VIRGINIA
EPIDEMIOLOGY BULLETIN

Robert B. Stroube, M.D., M.P.H., Health Commissioner
Carl W. Armstrong, M.D., State Epidemiologist
Christopher Novak, M.D., M.P.H., Editor
Vickie L. O'Dell, Layout Editor

June 2005 Volume 105, No. 6

Meningococcal Disease and Vaccine

Introduction

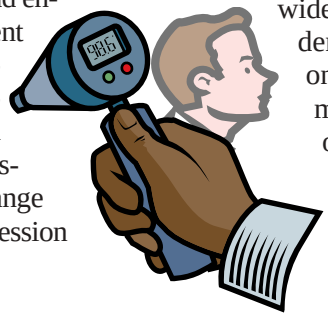
In January 2005, a tetravalent meningococcal polysaccharide-protein conjugate vaccine (MCV4) was licensed by the Food and Drug Administration for use among persons 11-55 years of age. This article reviews meningococcal disease and updates healthcare providers in Virginia on the current meningococcal vaccination recommendations.

Neisseria meningitidis

Neisseria meningitidis, or meningococcus, is an aerobic gram-negative diplococcus. Meningococci are classified using serological methods based on antigens to the organisms' polysaccharide capsule. Thirteen antigenically and chemically distinct polysaccharide capsules have been described. Almost all invasive meningococcal disease is caused by one of five serogroups: A, B, C, Y, and W-135. The relative importance of each serogroup depends on geographic location, as well as other factors such as age. For instance, serogroup A is a major cause of disease in sub-Saharan Africa but is rarely isolated in the United States. In addition, some strains, often those found to cause asymptomatic nasopharyngeal carriage, are not groupable and do not have a capsule.

In This Issue:

Meningococci are transmitted by droplet aerosol or direct contact with secretions from the nose or throat of colonized or infected persons. The bacteria attach to and multiply on the mucosal cells of the nasopharynx. In a small proportion (<1%) of colonized persons the organism penetrates the mucosal cells and enters the bloodstream (a recent upper respiratory tract infection may be a contributing factor). The incubation period of meningococcal disease is 3-4 days, with a range of 2-10 days. Disease progression can be extremely rapid.



Clinical Features

Meningitis is the most common presentation of invasive meningococcal disease (49% of cases). Meningeal infection is similar to other forms of acute purulent meningitis, characterized by a sudden onset of fever, headache, and stiff neck often with nausea, vomiting, photophobia, and altered mental status.

Meningococcal sepsis (meningococcemia) occurs without meningitis in 33% of invasive meningococcal infections. This condition is characterized by an abrupt onset of fever and a petechial or purpuric rash, often associated with hypotension, shock, acute adrenal hemorrhage, and multiorgan failure. Less common presentations of meningococcal disease include pneumonia (9% of cases), arthritis (2%), otitis media (1%), and epiglottitis (<1%).

The case-fatality rate for invasive meningococcal disease is 10%-14%, even with appropriate antibiotic therapy. The case-fatality rate of meningococcemia may reach 40%. Up to 20% of survivors have permanent sequelae such as hearing loss, neurologic deficits or loss of a limb.

Occurrence

Meningococcal disease occurs worldwide in both endemic and epidemic form. Humans are the only natural reservoir of the meningococcus, and up to 10% of adolescents and adults are asymptomatic transient carriers of mostly non-pathogenic *N. meningitidis*.

Risk factors for the development of invasive meningococcal disease include antecedent viral infection, household crowding, deficiencies in the terminal common complement pathway, functional or anatomic asplenia, and both active and passive smoking. Family members of a person with meningococcal disease are at an increased risk of infection. In the United States, blacks and persons of low socioeconomic status have been consistently at higher risk although race and socioeconomic status are likely markers for other factors (e.g., household crowding). Persons with HIV infection are probably at an increased risk. Cases of invasive meningococcal disease, including at least two fatal cases, have been reported among microbiologists working with *N. meningitidis* isolates. In addition, recent studies have shown that college freshmen living in dormitories are at a moderately increased risk of acquiring meningococcal disease. However, overall, U.S. college students are not at a higher risk for meningococcal disease than other people

of similar age. During outbreaks, bar or nightclub patronage and alcohol use have been associated with higher risk for meningococcal disease.

