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Grayson B. Miller, Jr., M.D., Epidemiologist *Editor: Carl W. Armstrong, M.D.*

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Meningococcal Vaccines

Editor's note: Two recent outbreaks of meningococcal disease in Virginia, one in Rockbridge County due to serogroup C and the other in Staunton due to serogroup B, have prompted a number of questions regarding the use of meningococcal vaccines. Reprinted below are the most recent recommendations of the Immunization Practices Advisory Committee (ACIP) of the U.S. Public Health Service.

Introduction

A polysaccharide vaccine against disease caused by *Neisseria meningitidis* serogroups A, C, Y, and W-135 is currently licensed in the United States. This statement updates the previous statement (*MMWR* 1978;27:327-9), summarizes available information on the vaccine, and offers guidelines for its use in the civilian population of the United States.

Meningococcal Disease

N. meningitidis causes both endemic and epidemic disease, principally meningitis and meningococcemia. It is the second most common cause of bacterial meningitis in the United States (approximately 20% of all cases), affecting an estimated 3,000-4,000 people each year. The case-fatality rate is approximately 10% for meningococcal meningitis and 20% for meningococcemia, despite therapy with antimicrobial agents, such as penicillin, to which all strains remain highly sensitive.

No major epidemic of meningococcal disease has occurred in the United States since 1946, although localized community outbreaks have been reported. The incidence of endemic meningococcal disease peaks in the late winter to early spring. Attack rates are highest among children aged 6-12 months and then steadily decline; by age 5 years, the incidence approximates that for adults. Serogroup B, for which a vaccine is not yet available, accounts for 50%-55% of all cases; serogroup C, for 20%-25%; and serogroup W-135, for 15%. Serogroups Y (10%) and A (1%-2%) account for nearly all remaining cases. Serogroup W-135 has emerged as a major cause of disease only since 1975 (*1*). While serogroup A causes only a small proportion of endemic disease in the United States, it is the most common cause of epidemics elsewhere. Less commonly, serogroups C and B can also cause epidemic disease.

People with certain chronic conditions appear to be at increased risk of developing meningococcal infection. Meningococcal disease is particularly common among individuals with component deficiencies in the final common complement pathway (C3, C5-C9), many of whom experience multiple episodes of infection (*2*). Asplenic persons seem also to be at increased risk of developing meningococcal disease and experience particularly severe infections (*3*). It is uncertain whether individuals with other diseases associated with immunosuppression are at higher risk of acquiring meningococcal disease, as

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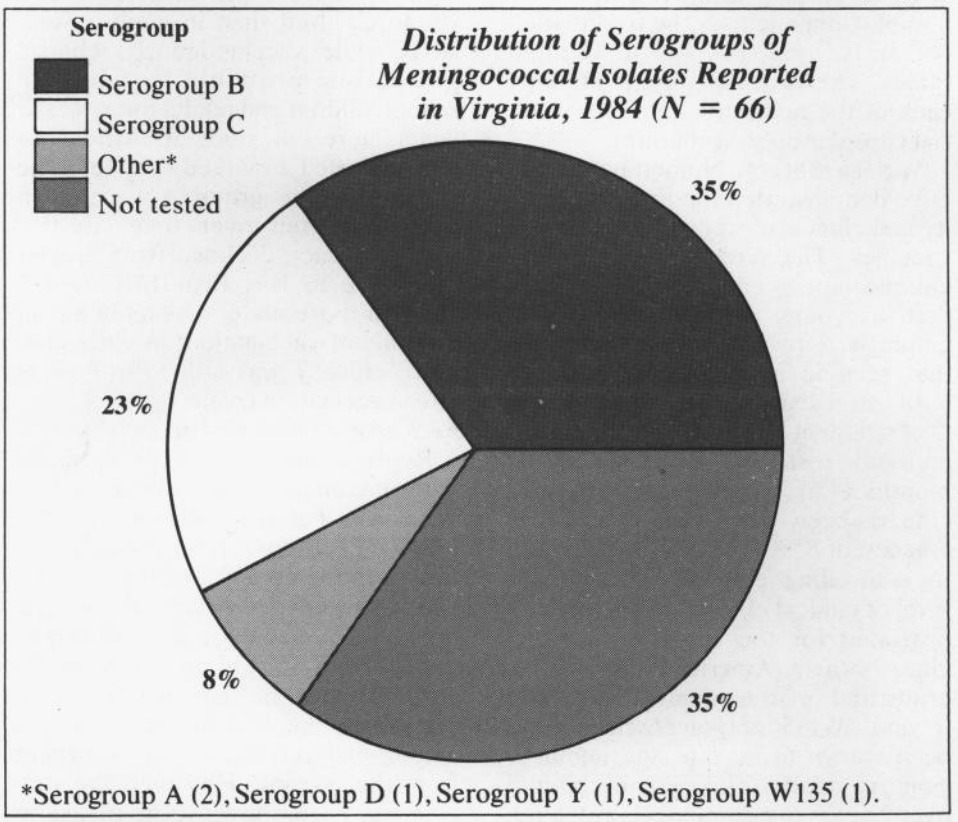

