hazards and Health Effects, Center for Environmental Health, CDC.

Editorial Note: Ammonia (NH₃) is a colorless gas with a characteristic strong, pungent, penetrating odor. It is one of the more common industrial chemicals; an estimated 20-30 million tons are used per year in the United States (1,2). It is widely used in fertilizer manufacture; other uses include dye, synthetic fiber, plastic, and nitric acid production, as well as refrigeration (2). In its aqueous form as ammonium hydroxide (NH₄OH), it is extremely alkaline and can be highly caustic (2). Aqueous ammonia is 28% (280,000 ppm) ammonia, whereas household ammonia is 10% ammonia (100,000 ppm) (3). Mild to moderate ammonia exposures can produce headaches, salivation, burning of the throat, anosmia, nausea, vomiting, and substernal pain. Moderate doses may produce laryngospasm or bronchospasm (1).

The Occupational Safety and Health Administration standard for ammonia inhalation is 50 ppm as an 8-hour time-weighted average, but the National Institute for Occupational Safety and Health has recommended that 50 ppm be a 5-minute ceiling for exposure. The characteristic ammonia odor is readily perceptible below toxic levels. Most persons can

MMWR

Ammonia Contamination - Continued

detect an odor at 30 ppm, and eye and nose irritation become more severe as the levels increase to 50 ppm (4). The students involved in this incident were unable to smell the ammonia probably because the milk cartons were closed. The students first became aware of a problem when they felt burning in their throats.

Outbreaks of ammonia poisoning of milk, other beverages, or food have not been previously documented. The ammonium hydroxide apparently penetrated the milk cartons when the refrigerant tank leaked. Additional studies need to be done to determine how the ammonia contaminated the milk, and criteria need to be established to prevent contaminated milk from being distributed.

Recommendations for ammonia spills are as follows (1,5):

- 1. Following ammonia ingestion, a conscious person should immediately be given large quantities of water to dilute the ammonia.
- 2. Persons who have inhaled ammonia should be observed closely for visual disturbances, upper airway obstruction, and hypoxia.
- 3. The area of the ammonia spill or leak should be ventilated to disperse the gas. A flow of gaseous ammonia should be stopped; liquid ammonia should be allowed to vaporize.
- 4. Persons not wearing protective equipment and clothing should be restricted from areas of spills or leaks until the clean-up has been completed.

References

- 1. Rom WN, Barkman H. Respiratory irritants. In: Rom WN, ed. Environmental and occupational medicine. Boston: Little, Brown and Company, 1983:273-4.
- 2. Anderson HA. Irritant gases and fumes. In: Last JM, ed. Public health and preventive medicine, 11th ed. New York: Appleton-Century-Crofts, 1980:760-70.
- Wands RC. Alkaline materials. In: Clayton GD, Clayton FE, eds. Patty's industrial hygiene and toxicology. Third revised edition. New York: John Wiley & Sons, 1981:3045-70.
- National Institute for Occupational Safety and Health. Criteria for a recommended standard . . . occupational exposure to ammonia. Washington, D.C.: Public Health Service, CDC, 1974; publication no. (NIOSH) 74-136.
- 5. Occupational health guidelines for ammonia. U.S. Department of Health and Human Services, U.S. Department of Labor, September 1978.

Current Trends

Rubella Vaccination during Pregnancy — United States, 1971-1985

From January 1971 through December 1985, 1,142 pregnant women who received rubella vaccine either within 3 months before or 3 months after their presumed dates of conception were reported to CDC. These women were followed prospectively to determine the risk of fetal abnormalities following exposure to the vaccine.

Cendehill and HPV-77 Vaccines. Before April 1979, data were collected on 538 women vaccinated during pregnancy with either Cendehill or HPV-77 rubella vaccine (1). The outcomes of conception—live birth, stillbirth, or spontaneous or induced abortion—were known for 143 (96%) of the 149 women known to be susceptible at the time of vaccination. Ninety-four (66%) of these 143 women carried their infants to term. All gave birth to infants free of defects compatible with congenital rubella syndrome (CRS) (2), although eight infants had serologic evidence of intrauterine infection (1,3). These eight children were all followed for at least 2 years, at which time all were growing and developing normally. The longest follow-up

Rubella Vaccination – Continued

is for a child who is now 10½ years old who had both an elevated rubella-specific immunoglobulin M (IgM) titer at birth and persistence of hemagglutination inhibition (HI) antibodies. Although he is still HI-antibody positive (he has not been vaccinated), he continues to grow and develop normally.

An additional 196 infants born to women who either were immune (22) or of unknown immune status (174) at the time of vaccination were also free of CRS-associated defects. Three other women (one susceptible, one immune, and one of unknown immune status) received unknown strains of rubella vaccine. All three delivered normal-appearing, healthy infants.

RA 27/3 Vaccine. Since licensure of the RA 27/3 rubella vaccine in January 1979, 614 pregnant women who received this vaccine during pregnancy have been reported to CDC (Table 3). Two hundred three (33%) of these 614 women were known to be susceptible at the time of vaccination. Outcomes of pregnancy are known for 191 (94%) of these susceptible women. Of the 191 women, 153 (80%) delivered 155 live infants. An additional 30 immune women and 319 women of unknown immune status delivered 350 live infants. All 505 infants were free of defects compatible with CRS.

(Continued on page 281)

		17th Week Endi	ng	Cumu	lative, 17th We	ek Ending
Disease	Apr. 26, 1986	Apr. 27, 1985	Median 1981-1985	Apr. 26, 1986	Apr. 27, 1985	Median 1981-1985
Acquired Immunodeficiency Syndrome (AIDS)	253	180	N	4,111	2,210	N
Aseptic meningitis	100	81	69	1,391	1,164	1,301
Encephalitis: Primary (arthropod-borne	,					
& unspec.)	10	12	16	253	295	295
Post-infectious	1	5	2	26	45	31
Gonorrhea: Civilian	14.655	15,252	16,983	260,725	254,401	289,192
Military	270	478	450	5,060	6,078	7,851
Hepatitis: Type A	426	436	438	7,243	7,002	7,439
Type B	528	495	473	8,080	8,081	7,436
Non A, Non B	76	88	N	1.083	1,373	N
Unspecified	89	114	158	1.612	1,712	2,375
Legionellosis	15	17	N	181	201	N
Leprosv	7	7	6	93	137	73
Malaria	18	23	25	227	230	230
Measles: Total*	185	185	134	2.040	1,001	1,001
Indigenous	176	140	N	1,976	816	N
Imported	9	45	N	64	185	N
Meningococcal infections: Total	54	61	70	1.050	1,012	1,185
Civilian	54	60	70	1.048	1,009	1,183
Military		1	-	2	3	5
Mumps	67	вò	110	1,103	1,358	1.414
Pertussis	61	26	33	708	495	495
Rubella (German measles)	10	13	58	172	135	396
Syphilis (Primary & Secondary): Civilian	532	574	586	7.971	8,125	9,838
Syphilis (Frimary & Secondary). Civilian Military	2	9	11	72	67	122
Toxic Shock syndrome	11	9	Ň	123	128	N
Tuberculosis	431	532	499	6.280	6.364	7,157
Tularemia	431	332		19	26	32
Typhoid fever	i 7	6	ż	72	85	121
	8	10	10	28	34	36
Typhus fever, tick-borne (RMSF) Rabies, animal	140	151	151	1,664	1,567	1,885