Trends in the United States

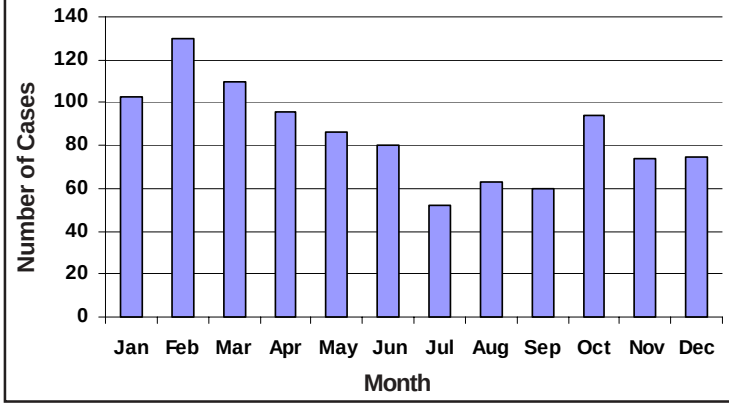
Approximately 2,500 to 3,000 cases of meningococcal disease are reported each year in the United States (0.8-1.3 cases per 100,000 population). In 2002, an estimated 150 deaths due to meningococcal disease occurred in the U. S. In Virginia, 24 cases (including two deaths) were reported in 2004, the lowest number of cases in over 15 years and 45% below the five-year mean of 44 cases per year. While meningococcal infections can occur throughout the year, the incidence is highest in the late winter (peak: December/ January) and early spring (Figure 1).

Nationwide, infants (persons less than 12 months of age) have the highest risk of disease from *N. meningitidis***.** Incidence of disease declines in early childhood, increases during adolescence and early adulthood, then declines among older adults (Figure 2). The proportion of cases among adolescents and young adults has increased in recent years. During 1992-1998, 28% of reported cases were in persons 12-29 years of age.

The proportion of disease caused by different serogroups has also changed during the last 15 years. From 1988 to 1991, most cases of meningococcal disease in the United States were due to either serogroup B or C; serogroup Y accounted for only 2% of cases. Currently, serogroups B, C, and Y each cause approximately one-third of cases. In addition, the proportion of cases caused by each serogroup varies by age group. Among infants, more than half of meningococcal infections are caused by serogroup B, for which no vaccine is available in the U.S. However, 75% of all cases of meningococcal disease among persons 11 years of age or older are caused by serogroups C, Y, or W-135 (all of which are included in both available vaccines).

Large outbreaks of serogroup A meningococcal disease occur in the African "meningitis belt", an area that extends from Ethiopia to Senegal. Rates of endemic meningococcal disease in this area are several times higher than in industrialized countries. In addition, outbreaks occur every 8-12 years with attack rates of 500-1,000 cases per 100,000 population. In the United States, meningococcal outbreaks account for less than two percent of reported cases (i.e., more than 98% of cases are sporadic), although the frequency of localized outbreaks has increased since 1991.

Figure 1. Invasive Meningococcal Infections, by Month of Onset, Virginia, 1986-2003



Meningococcal Vaccines

Meningococcal Polysaccharide Vaccine (MPSV4)

The tetravalent A, C, Y, W-135 polysaccharide vaccine (Menomune™, Sanofi Pasteur, Inc., Swiftwater, PA) was licensed in 1981 and until recently was the only formulation available in the United States. Each dose consists of 50 µg of each of the four purified bacterial capsular polysaccharides. The vaccine contains lactose as a stabilizer and is available in single- and 10-dose vials (50-dose vials are no longer available). Diluent for the single dose vial is sterile water without preservative. Diluent for the 10-dose vial is sterile water with thimerosal as a preservative.

The characteristics of MPSV4 are similar to other polysaccharide vaccines (e.g., pneumococcal polysaccharide vaccine). Since bacterial polysaccharides, including those in the capsule of *N. meningitidis*, are T-cell-independent antigens, they stimulate mature B-lymphocytes but not T-lymphocytes. This leads to relatively short-term protection, does not induce an anamnestic response, does not cause the sustainable reduction of nasopharyngeal carriage of *N. meningitidis* that would create 'herd immunity', and cannot be boosted with subsequent vaccinations. In addition, the response to the vaccine is age-dependent, resulting in poor immunogenicity in children less than two years of age. Overall, this limits the effectiveness of polysaccharide vaccines.

Following vaccination with MPSV4, a protective level of antibody is usually achieved within 7-10 days . Among infants and children less than five years of age, measurable levels of antibodies against serogroups A and C polysaccharides decrease substantially during the first three years following a single dose of vaccine. In healthy adults, antibody levels also decrease, but antibodies are detectable as long as 10 years after vaccination. Although vaccine-induced protection likely persists in school-aged children and adults for at least three years, the efficacy of the serogroup A vaccine in children less than five years of age may decrease markedly. In one study, efficacy declined from greater than 90% to less than 10% three years after vaccination among children who were less than four years of age when vaccinated.