*Serogroup A (2), Serogroup D (1), Serogroup Y (1), Serogroup W135 (1).

they are for disease caused by other encapsulated bacteria. In the past, new military recruits were at especially high risk, particularly for serogroup C disease; however, since routine vaccination of recruits with the bivalent A/C vaccine began in 1971, disease caused by those serogroups has been uncommon. Military recruits currently receive the A,C,Y,W-135 vaccine.

Meningococcal Polysaccharide Vaccines

The recently licensed quadrivalent A,C,Y,W-135 vaccine (Menomune®—A/C/Y/W-135, manufactured by Squibb-Connaught) is the formulation currently available in the United States. The vaccine consists of 50 μg each of the respective purified bacterial capsular polysaccharides.

Vaccine efficacy. Numerous studies have demonstrated the immunogenicity and clinical efficacy of the A and C vaccines. The serogroup A polysaccharide induces antibody in some children as young as 3 months of age, although a response comparable to that seen in adults is not achieved until 4 or 5 years of age; the serogroup C component does not induce a good antibody response before age 18-24 months (*4,5*). The serogroup A vaccine has been shown to have a clinical efficacy of 85%-95% and to be of use in controlling epidemics. A similar level of clinical efficacy has been demonstrated for the serogroup C vaccine, both in American military recruits and in an epidemic. The group Y and W-135 polysaccharides have been shown to be safe and immunogenic in adults (*6-9*) and in children over 2 years of age; clinical protection has not been demonstrated directly, but is assumed, based on the production of bactericidal antibody, which for group C has been correlated with clinical protection. The antibody responses to each of the four polysaccharides in the quadrivalent vaccine are serogroup-specific and independent.

Duration of efficacy. Antibodies against the group A and C polysaccharides decline markedly over the first 3 years following a single dose of vaccine (*5,10-13*). This antibody decline is more rapid in infants and young children than in adults. Similarly, while vaccine-induced clinical protection probably persists in schoolchildren and adults for at least 3 years, a recent study in Africa has demonstrated a marked decline in the efficacy of the group A vaccine in young children over time. In this study, efficacy declined from greater than 90% to less than 10% over 3 years in those under 4 years of age at the time of vaccination; in older children, efficacy was still 67% 3 years after vaccination (*14*).

Recommendations for Vaccine Use

Routine vaccination of civilians with meningococcal polysaccharide vaccine is not recommended for the following reasons: (1) the risk of infection in the United States is low; (2) a vaccine against serogroup B, the major cause of meningococcal disease in the United States, is not yet available; and (3) much of the meningococcal disease in the United States occurs among children too young to benefit from the vaccine. However, the vaccine has been shown to be of use in aborting outbreaks due to serogroups represented in the vaccine and should be used in their control. In an outbreak, the serogroup should be determined and the population at risk delineated by neighborhood, school, dormitory, or other reasonable boundary. Although endemic disease is very uncommon above age 5 years, older children, adolescents, and young adults constitute a higher proportion of cases during epidemics and may warrant vaccination during an outbreak (*15*).