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

TABLE II. Notifiable diseases of low frequency, United States

	Cum 1986		Cum 1986
Anthrax Botulism: Foodborne (Mich. 1) Infant (Md. 1, Ariz. 1, Calif. 2) Other Brucellosis (Nebr. 1) Cholera Congenital rubella syndrome Congenital syphilis, ages < 1 year Diphtheria	4 20 17 1 1 1	Leptospirosis (N.C. 1) Plague Poliomyelitis, Paralytic Psittacosis Rabies, human Tetanus Trichinosis Typhus fever, flea-borne (endemic, murine)	15 - - 16 - 12 7 6

*Four of the 185 reported cases for this week were imported from a foreign country or can be directly traceable to a known internationally imported case within two generations.

			Apr	il 26, 19	986 and A	pril 27, 198	85 (17th	Week)				
	AIDS	Aseptic	Ence	phalitis	Gon	orrhea	н	lepatitis (V	(iral), by ty		Legionel-	
Reporting Area	AIDS	Menin- gitis	Primary	Post-in- fectious		vilian)	A	В	NA,NB	Unspeci- fied	losis	Leprosy
	Cum. 1986	1986	Cum. 1986	Cum. 1986	Cum. 1986	Cum. 1985	1986	1986	1986	1986	1986	Cum. 1986
UNITED STATES	4,111	100	253	26	260,725	254,401	426	528	76	89	15	93
NEW ENGLAND	171	4	9	1	6,022	7,865	7	25	3	4	1	1
Maine N.H.	9 7	-	2	-	306 162	323 163	-	-	1	-	1	-
Vt. Mass.	2 92	1 1	2 2	1	94 2,601	81 2,893	1 2	17	1	3	-	1
R.J.	9	-	-	-	567	571	1	3	-	1	-	-
Conn.	52	2	3	-	2,292	3,834	3	5	1	-	-	-
MID ATLANTIC Upstate N.Y.	1,567 122	9 2	43 15	-	46,216 5,236	35,689 5,053	27 3	44 4	6 1	21 3	-	9 1
N.Y. City	1,107	-	10	-	27,015	16,159	2	2	-	17	-	7
N.J. Pa.	251 87	6 1	5 13	-	6,154 7,811	7,048 7,429	6 16	17 21	3 2	- 1	-	1
E.N. CENTRAL	215	10	49	4	30,177	36,038	16	42	1	3	3	4
Ohio	30	3	15	2	8,812	9,457	6	9	-	2	2	-
Ind. III.	25 106	2	5 7	2	4,059 4,638	3,326 9,726	3	3	-	-	-	3
Mich. Wis.	49 5	5	21	-	10,797 1,871	10,699 2,830	7	30	1	1	1	1
	-						- 14	- 15	2	-	- 1	1
W.N. CENTRAL Minn	78 37	2	7 4	5	11,778 1,787	13,060 1,904	7	5	1	-	-	i
lowa Mo.	8	-	3	-	1,166	1,386 6,004	3	2 5	1	-	-	-
N. Dak.	19 2	-	-	-	5,770 112	93	-	-	-	-	-	-
S. Dak Nebr	1 3	1	-	-	235 867	239 1,304	1	2	-	-	1	-
Kans	8	1	-	5	1,841	2,130	3	1	-	-	-	-
S. ATLANTIC	555	26	44	11	63,670	54,830	46	100	16	13	8	1
Del Md	10 59	1 5	3 10	-	1,098 7,901	1,202 8,745	1 12	1 13	1	2	-	-
D.C.	78	-	-	-	5,271	4,655	-	11	-	-	-	-
Va. W. Va.	59 2	5	17	-	5,764 830	5,867 774	1 2	7	2 2	1	1	1
N.C.	24	3	7	1	11,225	10,179	2	12	2	2	2	-
S.C. Ga	15 64	1 2			6,081 6,682	6,911	1	19 8	1	1	2 1	
Fla	244	9	1	10	18,818	16,497	27	26	8	6	2	-
E.S. CENTRAL	39	4	19	1	22,367	22,242	14	45	4	5	1	-
Ky. Tenn	12 17	2	8 1	1	2,623 8,815	2,449 8,792	9 3	8 19	2	3 2	-	-
Ala	6	-	9	-	6,256	7,023	2	14	4	-	1	-
Miss	4	-	1	-	4,673	3,978	-	4	-	-	-	-
W.S. CENTRAL Ark	344 10	13	20	1	33,371 3,170	35,834 3,440	42	56 2	7	21 2	-	6
La	51	-	2	-	5,952	7,340	1	2	2	1	-	-
Okla. Tex.	16 267	13	4 14	1	3,856 20,393	3,698 21,356	9 32	7 45	1 4	18	-	-
MOUNTAIN	86	6	12	1	8,588	8,436	43	35	8	1	-	7
Mont.	2	-	-	i	220	255	1	1	-	-	-	-
ldaho Wyo	1	-	2	-	254 195	278 211	1	1	1	:	-	:
Colo	37	3	2	-	2,178	2,571	4	6	-	-	-	3
N. Mex. Ariz.	6 22	1	1 5	-	847 2,745	991 2,462	7 20	4 19	- 6	1	-	2
Utah Nev	6 10	-	1	-	346 1,803	351 1,317	1	1	1	-	:	2
		-						-		-		
PACIFIC Wash	1,056 34	26 3	50 5	2	38,536 2,854	40,407 2,870	217 11	166 15	29 4	21 1	1	64 7
Oreg. Calif.	21	23	43	2	1,503 32,761	2,036 33,886	25 181	10 136	6	20	1	48
Alaska	983 8	23	43	-	1,000	987	-	3	18 1	20	-	-
Hawaii	10	-	-	-	418	628	-	2	-	-	-	9
Guam P.R.	-	1	2	-	32 748	59 1,263	1	2	-	-	-	1
V.I.	31	ů	-	-	71	151	Ū	6 U	Ū	14 U	Ū	-
Pac. Trust Terr. Amer. Samoa	-	-	-	-	57	322	14	-	-	2	-	1
	-		-	-	13	-	4	•	-	-	-	-