Improved vaccine efficacy has not been demonstrated among persons who receive multiple doses of MPSV4. In fact, recent serologic studies have reported that multiple doses of serogroups A and C polysaccharide vaccine might cause immunologic hyporesponsiveness (i.e., a reduced antibody response after subsequent challenge with the same polysaccharide antigen) to serogroups A and C polysaccharide, although the clinical relevance of this finding is unknown.

For both children and adults, MPSV4 is administered **subcutaneously** as a single 0.5 mL dose. The vaccine can be administered at the same time as other vaccines but should be given at a different anatomic site.

Adverse reactions to MPSV4 are generally mild. The most frequent complaints are local reactions such as pain and redness at the injection site. These reactions last for 1-2 days and occur in 5%-10% of recipients. Systemic reactions such as headache and malaise are reported in 2%-5% of recipients,

and low grade fever occurs in up to 3% of vaccinees. Severe reactions to polysaccharide meningococcal vaccine are uncommon.

A severe allergic (anaphylactic) reaction to a vaccine component or following a prior dose of meningococcal polysaccharide vaccine is a contraindication to the receipt of further doses. A moderate or severe acute illness is reason to defer routine vaccination but a minor illness is not. Pregnancy, breastfeeding and immunosuppression are not contraindications to vaccination.

MPSV4 should be shipped in insulated containers and stored at refrigerator temperature [2-8°C (35-46°F)]. The vaccine must not be exposed to freezing temperature. Single dose vials of MPSV4 must be used within 30 minutes of reconstitution. Multidose vials must be discarded 35 days after reconstitution. Providers should consult the drug package insert for additional information as needed.

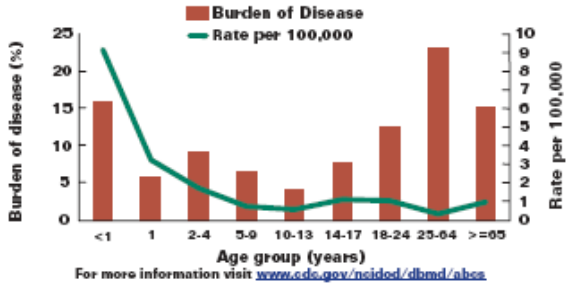
Meningococcal Conjugate Vaccines (MCV4)

Conjugation (i.e., covalent coupling) of polysaccharide to a protein carrier that contains T-cell epitopes changes the nature of the immune response from T-cell-independent to T-cell-dependent, leading to a significant primary response among infants and a strong anamnestic response at re-exposure. Both conjugate *Haemophilus influenzae* type b (Hib) and conjugate *Streptococcus pneumoniae* vaccines (introduced for mass infant immunization in the U.S. in 1990 and 2000, respectively) have substantially reduced the incidence of disease caused by vaccine-preventable serotypes.

Efforts to produce a conjugated meningococcal vaccine have yielded MCV4, a tetravalent meningococcal conjugate vaccine (Menactra™, Sanofi Pasteur, Inc., Swiftwater, PA) licensed for use in persons 11-55 years of age in the United States as of January 2005. A 0.5-mL single dose of vaccine contains 4 µg each of capsular polysaccharide from serogroups A, C, Y, and W-135 conjugated to 48 µg of diphtheria toxoid. MCV4 is available only in single-dose vials.

Figure 2

Rates of Meningococcal Disease by Age Group and Burden of Disease, United States, Active Bacterial Core Surveillance System, 1991–2002



MCV4 was licensed on the basis of findings indicating that it was comparable to MPSV4 in terms of immunogenicity and safety (i.e., demonstrated noninferiority).

For both children and adults, MCV4 is administered **intramuscularly** as a single 0.5 mL dose. The vaccine can be administered at the same time as other vaccines but should be given at a different anatomic site.

Adverse reactions to MCV4 are similar to those of MPSV4 and are also generally mild. The most frequent are local reactions such as pain and redness at the injection site. A severe allergic (anaphylactic) reaction to a vaccine component or following a prior dose of meningococcal conjugate vaccine is a contraindication to the receipt of further doses. A moderate or severe acute illness is reason to defer routine vaccination, but a minor illness is not. Data are not available at this time on the safety of MCV4 during pregnancy.

As with MPSV4, MCV4 should be shipped in insulated containers. Vaccine should be stored at refrigerator temperature [2-8 °C (35-46 °F)] and must not be exposed to freezing temperature. Providers should consult the drug package insert for additional information as needed.