Routine immunization with the quadrivalent vaccine is recommended for particular high-risk groups, including individuals with terminal complement component deficiencies and those with anatomic or functional asplenia. Persons splenectomized because of trauma or nonlymphoid tumors and those with inherited complement deficiencies have acceptable antibody responses to meningococcal vaccine, although clinical efficacy has not been documented (*2,16*). It should be recognized that such individuals frequently have preexisting antibody against *N. meningitidis* and may not be protected by vaccination.

Vaccination with the A-C vaccine may benefit some travelers to countries recognized as having hyperendemic or epidemic disease and Americans living in these areas, particularly those who will have prolonged contact with the local populace. One area of the world recognized as having recurrent epidemics of meningococcal disease is the part of sub-Saharan Africa known as the "meningitis belt," which extends from Mauritania in the west to Ethiopia in the east. Epidemics have been recognized in other parts of the world, and updated information can be obtained from travelers' clinics, state health departments, and CDC.

Primary Immunization. For both adults and children, vaccine is administered subcutaneously as a single 0.5-ml dose. The vaccine can be given at the same time as other immunizations, if needed. Good antibody levels are achieved within 10-14 days after vaccination.

Precautions and Contraindications

Reactions. Adverse reactions to meningococcal vaccine are mild and infrequent, consisting principally of localized erythema lasting 1-2 days. Up to 2% of young children develop fever transiently after vaccination (*13*). *(Continued to page 3)*

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Pregnancy. On theoretical grounds, it is prudent not to immunize pregnant women unless there is a substantial risk of infection. However, evaluation of the vaccine in pregnant women during an epidemic in Brazil demonstrated no adverse effects. Further, antibody studies in these women showed good antibody levels in maternal and cord blood following vaccination during any trimester; antibody levels in the infants declined over the first few months and did not affect their subsequent response to immunization (*17*).

Revaccination

Revaccination may be indicated for individuals at high risk of infection, particularly children who were first immunized under 4 years of age; such children should be considered for revaccination after 2 or 3 years if they remain at high risk. The need for revaccination in older children and adults remains unknown.

Prospects for Future Meningococcal Vaccines

Work is continuing on a serogroup B meningococcal vaccine, as well as on improved A and C vaccines. Candidate vaccines include capsular polysaccharides complexed with meningococcal outer-membrane proteins or covalently linked to carrier proteins. Clinical efficacy data for these vaccines are not available.

Antimicrobial Chemoprophylaxis

Antimicrobial chemoprophylaxis of intimate contacts remains the chief preventive measure in sporadic cases of *N. meningitidis* disease in the United States. Intimate contacts include (1) household members, (2)day-care-center contacts, and (3) anyone directly exposed to the patient's oral secretions, such as through mouth-to-mouth resuscitation or kissing. The attack rate for household contacts is 0.3%-1%, 300-1,000 times the rate in the general population.

Unless the causative organism is known to be sensitive to sulfadiazine, the drug of choice is rifampin, given twice daily for 2 days (600 mg every 12 hours to adults; 10 mg/kg every 12 hours to children 1 month of age or older; 5 mg/kg every 12 hours to children under 1 month of age). Rifampin has been shown to be 90% effective in eradicating nasopharyngeal carriage. No serious adverse effects have been noted. However, rifampin prophylaxis is not recommended for pregnant women, as the drug is teratogenic in laboratory animals. Also, as well as turning urine orange, rifampin is excreted in tears, resulting in staining of contact lenses; thus, they should not be used during the course of therapy.