TABLE III. Cases of specified notifiable diseases, United States, weeks ending April 26, 1986 and April 27, 1985 (17th Week)

N: Not notifiable

U: Unavailable

			мрін	20, 1	900 a		ril 27, 19	00 (1	/	ek)					
	Malaria			sles (Rut			Menin- gococcal	Mur	nps		Pertussis			Rubella	
Reporting Area	Cum.	Indig	enous Cum.	1986	rted * Cum.	Total Cum.	Infections Cum.	1986	Curn.	1986	Cum.	Cum.	1986	Cum.	Cum.
UNITED STATES	1986 5 227	176	1986 1,976	9	1986 64	1985 1,001	1986 1,050	67	1986 1,103	61	1986 708	1985 495	10	1986 172	1985
NEW ENGLAND		6	16	-	-	69	77	2	34	4	42	495 23	-	1/2	135
Maine N.H.	-	:	:	:	-	-	16 3	-	10	-	2	2 13	2	1	2
Vt. Mass.	1 8	- 6	15	:	-	- 68	11 16	-	- 1	1	2 9	2 3	-	-	-
R.I. Conn.	1	-	1	-	-	-	11 20	1	5 18	- 3	1	1	-	-	3
MID ATLANTIC	26	60	785		3	66		1	61	3 1	14 83	2 57	-	-	-
Upstate N.Y.	4	-	2	-	2	30	49		25	1	57	32		23 15	35
N.Y. City N.J.	8 3	10 50	101 682	2	1	23		-	5	-	3	8	-	5	10
Pa.	11	-		-	-	76	27 58	1	15 16	-	5 18	1 16	-	3	5 12
E.N. CENTRAL Ohio	6 1	7	211	-	2	350 38		20 4	529 57	1 1	138 63	72 13	-	5	11
Ind.	-	-	-	-	-	1	13	-	15	-	16	11	-	-	-
III. Mich.	3 2	7	125	-	-	216 48		16	276	-	16	12	-	4	5
Wis.	-	-	86	-	2	48		16	90 91	-	16 27	7 29	-	1	5 1
W.N. CENTRAL Minn.	6 2	3	86	1	3	5		8	55	-	33	44	1	7	8
lowa	1		1	-	1	2	12 6	2	1 10	-	16 5	11 3	-	-	-
Mo. N. Dak.	2	2	2	-	1	2		4	13	-	4	8	-	1	-
S. Dak.	-	-		-	-	-	1		2 1	-	2	6	-	-	1
Nebr. Kans.	1	1	83	ī †	-	-	7	-	-	-	-	1	-		-
S. ATLANTIC	-					1		2	28	-	6	15	1	6	7
Del.	29	23 1	284 1	1	8	115	224 1	5	83	40 34	213 102	120	-	6	12
Md.	3	2	11	1†	5	12	29	2	6	1	24	41		-	1
D.C. Va.	6	1	3	2	1	2 12		3	15	-	-	-	-	-	-
W. Va.	-	-	2	-	-	3		-	26	-	9 4	3		-	2
N.C. S.C.	3	19	256	-	-	-	37	-	7	-	15	7	-	-	-
Ga.	3	-	250	-	1	8		-	11 5	1	3 49	46	-	-	2
Fla.	12	-	11	-	1	78	56	-	13	-	7	23	-	6	7
E.S. CENTRAL	5	-	1	-	-	-	58	1	14	-	15	4	-	1	1
Ky. Tenn.	2	:	1	2	-	-	10 26	1	2 10	:	1	1	-	1	1
Ala.	2	-	-	-	-	-	15		1	-	5 9	1	:	-	-
Miss.	1	-	-	-	-	-	7	-	1	-	-	-	-	-	-
W.S. CENTRAL Ark.	17	9	304 273	4	22	48	78 10	9	84 6	1	26 2	57 9	:	35	13
La. Okla.	4	-	-	- 6	-	1	9	-	-	-	3	2	-	-	1
Tex.	· 2 11	9	31	2 § 2 †	4 18	47	12 47	N 9	N 78	1	21	46	2	35	12
MOUNTAIN Mont	6	21	115	2	10	255		16	125	4	86	22	1	1	3
Idaho	1	-	-	-	1	129 4		1	3 2	1	1	3	-	-	-
Wyo.	-	-	-		-	-	2	-		-	26	-		-	1
Colo. N. Mex.	1	1	16	2§	5	5		-	6	2	18	8	-	-	-
Ariz.	2	20	99	-	4	2 115		N 15	N 110	1	9 23	3	1	-	1
Utah Nev.	1	-	:	-	-	-	4	-	1	-	9	4	-	1	1
PACIFIC	119	47	174	1	16	- 93		5		-		-	-	•	-
Wash.	10	12	35	-	7	93		5	118 5	10 1	72 26	96 15	8	93 1	47
Oreg. Calif.	9 100	-	-	1 §	2	3	15	N	N	-	5	16	-	-	1
Alaska		35	120	- 13	6	82	163 7	5	103 4	9	38 1	60 2	8	91	34
Hawaii	-	-	19	-	1	7		-	6	-	2	23	:	1	12
Guam P.R.	1	-	3	-	-	10		-	2	-	-	-	-	2	1
V.I.	3	Ū	-	Ū	-	40			15	1	4	1	-	58	6
Pac. Trust Terr.	-	-	-	-	-	9		U -	7	U -	-	-	U	-	-
Amer. Samoa	-		-	-	-		-	-		-	:	:	-	-	-

TABLE III. (Cont'd.) Cases of specified notifiable diseases, United States, weeks ending April 26 1986 and April 27 1985 (17th Week)

For measles only, imported cases includes both out-of-state and international importations. §Out-of-state U: Unavailable