Recommendations for Use of Meningococcal Vaccines

In general, the use of MCV4 is preferred among persons 11-55 years of age; if MCV4 is unavailable, MPSV4 is an acceptable alternative. Use of MPSV4 is recommended among children 2-10 years of age and persons 56 years of age or older. MPSV4 and MCV4 are both available through the Vaccines for Children (VFC) Program.

Routine Vaccination of Adolescents

The Advisory Committee on Immunization Practices (ACIP) recommends routine vaccination of young adolescents (defined as persons 11-12 years of age) with MCV4 at the preadolescent healthcare visit. For those adolescents who have not previously received MCV4, ACIP recommends vaccination before high school entry (at approximately 15 years of age) as an effective strategy to reduce meningococcal disease incidence among adolescents and young adults. Other adolescents who wish to decrease their risk for meningococcal disease may elect to receive MCV4.

Other Populations at Increased Risk

Vaccination is recommended for the following populations considered to be at an increased risk for invasive meningococcal disease:

- College freshmen living in dormitories (Note: In Virginia, all new full-time students at any public four-year college or university must be vaccinated against meningococcal disease or must sign a waiver refusing the vaccine);
- Microbiologists who are routinely exposed to isolates of *N. meningitidis*;
- Military recruits;
- Persons who travel to or reside in countries where *N. meningitidis* is hyperendemic or epidemic, particularly if contact with the local population will be prolonged. Vaccination is especially recommended for those visiting the parts of sub-Saharan Africa known as the "meningitis belt" during the dry season (December-June). Vaccination is required by the government of Saudi Arabia for all travelers to Mecca during the annual Hajj;
- Persons who have terminal complement component deficiencies; and,

- Persons who have anatomic or functional asplenia.

Persons with human immunodeficiency virus (HIV) are likely at an increased risk for meningococcal disease although not to the extent that they are at risk for invasive *S. pneumoniae* infection. While the efficacy of MCV4 among HIV-infected patients is unknown, HIV-infected patients may elect to be vaccinated with MCV4.

Adults 20-55 Years of Age

In adults 20-55 years of age, MCV4 is safe, immunogenic, and likely provides relatively long-lasting protection against meningococcal disease caused by serogroups A, C, Y, and W-135. Since rates of meningococcal disease are low for this age group, and since vaccination will decrease but not eliminate risk, routine vaccination is not recommended. However, persons who wish to decrease their risk for meningococcal disease may elect to be vaccinated.

Children <11 Years of Age and Adults >55 Years of Age

MCV4 is not licensed for use among children aged less than 11 years of age or adults older than 55 years of age. However, it is likely that this or a similar vaccine will be licensed for younger age groups in the future.

Routine vaccination with MPSV4 is not recommended for children less than two years of age because it is relatively ineffective and offers a short duration of protection. Routine vaccination with MPSV4 is not recommended for children 2-10 years of age and adults older than 55 years of age except for those identified as being at increased risk for meningococcal disease.

Outbreaks of Meningococcal Disease

Both MPSV4 and MCV4 are appropriate for use in the control of meningococcal outbreaks caused by vaccine-preventable serogroups (A, C, W-135, and Y) of *N. meningitidis*. However, use of MCV4 is preferred if the population targeted for vaccination includes age groups for which MCV4 is licensed. Detailed recommendations on the evaluation and management of suspected outbreaks of meningococcal disease have been published previously [MMWR. 1997. 46(RR-5);13-21].

Revaccination

Revaccination might be indicated for persons previously vaccinated with MPSV4 who remain at an increased risk for infection. In particular, children who were first vaccinated at less than four years of age should be considered for revaccination after 2-3 years if they remain at an increased risk. Although the need for revaccination among older children and adults after receiving MPSV4 has not been determined, if indications still exist for vaccination, revaccination might be considered after five years. While the use of MPSV4 is acceptable for revaccination of persons aged 11-55 years, MCV4 is recommended since it will likely provide longer protection than MPSV4. More data on the revaccination of persons previously vaccinated with MCV4 is necessary.

Public Health Response

Overall, the communicability of *N. meningitidis* is relatively limited. In studies of households where a case of meningococcal disease has occurred, only 3%-4% of households had additional cases (most with only a single additional case). As a result, the estimated occurrence of co-primary or secondary cases is 2-4 per 1,000 household members. This risk is 500-800 times that of the general population.