Because systemic antimicrobial therapy of meningococcal disease does not reliably eradicate nasopharyngeal carriage of *N. meningiditis*, it is also important to give chemoprophylaxis to the index patient before discharge from the hospital (*18*).

Nasopharyngeal cultures are not helpful in determining who warrants chemoprophylaxis and unnecessarily delay institution of this preventive measure.

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Additional Recommendations to Reduce Sexual And Drug Abuse-Related Transmission of HTLV-III/LAV

Background

Human T-lymphotropic virus type III/lymphadenopathy-associated virus (HTLV-III/LAV), the virus that causes acquired immunodeficiency syndrome (AIDS), is transmitted through sexual contact, parenteral exposure to infected blood or blood components, and perinatally from mother to fetus or neonate. In the United States, over 73% of adult AIDS patients are homosexual or bisexual men; 11% of these males also had a history of intravenous (IV) drug abuse. Seventeen percent of all adult AIDS patients were heterosexual men or women who abused IV drugs (*1,2*). The prevalence of HTLV-III/LAV antibody is high in certain risk groups in the United States (*3,4*).

Since a large proportion of seropositive asymptomatic persons have been shown to be viremic (*5*), all seropositive individuals, whether symptomatic or not, must be presumed capable of transmitting this infection. A repeatedly reactive serologic test for HTLV-III/LAV has important medical, as well as public health, implications for the individual and his/her health-care provider. The purpose of these recommendations is to suggest ways to facilitate identification of seropositive asymptomatic persons, both for medical evaluation and for counseling to prevent transmission.

Previous U.S. Public Health Service recommendations pertaining to sexual, IV drug abuse, and perinatal transmission of HTLV-III/LAV have been published (*6-8*). Reduction of sexual and IV 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 the further transmission of this virus.

Since the objective of these additional recommendations is to help interrupt transmission by encouraging testing and counseling among persons in high-risk groups, careful attention must be paid to maintaining confidentiality and to protecting records from any unauthorized disclosure. The ability of health departments to assure confidentiality—and the public confidence in that ability—are crucial to efforts to increase the number of persons requesting such testing and counseling. Without appropriate confidentiality protection, anonymous testing should be considered. Persons tested anonymously would still be offered medical evaluation and counseling.

Persons at Increased Risk of HTLV-III/LAV Infection

Persons at increased risk of HTLV-III/LAV infection include: (1) homosexual and bisexual men; (2) present or past IV drug abusers; (3) persons with clinical or laboratory evidence of infection, such as those with signs or symptoms compatible with AIDS or AIDS-related complex (ARC); (4) persons born in countries where heterosexual transmission is thought to play a major role*; (5) male or female prostitutes and their sex partners; (6) sex partners of infected persons or persons at increased risk; (7) all persons with hemophilia who have received clotting-factor products; and (8) newborn infants of high-risk or infected mothers.

Recommendations

1. Community health education programs should be aimed at members of high-risk groups to: (a) increase knowledge of AIDS; (b) facilitate behavioral changes to reduce risks of HTLV-III/LAV infection; and (c) encourage voluntary testing and counseling.
2. Counseling and voluntary serologic testing for HTLV-III/LAV should be routinely offered to all persons at increased risk when they present to health-care settings. Such facilities include, but are not limited to, sexually transmitted disease clinics, clinics for treating parenteral drug abusers, and clinics for examining prostitutes.
   a. Persons with a repeatedly reactive test result (see section on Test Interpretation) should receive a thorough medical evaluation, which may include history, physical examination, and appropriate laboratory studies.