N: Not notifiable

		April 2	6, 1986 an	d April 2	7, 1985 (*	17th Wee	k)		
Reporting Area	Syphilis (Primary & S	(Civilian) Secondary)	Toxic- shock Syndrome	Tuber	culosis	Tula- remia	Typhoid Fever	Typhus Fever (Tick-borne) (RMSF)	Rabiés, Animal
	Cum. 1986	Cum. 1985	1986	Cum. 1986	Cum. 1985	Cum. 1986	Cum. 1986	Cum. 1986	Cum. 1986
JNITED STATES	7,971	8,125	11	6,280	6,364	19	72	28 + 8	1,664
NEW ENGLAND	158	183	-	195	222	-	3	1	1
N.H.	10 6	7 3	-	18 4	16 7	-	-	-	-
/t. Aass	6 75	96	-	7 103	3 132	-	2	1	-
ul.	10	6	-	11	21	-	-	-	1
Conn.	51	71	-	52	43	-	1	-	-
ID ATLANTIC	1,140 62	1,078 77	-	1,244 197	1,190 189	-	7	1	151 24
Y City	636	671	-	610	620	-	4	-	1
l.J. a.	224	229	:	239	116		2	-	-
	218	101		198	265	-		-	126
.N. CENTRAL	210 45	378 39	1	796 122	772 140	-	4	2 + 2 2 2	30 2
d.	41	32	-	96	90	-		2 L	7
lich	58 46	192 96	1	340 193	343 156	-	3	-	10 4
Vis.	20	19	-	45	43	-	1	-	7
N. CENTRAL	80	86	1	173	165	6	3	1	227
1inn Swa	12	23	-	45	33	-	1	-	26
1 0.	5 45	12 32	1	14 85	26 74	1 5	2	-	54 22
Dak. Dak	2	4	-	2	2	-	-	-	58
ebr	8	4	-	6 4	7 7	-	-	-	41 5
ans.	7	9	-	17	16	-	-	1	21
ATLANTIC	2,274	2,027	6	1,251	1,279	4	7	7+1	393
el. Id	12 155	16 142	2	13 92	13 110	- 1	1	-	231
.C.	116	118	-	51	60	-	-	-	-
a. V. Va.	147	111	1	120 43	105 28	1	2	1	73 9
.C.	176	237	2	183	162	1	2	2	ĩ
a.	235 256	253	1	139 163	150 185	1	-	3	11 47
la	1,170	1,146	-	447	466	-	2	-	21
S. CENTRAL	584	742	-	570	568	3	-	7+2	104
y. enn	25 223	31 207	-	150 154	112 170	2 1	-	11	25 55
la	194	236	-	182	196	-	-	21	24
liss	142	268	-	84	90	-	-	3	-
S. CENTRAL	1,725	2,062	-	757	715	5	3	8 +3	247
rk. B.	88 293	105 352	-	90 145	75 119	3	-	-	51 6
kla.	51	60	-	65	75	2	1	6 3	20
BX.	1,293	1,545	-	457	446	-	2	2	170
OUNTAIN	206	259	2	117	149	-	2	1	293
lont. Iaho	2 1	1 2	-	5 5	19 4	-	-		110
vo. olo	-	5		-	3	-	-	1	129
Mex.	63 26	62 36	-	1 32	18 27	-	-	-	3
riz.	90	136	1	57	67	-	1		51
tah ev.	4 20	3 14	1	4 13	5 6	:	1	-	-
ACIFIC	1,594	1,310	1	1,177	1,304	1	43		218
/ash	27	51		67	64	-	43	-	210
reg. alif.	33 1,517	31 1,201	1	44	44 1,087	:	- 20	-	212
laska	-	1	-	991 17	50	1	39	-	212
awaii	17	26	-	58	59	-	2	-	-
uam R.	1	2	-	-	12	-	-	-	-
	284	302	Ū	81 1	99 1	-	2	-	15
.l. ac. Trust Terr.	-								

TABLE III. (Cont'd.) Cases of specified notifiable diseases, United States, weeks ending April 26, 1986 and April 27, 1985 (17th Week)

U: Unavailable

TABLE IV. Deaths in 121 U.S. cities,* week ending
April 26, 1986 (17th Week)