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b. High-risk persons with a negative test result should be counseled to reduce their risk of becoming infected by:
   (1) Reducing the number of sex partners. A stable, mutually monogamous relationship with an uninfected person eliminates any new risk of sexually transmitted HTLV-III/LAV infection.
   (2) Protecting themselves during sexual activity with any possibly infected person by taking appropriate precautions to prevent contact with the person's blood, semen, urine, feces, saliva, cervical secretions, or vaginal secretions. Although the efficacy of condoms in preventing infections with HTLV-III/LAV is still under study, consistent use of condoms should reduce transmission of HTLV-III/LAV by preventing exposure to semen and infected lymphocytes (*9,10*).
   (3) For IV drug abusers, enrolling or continuing in programs to eliminate abuse of IV substances. Needles, other apparatus, and drugs must never be shared.

c. Infected persons should be counseled to prevent the further transmission of HTLV-III/LAV by:
   (1) informing prospective sex partners of his/her infection with HTLV-III/LAV, so they can take appropriate precautions. Clearly, abstention from sexual activity with another person is one option that would eliminate any risk of sexually transmitted HTLV-III/LAV infection.
   (2) Protecting a partner during any sexual activity by taking appropriate precautions to prevent that individual from coming into contact with the infected person's blood, semen, urine, feces, saliva, cervical secretions, or vaginal secretions. Although the efficacy of using condoms to prevent infections with HTLV-III/LAV is still under study, consistent use of condoms should reduce transmission of HTLV-III/LAV by preventing exposure to semen and infected lymphocytes (*9,10*).
   (3) Informing previous sex partners and any persons with whom needles were shared of their potential exposure to HTLV-III/LAV and encouraging them to seek counseling/testing.
   (4) For IV drug abusers, enrolling or continuing in programs to eliminate abuse of IV substances. Needles, other apparatus, and drugs must never be shared.
   (5) Not sharing toothbrushes, razors, or other items that could become contaminated with blood.
   (6) Refraining from donating blood, plasma, body organs, other tissue, or semen.
   (7) Avoiding pregnancy until more is known about the risks of transmitting HTLV-III/LAV from mother to fetus or newborn (*8*).
   (8) Cleaning and disinfecting surfaces on which blood or other body fluids have spilled, in accordance with previous recommendations (*2*).
   (9) Informing physicians, dentists, and other appropriate health professionals of his/her antibody status when seeking medical care so that the patient can be appropriately evaluated.

3. Infected patients should be encouraged to refer sex partners or persons with whom they have shared needles to their health-care provider for evaluation and/or testing. If patients prefer, trained health department professionals should be made available to assist in notifying their partners and counseling them regarding evaluation and/or testing.
4. Persons with a negative test result should be counseled regarding their need for continued evaluation to monitor their infection status if they continue high-risk behavior (*8*).
5. State and local health officials should evaluate the implications of requiring the reporting of repeatedly reactive HTLV-III/LAV antibody test results to the state health department.
6. State or local action is appropriate on public health grounds to regulate or close establishments where there is evidence that they facilitate high-risk behaviors, such as anonymous sexual contacts and/or intercourse with multiple partners or IV drug abuse (e.g., bathhouses, houses of prostitution, "shooting galleries").

Test Interpretation

Commercially available tests to detect antibody to HTLV-III/LAV are enzyme-linked immunosorbant assays (ELISAs) using antigens derived from disrupted HTLV-III/LAV. When the ELISA is reactive on initial testing, it is standard procedure to repeat the test on the same specimen. Repeatedly reactive tests are highly sensitive and specific for HTLV-III/LAV antibody. However, since falsely positive tests occur, and the implications of a positive test are serious, additional more specific tests (e.g., Western blot, immunofluorescent assay, etc.) are recommended following repeatedly reactive ELISA results, especially in low-prevalence populations. If additional more specific test results are not readily available, persons in high-risk groups with strong repeatedly reactive ELISA results can be counseled before any additional test results are received regarding their probable infection status, their need for medical follow-up, and ways to reduce further transmission of HTLV-III/LAV.

Other Considerations

State or local policies governing informing and counseling sex partners and those who share needles with persons who are HTLV-III/LAV-antibody positive will vary, depending on state and local statutes that authorize such actions. Accomplishing the objective of interrupting transmission by encouraging testing and counseling among persons in high-risk groups will depend heavily on health officials paying careful attention to maintaining confidentiality and protecting records from unauthorized disclosure.

The public health effectiveness of various approaches to counseling, sex-partner referral, and laboratory testing will require careful monitoring. The feasibility and efficacy of each of these measures should be evaluated by state and local health departments to best utilize available resources.

**e.g., Haiti, Central African countries.*

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Delta Virus

Originally considered an antigenic variant of hepatitis B virus (HBV) (1), delta is a distinct transmissible hepatotropic virus. It is considered defective in that its ability to infect and cause hepatitis is dependent on presence of *active* HBV infection. The delta virus circulates as a 35- to 37-nm size virus particle containing an internal protein antigen, the delta antigen. This antigen is associated with single-stranded RNA of uniquely low molecular weight and is coated with hepatitis B surface antigen (HBsAg) (2).

Delta infection can occur together with HBV (co-infection) or as acute hepatitis superimposed on the chronic HBV carrier state (superinfection) (3). Co-infection with HBV/delta usually causes acute hepatitis, which is most often clinically indistinguishable from that with HBV infection alone. It may have a biphasic course, with the second liver enzyme elevation representing delta infection (4). Co-infection appears to be associated with a higher rate of fulminant hepatitis (5) and does not increase the subsequent risk of becoming a chronic HBV carrier. The incubation period of HBV/delta co-infection ranges from 4-20 weeks in chimpanzees. Clinically, superinfection of an HBV carrier ranges from asymptomatic liver enzyme elevation and seroconversion, to fulminant hepatitis (5). The incubation period ranges from 3-6 weeks in chimpanzees. Superinfection frequently results in the establishment of persistent delta infection and constitutes the reservoir for the virus. Chronic HBV/delta infection is associated with the development of chronic active hepatitis and cirrhosis (6).

The diagnosis of delta infection is made on the basis of detection of delta antigen in serum during early infection, immunofluorescent staining of delta antigen in liver, or the appearance of delta antibodies during or af-

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ter infection. Testing for delta infection is currently indicated in fulminant HBV infection or in the case of acute non-A hepatitis occurring in a known HBV carrier.

The antibody response to delta infection has both IgM and IgG components typical of most viral infections. IgM anti-delta antibody appears early during acute delta infection and may persist for years when chronic infection ensues. IgG anti-delta antibody usually appears later during acute delta infection; it may only persist for a few months thereafter in self-limited delta co-infection. Chronic delta hepatitis is usually associated with high titers of IgG anti-delta. Tests for anti-delta antibody and delta antigen are becoming available commercially.

Epidemiology of Delta Infection

Delta hepatitis was originally discovered in Italy and has been demonstrated to be endemic in southern Italy. Delta infection in this region is often unrelated to overt blood contact, suggesting sexual or other inapparent percutaneous modes of transmission (7) similar to transmission of hepatitis B. Seroprevalence studies in areas with low HBV endemnicity (HBV carrier rate <1.0%) have demonstrated delta antibodies in 0%-10% of HBsAg carriers in Europe, the United States, and Australia with seropositivity found primarily in parenteral drug users and hemophiliacs (8). For unknown reasons, delta has as yet been found in only a few homosexual men with HBV infection. Evidence of delta infection has also been found in up to 30%-50% of persons with fulminant hepatitis B. In the United States, co-infection with HBV/delta virus has been recently associated with fulminant hepatitis in parenteral drug abusers (9,10).