		All Caus	es, By A	ge (Year	s)					All Cause	es, By Ag	ge (Years	;)		
Reporting Area	All Ages	≥65	45-64	25-44	1-24	<1	P&I** Total	Reporting Area	All Ages	≥65	45-64	25-44	1-24	<1	P&I** Total
NEW ENGLAND	612	435	122	28	16	11	45	S. ATLANTIC	1,255	792	290	104	37	32	66
Boston, Mass.	156	99	37	12	7	1	16	Atlanta, Ga.	146	83	40	12	4	7	4
Bridgeport, Conn.	35	25	7	3	-	-	1	Baltimore, Md.	226	148	49	17	9	3	7
Cambridge, Mass. Fall River, Mass.	27 33	25 27	1	1	2	-	2 1	Charlotte, N.C.	61 129	33	14	9	2	3	7
Hartford, Conn.	56	39	11	3	2	1	4	Jacksonville, Fla. Miami, Fla.	108	84 59	28 38	7 9	7	3	10
Lowell, Mass.	15	13	1	ĩ	-	-	-	Norfolk, Va.	38	19	13	5	'	1	2 6
Lynn, Mass.	15	12	3	-	-	-	1	Richmond, Va.	78	49	18	6	3	2	2
New Bedford, Mass		17	3	-	-	1	1	Savannah, Ga.	54	39	10	2	2	1	10
New Haven, Conn. Providence, R.I.	48 69	27 52	11 15	4	4	2 2	3 4	St. Petersburg, Fla		95	14	5	-	2	9
Somerville, Mass.	5	3	2		-	2	4	Tampa, Fla. Washington, D.C.	62 208	47 122	10	2	1	2	3
Springfield, Mass.	47	29	13	2	-	3	6	Wilmington, Del.	208	14	46 10	27 3	6 2	7	6
Waterbury, Conn.	35	30	4	1	-	-	2	tennington, Der	20	17	10	5	~	•	-
Worcester, Mass.	50	37	10	1	1	1	4	E.S. CENTRAL	697	425	177	49	25	21	32
MID ATLANTIC	2.594	1,703	546	232	51	60	122	Birmingham, Ala.	125	79	36	6	2	2	1
Albany, N.Y.	48	29	13	232	1	3	2	Chattanooga, Ten Knoxville, Tenn.	n. 66 101	43 57	14 30	4 5	1 6	4 3	6 9
Allentown, Pa.	15	13	2	-	-	-	-	Louisville, Ky.	102	62	24	9	4	3	9 5
Buffalo, N.Y.	111	67	30	9	3	2	4	Memphis, Tenn.	98	59	17	12	5	5	2
Camden, N.J. Elizabeth, N.J.	32 26	21	8	4	-	3	-	Mobile, Ala.	70	44	20	5	1	-	4
Erie, Pa.t	49	16 33	6 14	4	-	1	2	Montgomery, Ala.	30	16	10	-	2	2	1
Jersey City, N.J.	53	42	5	3	2	ł	1	Nashville, Tenn.	105	65	26	8	4	2	4
	1,320	838	265	152	32	33	52	W.S. CENTRAL	1,356	771	328	129	74	53	55
Newark, N.J.	56	19	17	16		2	2	Austin, Tex.	52	28	13	6	3	2	55
Paterson, N.J.	30	18	10	2	-	-	5	Baton Rouge, La.	31	22	5	-	-	4	3
Philadelphia, Pa. Pittsburgh, Pa.†	392 68	262 47	93	22	6	9	30	Corpus Christi, Te	x. 32	21	6	2	1	2	3
Reading, Pa.	38	27	13 8	5 2	2	1	2 5	Dallas, Tex.	220	121	52	24	13	10	4
Rochester, N.Y.	136	103	23	4	ż	4	о 8	El Paso, Tex. Fort Worth, Tex.	69 89	38 50	17 23	3 8	6	5	4
Schenectady, N.Y.	22	16	2	3	ī	-	-	Houston, Tex	433	237	102	56	6 23	2 15	3 12
Scranton, Pa.†	39	27	11	-	1	-	1	Little Rock, Ark.	37	21	7	7	1	15	2
Syracuse, N.Y. Trenton, N.J.	81	66	12	3	-	-	4	New Orleans, La.	121	68	37	ż	5	3	-
Utica, N.Y.	31 16	21 11	7	2	-	1	-	San Antonio, Tex.	133	76	30	9	12	6	11
Yonkers, N.Y.	31	27	3	1	-	2	2 2	Shreveport, La. Tulsa, Okla.	49 90	32 57	14 22	1 6	1 3	1	3 6
E.N. CENTRAL												0	3	2	0
Akron, Ohio	2,346 63	1,534 43	516 16	156 1	70 1	70 2	105 3	MOUNTAIN	694	432	143	71	23	24	43
Canton, Ohio	35	29	5			1	3	Albuquerque, N.M. Colo. Springs, Col		55	19	10	8	4	4
Chicago, III.§	561	353	127	47	11	23	16	Denver, Colo.	121	26 65	8 32	4 10	4	1 10	8 3
Cincinnati, Ohio	169	113	39	7	6	4	22	Las Vegas, Nev.	98	55	28	12	2	1	9
Cleveland, Ohio	190	116	47	10	11	6	6	Ogden, Utah	20	15	5		-		1
Columbus, Ohio Dayton, Ohio	173	99	52	10	7	5	2	Phoenix, Ariz.	135	86	22	20	4	3	3
Detroit, Mich.	109 234	73 150	22 50	8 18	3 11	3 5	2 8	Pueblo, Colo.	26	19	6	1	-	-	4
Evansville, Ind.	40	31	50	10	2	5	8	Salt Lake City, Uta Tucson, Ariz.	h 49 109	30	.8	5	2	4	
Fort Wayne, Ind.	53	34	15	2	ī	1	6		109	81	15	9	3	1	11
Gary, Ind.	13	9	2	2	-	-	-	PACIFIC	1,979	1,313	366	179	62	51	102
Grand Rapids, Mich Indianapolis, Ind.		46	13	4	3	3	2	Berkeley, Calif.	21	16	3	-	1	1	1
Madison, Wis.	148	95	37	12	-	4	7	Fresno, Calif.	80	50	17	4	3	6	6
Milwaukee, Wis.	43 167	28 125	8 28	1 8	3 4	3 2	4 6	Glendale, Calif. Honolulu, Hawaii	19 59	14 38	4	Ē	1	:	3
Peoria, III.	44	30	20	4	2	1	4	Long Beach, Calif.	59 79	38 52	12 14	6 9	2	1	3 8
Rockford, III.	36	23	8	3	ĩ	i	4	Los Angeles, Calif.	663	412	136	74	26	28	26
South Bend, Ind.	39	24	6	5	1	3	1	Oakland, Calif.	42	31	5	ĩ	20	3	20
Toledo, Ohio	97	66	17	10	3	1	9	Pasadena, Calif. §	31	24	5	1	-	1	2
Youngstown, Ohio	63	47	10	4	-	2	1	Portland, Oreg.	136	96	22	13	2	3	9
W.N. CENTRAL	740	484	155	51	23	27	28	Sacramento, Calif. San Diego, Calif.	137 132	92	21	15	2	7	8
Des Moines, Iowa	51	39	10	1		2 <i>1</i>	20	San Francisco, Call		88 100	25 26	9 19	7	3 8	13 6
Duluth, Minn.	15	11	4	-	-			San Jose, Calif.	176	121	20	13	4	3	7
Kansas City, Kans.	44	26	13	3	1	1	-	Seattle, Wash.	147	104	23	10	6	4	2
Kansas City, Mo.	110	71	26	5	3	5	4	Spokane, Wash.	53	41	8	3	ĭ	-	6
Lincoln, Nebr. Minneapolis, Minn.	44	33	.7	3		1	3	Tacoma, Wash.	49	34	11	2	1	1	ĩ
Omaha, Nebr.	79 88	56 56	14 24	5 3	1	3	2	TOTAL	to one th	+					
St. Louis, Mo.	88 154	56 91	24 20	3 22	3 13	2	9 2	IUIAL	12,273	7,889	2,643	999	381	349	598
St. Paul, Minn.	65	45	16		13	3	2								
Wichita, Kans.	90		21	9	•	3									

* 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.

P Because of changes in reporting methods in these 3 Pennsylvania cities, these numbers are partial counts for the current week. Complete counts will be available in 4 to 6 weeks.
 t1Total includes unknown ages.
 § Data not available. Figures are estimates based on average of past 4 weeks.

Vol. 35/No. 17

Rubella Vaccination - Continued

The dates of vaccination and estimated dates of confinement were known for all 153 susceptible women who had full-term pregnancies (Figure 3). Fifty-three (35%) women were vaccinated within 1 week before to 4 weeks after conception, the period of presumed highest risk.

Serologic evaluations (rubella HI and specific IgM titers on cord or neonatal blood specimens) were performed on 121 (78%) of the 155 infants whose mothers were susceptible. One normal-appearing infant had a rubella-specific IgM antibody titer of 1:8 in cord blood and a corresponding HI titer of 1:128. The maternal titer was also 1:128. Retesting of cord blood and testing of a 2-month follow-up specimen run simultaneously showed a decline in HI antibody over the 2-month period from a titer of 1:64 to 1:16, suggesting that subclinical infection may not have occurred, since this pattern of decrease would be expected in maternally

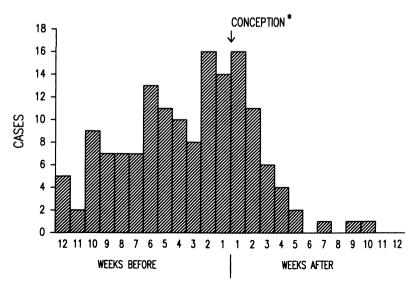
TABLE 3. Pregnancy outcomes for 614 recipients of RA 27/3 vaccine — United States, January 1979 through December 1985

Prevaccination immunity status	Total women	Live births	Spontaneous abortions and stillbirths	Induced abortions	Outcome unknown
Susceptible	203	155*	8	30	12
Immune	32	30	1	0	1
Unknown	379	320†	8	24	28
Total	614	505	17	54	41

*Includes two twin births.

[†]Includes one twin birth.

FIGURE 3. Interval between receipt of rubella RA 27/3 vaccine and estimated date of conception, in weeks, among 153 susceptible women with live births — United States, January 1979 through December 1985



*Two cases not included on estimated day of conception.

Rubella Vaccination - Continued

derived antibody. The infant had no evidence of defects compatible with CRS at birth or at the 18-month and 29-month follow-up examinations. Further follow-up sera could not be obtained to document persistence or disappearance of HI antibodies.

Blood studies were also obtained on 156 of the 320 infants born to mothers whose immune status was unknown at the time of vaccination. Subclinical infection was documented in two infants. One infant had a rubella-specific IgM antibody titer of 1:16 in cord blood. Both mother and infant had HI titers of 1:32 at the time of birth; the infant had a persistent HI titer of 1:32 at 4 months of age. This infant had no evidence of defects compatible with CRS at birth or at the 10-month and 17-month examinations. A serum specimen was not obtained at the follow-up visits. The second infant had a persistent HI titer of 1:8 at 3 months of age, suggesting that there had been subclinical infection. This infant was diagnosed as normal at the 3-month follow-up visit.

While none of the 155 infants born to susceptible women had defects compatible with CRS, two infants did have asymptomatic glandular hypospadias. However, both had negative rubella-specific IgM titers (less than 1:4) in cord blood at birth. A 6-month follow-up serum was available for one of the infants; he had a rubella HI antibody titer of less than 1:8 (i.e., a negative titer).

Reported by Surveillance, Investigations, and Research Br, Div of Immunization, Center for Prevention Svcs, CDC.

Editorial Note: Since 1971, CDC has maintained a register to monitor and quantitate the risks to the fetus following exposure to attenuated rubella vaccine virus. Data are obtained through reports from physicians and from state and local health departments, as well as directly from women vaccinated either within 3 months before or 3 months after conception. The patients are followed prospectively to determine the outcome of pregnancy. In 1979, when RA 27/3 rubella vaccine replaced the other rubella vaccines, concern was raised that it might have greater fetotropic and teratogenic potential than earlier vaccines. As with the other vaccines, data collected so far show no evidence that the RA 27/3 rubella vaccine can cause defects compatible with CRS.

Fifty-three (35%) of the 153 susceptible mothers were vaccinated with RA 27/3 vaccine during the presumed highest-risk period for viremia and fetal defects (1 week before to 4 weeks after conception) (4,5). None of their infants were born with CRS; therefore, the observed risk of CRS following rubella vaccination continues to be zero (Table 4). The theoretical maximum risk for the occurrence of CRS in this group of 155 children, however, based on the 95% confidence limits of the binomial distribution, could be as high as 2.4%. (If the 95 infants exposed to other rubella vaccines are included, the maximum theoretical risk is 1.5%.) This overall maximum risk remains far less than the 20% or greater risk of CRS associated with maternal infection with wild rubella virus during the first trimester of pregnancy (3) and is no greater than the 2%-3% rate of major birth defects in the absence of exposure to rubella vaccine (6).

These favorable data are consistent with the experiences in the Federal Republic of Germany and the United Kingdom (7,8). In neither country has the vaccine been associated with the occurrence of CRS among infants born to susceptible mothers who had been vaccinated. In Germany, 98 susceptible women vaccinated with either the Cendehill or RA 27/3 strain of vaccine gave birth to infants with no evidence of CRS. In the United Kingdom, one of 42 infants born to vaccinated mothers (rubella-immune status not specified) had pulmonic valve atresia but had no rubella-specific IgM or other serologic evidence of rubella infection.

The occurrence of any congenital defect following maternal vaccination deserves careful analysis and follow-up. In the U.S. series, two infants born to susceptible mothers had asymptomatic glandular hypospadias. While hypospadias has been noted in CRS cases (9,10),

MMWR

Rubella Vaccination - Continued

there are no data to suggest that glandular hypospadias should be considered a CRSassociated defect. In any case, neither of the two infants in question had serologic evidence of rubella virus infection. Ten other infants born to mothers of unknown immune status (eight) or known to be immune (two) at the time of vaccination had some type of defect (11). However, none of the defects were compatible with CRS, and serologic testing, when done, did not confirm rubella virus infection.

While no CRS-like defects have been noted, it is clear that rubella vaccine viruses, including the RA 27/3 strain, can cross the placenta and infect the fetus. Approximately 1%-2% of infants born to susceptible vaccinees had serologic evidence of subclinical infection, regardless of vaccine strain (3). On the other hand, while the rubella virus isolation rate from the products of conception for the RA 27/3 vaccine is only 3% (1/34), the rate of virus isolation for Cendehill and HPV-77 vaccines is 20% (17/85) (3). These data indicate that the risk of placental or fetal infection from RA 27/3 vaccine is minimal.

In view of the data collected through 1985, the Immunization Practices Advisory Committee (ACIP) continues to state that: (1) pregnancy remains a contraindication to rubella vaccination because of the theoretical, albeit small, risk of CRS; (2) reasonable precautions should be taken to preclude vaccination of pregnant women, including asking women if they are pregnant, excluding those who say they are, and explaining the theoretical risks to the others; and (3) if vaccination does occur within 3 months before or after conception, the risk of CRS is so small as to be negligible; thus, rubella vaccination of a pregnant woman should not ordinarily be a reason to consider interruption of pregnancy. The patient and her physician, however, should make the final decision (*12*).

CDC encourages reporting of cases to its register. Only cases involving women known to have been susceptible at the time of vaccination, by virtue of a negative serology obtained within the preceding year, should be reported to the Division of Immunization (telephone: [404] 329-1870). Although laboratory services for culture of placental and fetal tissue are no longer routinely available at CDC, services for serologic determination continue to be available for susceptible cases that are reported.