Recent studies in areas with moderate HBV endemicity have documented the presence of endemic delta infection in eastern Europe, the western Mediterranean, and the Middle East. In the developing world, where HBV is highly endemic, recent serotesting has documented the presence of delta virus infection in parts of West Africa, the South Pacific, and South America. As yet, there is little evidence of delta infection in the parts of Asia (China, Taiwan, Japan, Burma) where testing has been done. The severe nature of this disease is evidenced by a recent epidemic of

hepatitis due to delta superinfection of HBV carriers in a rural Venezuelan native Indian population (11). The epidemic was associated with a high mortality rate (17%) and frequent development of chronic liver disease. In parts of the Amazon Basin, delta infection appears to be highly endemic and has probably been present for many years. Nevertheless, in the developing world, where HBV is endemic, certain populations may be at risk for severe morbidity and mortality with introduction of delta virus.

Prevention of Delta Infection

Since the delta virus depends on hepatitis B for replication, prevention of hepatitis B infection, either preexposure or postexposure, will suffice to prevent delta infection in a person susceptible to hepatitis B. Known perinatal, sexual, or percutaneous exposure of HBV-susceptible persons to sera or to persons positive for both HBV and delta virus, should be treated exactly as such exposures to hepatitis B alone (12). Persons who are HBsAg carriers are at risk of delta infection, especially if they participate in activities that put them at high risk of repeated exposure to hepatitis B (parenteral drug abuse, homosexuality). However, at present there are no products available that might prevent delta infection in HBsAg carriers either before or after exposure. Counseling on the necessity of avoiding such exposures appears to be the only preventive measure currently available.

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Cases of selected notifiable diseases, Virginia, for the period April 1 through April 30, 1986

Disease	State				Regions					
	This Month	Last Month	Total to Date 1986	Total to Date 1985	Mean 5 Year To Date	This Month N.W.	This Month N.	This Month S.W.	This Month C.	This Month E.
Measles	4	0	4	12	9	0	3	1	0	0
Mumps	6	4	15	16	25	0	2	2	1	1
Pertussis	0	3	9	3	8	0	0	0	0	0
Rubella	0	0	0	0	2	0	0	0	0	0
Meningitis—Aseptic	13	4	50	60	42	1	3	2	4	3
*Bacterial	16	36	94	110	96	0	5	1	3	7
Hepatitis A (Infectious)	3	26	43	80	59	1	0	1	0	1
B (Serum)	28	62	144	179	166	5	7	5	5	6
Non-A, Non-B	4	8	22	35	30	1	0	1	2	0
Salmonellosis	70	88	290	369	316	9	9	13	18	21
Shigellosis	6	4	19	23	135	2	1	0	1	2
Campylobacter Infections	34	23	110	147	95	10	6	4	9	5
Tuberculosis	41	43	120	105	136	4	11	8	8	10
Syphilis (Primary & Secondary)	20	45	147	111	178	0	5	2	4	9
Gonorrhea	1331	1662	5764	5867	6299	—	—	—	—	—
Rocky Mountain Spotted Fever	0	1	1	0	1	0	0	0	0	0
Rabies in Animals	23	35	73	64	116	16	7	0	0	0
Meningococcal Infections	5	27	40	31	31	0	1	1	0	3
Influenza	184	1555	3449	896	1520	6	0	130	12	36
Toxic Shock Syndrome	1	3	7	0	2	1	0	0	0	0
Reyes Syndrome	0	0	0	1	4	0	0	0	0	0
Legionellosis	1	0	4	7	7	0	1	0	0	0
Kawasaki's Disease	3	3	10	15	10	0	0	0	1	2
Other: Acquired Immunodeficiency Syndrome	13	9	67	18	—	1	6	1	4	1

Counties Reporting Animal Rabies: Augusta 1 cat; Bath 1 raccoon; Caroline 3 raccoons; Clarke 1 raccoon; Frederick 1 skunk; King George 1 raccoon; Rockingham 2 raccoons; Shenandoah 2 raccoons; Spotsylvania 1 skunk; Warren 3 raccoons; Fairfax 6 raccoons; Loudoun 1 raccoon.

Occupational Illnesses: Pneumoconioses 29; Asbestosis 9; Silicosis 9; Carpal tunnel syndrome 6; Hearing loss 3; Dermatitis 2.

*other than meningococcal

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