References

- 1. CDC. Rubella vaccination during pregnancy—United States, 1971-1981. MMWR 1982;31:477-81.
- 2. CDC. Rubella and congenital rubella United States, 1983. MMWR 1984;33:237-42, 247.
- 3. Preblud SR, Stetler HC, Frank JA Jr, Greaves WL, Hinman AR, Herrmann KL. Fetal risk associated with rubella vaccine. JAMA 1981;246:1413-7.
- O'Shea S, Parsons G, Best JM, Banatvala JE, Balfour HH Jr. How well do low levels of rubella antibody protect? [Letter]. Lancet 1981;II:1284.
- Balfour HH Jr, Groth KE, Edelman CK, Amren DP, Best JM, Banatvala JE. Rubella viraemia and antibody responses after rubella vaccination and reimmunization. Lancet 1981;1:1078-80.

Vaccine	Susceptible	Normal live	Risk	of CRS
strain	vaccinees	births	Observed	Theoretical
RA 27/3	153	155†	0	0%-2.4%
Cendehill or HPV-77	94	94	0	0%-3.8%
Unknown	1	1	0	-
Total	248	250	0	0%-1.5%

 TABLE 4. Maximum theoretical risks of congenital rubella syndrome (CRS) following rubella vaccination, by vaccine strain — United States, 1971-1985*

*Through December 31, 1985. No women entered in the register after 1980 were vaccinated with Cendehill or HPV-77 vaccine.

^TIncludes two twin births.

Rubella Vaccination – Continued

- CDC. Congenital malformations surveillance report, January 1981-December 1983. Issued September 1985.
- 7. Enders G. Rubella antibody titers in vaccinated and nonvaccinated women and results of vaccination during pregnancy. Rev Infect Dis 1985;7(suppl 1):S103-7.
- Tobin JO, Sheppard S, Smithells RW, Milton A, Noah N, Reid D. Rubella in the United Kingdom, 1970-1983. Rev Infect Dis 1985;7(suppl 1):S47-S52.
- Desmond MM, Montgomery JR, Melnick JL, Cochran GG, Verniaud W. Congenital rubella encephalitis. Effects on growth and early development. Am J Dis Child 1969;118:30-1.
- 10. Ziring PR. Congenital rubella: the teenage years. Pediatr Annal 1977;6:762-70.
- CDC. Rubella vaccination during pregnancy United States, 1971-1982. MMWR 1983;32:429-32, 437.
- 12. ACIP. Rubella prevention. MMWR 1984;33:301-10, 315-8.

Human T-Lymphotropic Virus Type III/ Lymphadenopathy-Associated Virus Antibody Testing at Alternate Sites

On March 2, 1985, an enzyme-linked immunosorbant assay (ELISA) test to detect antibodies to human T-lymphotropic virus type III/lymphadenopathy-associated virus (HTLV-III/ LAV) was licensed by the U.S. Food and Drug Administration to screen blood and plasma collected for transfusion or manufactured into other products. Since it was recognized that many individuals in groups at high risk for AIDS might want testing to determine their antibody status, federal funds for alternate testing sites were made available so that HTLV-III/LAV antibody tests could be obtained free of charge outside the blood-bank setting. A primary goal was to protect the nation's blood supply by limiting the potential for donation of falsenegative units. The alternate sites were also needed to ensure that individuals wishing to be tested would receive appropriate pretest counseling, post-test counseling, and referral for medical evaluation, if indicated.

Cooperative agreements between CDC and 55 state and local health departments began April 26, 1985. The cooperative agreements were for a 90-day period, since they were intended to defray start-up costs only. Most agreements were subsequently extended for an additional 90 days without additional funding at the request of the individual health departments. Preliminary data on the activities supported by the cooperative agreements were reported to CDC in September 1985 and January 1986. As of September 6, 1985, at least one alternate testing site had been established by 52 of the 55 project areas; an estimated 518 sites had been established nationwide; and 21,200 persons had been tested.

Activities increased substantially during the last quarter of 1985. By December 31, 1985, 874 testing sites had been established in 53 project areas (Table 5). This total included 275 sites in New York City located in private physicians' offices. Nationwide, 79,100 persons had been tested. Pretest counseling had been provided to 93,900 persons, and post-test counseling, to 55,500. A total of 17.3% of the individuals tested at these sites had repeatedly reactive ELISA tests. No relationship was noted between the number of acquired immunodeficiency syndrome (AIDS) cases reported in a particular project area and the number of tests per-

284

TABLE 5. Alternate testing site activities - United States, 1985

Area	Testing sites	Pretest sessions	Persons tested	Post-test sessions	Percent positive*
UNITED STATES	874	93,917	79,083	55,499	17.3
New England					17.5
Maine	4	42	42	42	9.5
N.H.	i	73	429	53	
Vt.	19	Ő	110	110	9.8
Mass.	7	1,400	600	450	2.7
R.I.	1	308	695	214	11.8
Conn.	ò	0	095		10.6
	0	0	0	0	
Mid-Atlantic	•				
Upstate N.Y.	8	2,376	1,697	1,254	9.0
N.Y. City	275	7,042	2,032	2,032	30.7
N.J.	4	1,844	1,818	246	13.5
Pa.	7	2,204	1,608	1,333	10.1
E.N. Central					
Ohio	7	3,174	2,780	2,500	17.2
Ind.	9	3,338	827	756	18.1
111.	3	280	221	0	13.1
Mich.	5	2,633	1,897	303	15.1
Wis.	30	1,050	1,021	1,010	12.2
W.N. Central		1,000	1,021	1,010	12.2
Minn.	4	1 700	1 7 1 7		10.0
		1,730	1,717	1,614	13.8
lowa	11	947	947	67	7.1
Mo.	12	1,241	1,026	851	18.6
N. Dak.	2	120	120	120	5.0
S. Dak.	2	4	4	4	50.0
Nebr.	11	235	199	141	24.6
Kans.	18	651	306	289	9.8
S. Atlantic					
Del.	7	785	198	190	8.1
Md.	26	1,586	1,467	952	12.7
D.C.	2	1,269	1,235	1,235	19.0
Va.	5	687	611	587	15.1
W. Va.	7	269	240	178	11.7
N.C.	93	923	711	461	18.3
S.C.	46	1,131	1.064	990	12.0
Ga.	10	525	554	161	12.3
Fla.	23	6,074	5,811	3,756	21.4
E.S. Central	20	0,074	5,011	3,750	21.4
	-	44.7	450	400	47.4
(y.	5	417	152	132	17.1
Fenn.	5	946	684	513	13.0
Ala.	5	564	518	70	16.4
Miss.	14	150	143	0	18.2
N.S. Central					
Ark.	1	120	106	93	17.9
.a .	7	1,644	921	695	23.3
Okla.	7	711	691	595	21.4
Tex.	27	8,773	7,564	5,379	12.5
Nountain		•	·	- • -	-
Aont.	7	170	177	168	6.8
daho	1	137	380	108	0.8 8.4
	1		380		
Nyo.	10	0		1	0.0
Colo.		4,252	4,252	4,252	41.5
I. Mex.	6	434	243	170	17.3
Ariz.	1	662	427	427	20.1
A				140	20.4
Jtah Jev.	21 3	216 984	416 458	148 63	39.4 13.8

HTLV-III/LAV - Continued

Area	Testing sites	Pretest sessions	Persons tested	Post-test sessions	Percent positive*
Pacific					
Wash.	18	3,136	2,569	2,330	12.7
Oreg.	15	829	1,435	829	21.2
Calif.	51	17,721	17,546	11,552	13.7
San Fran.	2	5,898	5,898	5,047	19.9
Alaska	5	824	915	77	9.3
Hawaii	1	793	658	599	17.3
Guam	0	0	0	0	_
P.R.	2	595	904	351	40.7
V.I.	0	0	0	0	_
Pac. Trust Terr.	0	0	0	0	_

TABLE 5. Alternate testing site activities — United States, 1985 (Continued)

*On at least two ELISA tests.

Reported by Div of Sexually Transmitted Diseases, Center for Prevention Svcs, CDC.

Editorial Note: Many of the project areas reported they had underestimated the difficulty of establishing alternate sites on a short-term basis. Start-up delays were common because of administrative procedures and such factors as general hiring freezes and the development of systems to assure strict confidentiality of all records related to counseling and clinical laboratory test results. Moreover, the initial demand for services was less than most areas had anticipated. The number of tests performed in each area depended on many factors, including accessibility of services, perception of the benefits or risks of testing, and awareness of the existence of services by those at risk. The utilization of the sites varied widely in both high and low AIDS-incidence areas (Table 6), perhaps indicating that demand for testing depends on the degree to which it is encouraged and made accessible for persons at risk. In one project area with a high test-to-case ratio, testing was actively promoted by both public health authorities and AIDS risk-group representatives (1).

The goal of protecting the blood supply by providing alternate sites at which persons could be tested was achieved. In addition, experience with the HTLV-III/LAV ELISA tests since licensure in March 1985 has shown them to be remarkably sensitive and specific (2) and to be useful, not only for preventive purposes, but also for the diagnosis and differential diagnosis of clinical illness. An evaluation of the tests used to screen blood donors in a large metropolitan area showed a specificity of 99.8% (3). Thus, they have value in identifying individuals who are infected, and who are likely to be able to transmit the infection to others by the established routes of transmission, even if such individuals themselves are asymptomatic.

Accordingly, the U.S. Public Health Service has proposed additional applications to prevent perinatal transmission (4) and to help reduce drug abuse-related and sexual transmission of HTLV-III/LAV virus by infected persons (5). The main purpose of the additional applications is to facilitate identification of seropositive asymptomatic persons, both for medical evaluation and for counseling to prevent transmission. Reduction of sexual and drug-related transmission of HTLV-III/LAV should be enhanced by using available serologic tests to give asymptomatic, infected individuals in high-risk groups the opportunity to know their status so they can take appropriate steps to prevent further transmission (6).

The wide network of alternate testing sites that has been established by state and local health departments, frequently in cooperation with local community groups, may facilitate extension of testing services to selected populations at increased risk for HTLV-III/LAV infection. *References*

1. Judson FN. Personal communication.

HTLV-III/LAV - Continued

- CDC. Update: Public Health Service Workshop on Human T-Lymphotropic Virus Type III Antibody Testing—United States. MMWR 1985;34:477-8.
- 3. Ward JW, Grindon AJ, Fiorino PM. Laboratory and epidemiological evaluation of an enzyme immunoassay for antibodies to human T-lymphotropic virus, type III. JAMA (in press).
- CDC. Recommendations for assisting in the prevention of perinatal transmission of human T-lymphotropic virus type III/lymphadenopathy-associated virus and acquired immunodeficiency syndrome. MMWR 1985;34:721-6, 731-2.
- 5. CDC. Additional recommendations to reduce sexual and drug abuse-related transmission of human T-lymphotropic virus type III/lymphadenopathy-associated virus. MMWR 1986;35:152-5.
- 6. Handsfield HH, Dunphy CA, Bonin P. Unpublished data.

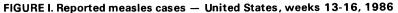
Project area	Reported AIDS cases [†]	HTLV-III/LAV tests	Tests per case	
New York City	2,140	2,032	1.0	
California	1,923	23,444	12.2	
Florida	516	5,811	11.3	
Texas	483	7,564	15.7	
New Jersey	460	1,818	4.0	
Colorado	61	4,252	69.7	
Ohio	53	2,780	52.5	
Oregon	33	1,435	43.5	
lowa	13	947	72.9	
Alaska	5	915	183.0	
United States	8,072	79,083	9.8	

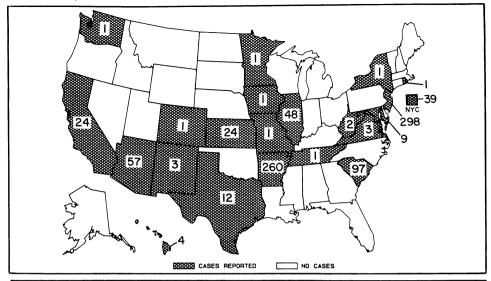
TABLE 6. Reported AIDS cases and tests for HTLV-III/LAV antibody performed at alternate sites, for 10 project areas — United States, 1985*

*This table shows five project areas with the highest number of reported cases and five project areas with the highest rates of tests per case.

[†]Provisional totals reported to MMWR through week 52, 1985.

[§]Includes the separately funded San Francisco project area.





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
 Editor

 James O. Mason, M.D., Dr.P.H.
 Michael B. Gregg, M.D.

 Director, Epidemiology Program Office
 Assistant Editor

 Carl W. Tyler, Jr., M.D.
 Karen L. Foster, M.A.

\$U.S. Government Printing Office: 1986-746-149/21053 Region IV

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 DR VERNE F NEWHOUSE VIROLOGY DIVISION CID 7-B14

X