The potential severity of invasive meningococcal infections warrants that public health interventions focus on the rapid identification and management of at-risk individuals. Therefore, the *Regulations for Disease Reporting and Control* (Title 12 VAC 5-90-80) require that suspicion or confirmation of invasive *N. meningitidis* infections must be reported within 24 hours to the local health department by healthcare providers, directors of medical care facilities and directors of laboratories. Close contacts (e.g., household members; daycare center classmates; personnel who performed mouth-to-mouth resuscitation, intubated, or suctioned the patient before antibiotics were begun; and persons who had intimate contact with the patient's oral secretions through kissing, sharing of food or drink, sharing cigarettes, etc.) are then identified by health department staff. These individuals are evaluated and, if appropriate, receive chemoprophylaxis and education.

In addition, laboratories are required by the *Regulations for Disease Reporting and Control* (Title 12 VAC 5-90-90) to submit the initial culture of a meningococcal infection to the Division of Consolidated Laboratory Services (state laboratory). This enables laboratory confirmation of the serogroup for monitoring trends as well as genotyping for linking suspected outbreaks.

Summary

While relatively rare in occurrence, invasive disease caused by *N. meningitidis* often leads to tragic consequences. In addition to the impact on the patient and family, this condition generates significant public anxiety and requires extensive public health follow-up to protect against additional cases. The introduction of a new, conjugated vaccine that may provide longer-lasting immunity may significantly reduce the risk of meningococcal disease in vulnerable populations and communities. In light of the new meningococcal vaccine recommendations, existing Virginia vaccination recommendations for college entry may need to be modified in the future.

Selected References

American Academy of Pediatrics. Meningococcal Disease. In: Pickering L ed. Red Book: 2003 Report of the Committee on Infectious Diseases. 26th ed. Elk Grove Village, IL: American Academy of Pediatrics, 2003: 430-6.

CDC. Epidemiology and Prevention of Vaccine-Preventable Diseases. Atkinson W, Hamborsky J, Wolfe S, eds. 8th ed. Washington DC: Public Health Foundation, 2004.

CDC. Control and Prevention of Serogroup C Meningococcal Disease: Evaluation and Management of Suspected Outbreaks: Recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR 1997;46(RR-5);13-21.

CDC. Meningococcal disease and college students. Recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR 2000;49(RR-7):1-22.

CDC. Prevention and control of meningococcal disease: Recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR 2005;54(RR-7):1-17.

Granoff DM, Feavers I, Borrow R. Meningococcal Vaccines. Vaccine 2004:959-988.

Submitted by Laura Ann Nicolai, M.P.H., Division of Immunization

Release of the Compendium of Measures to Prevent Disease Associated with Animals in Public Settings, 2005

Millions of human-animal interactions occur each year at county or state fairs, petting zoos, animal swap meets, pet stores, carnivals, farm tours, and educational exhibits. Although benefits of human-animal interactions exist, infectious diseases (e.g., due to *Escherichia coli* O157:H7, *Salmonella*, *Coxiella burnetti*, *Mycobacterium tuberculosis*, ringworm), rabies exposures, injuries, and other human health problems (e.g., allergies) potentially associated with these settings are of increasing concern. Challenges of diseases related to these settings include difficulty in identifying and contacting persons, in correctly assessing exposure risks, and in providing timely medical treatment.

The *Compendium of Measures to Prevent Disease Associated with Animals in Public Settings, 2005*, became available in March 2005 in the Morbidity and Mortality Weekly Report (Vol. 54/No. RR-4). The report, prepared by the National Association of State Public Health Veterinarians, Inc. (NASPHV), provides recommendations for public health officials, veterinarians, animal venue operators, animal exhibitors, and visitors to animal venues for minimizing risks associated with animals in public settings. Special thanks go to Dr. John Marr, Virginia Department of Health, for serving as a consultant on the NASPHV committee.

Some observations of note from the *Compendium* include:

- Reports of infectious disease outbreaks and injuries associated with animal exhibit settings have increased. This increase is likely multifactorial involving emerging pathogens (e.g. *Cryptosporidium* and *E. coli* O157), host immunity and the unique environments that enable disease transmission;
- Serious illnesses have occurred, especially in persons at high risk (e.g., children);
- Many of these pathogens do not cause illness in animals. Therefore, the removal of ill animals is necessary but not sufficient to protect animal and human health;
- Infected animals may shed pathogens intermittently, so attempts to screen for infected animals might not be effective in eliminating the risk of transmission;
- Antimicrobial treatment of animals cannot be depended upon to eliminate infection, reduce shedding of enteric pathogens or prevent re-infection of animals;
- Multiple factors (e.g., stress, commingling, age of animals) increase the probability of transmission at animal exhibits;
- Outbreaks are often associated with hand-to-mouth contact (fecal-oral transmission). The risk for infections is increased with certain human behaviors, including inadequate hand washing; a lack of close supervision of children; hand-to-mouth activities (e.g., use of pacifiers, thumb-sucking, smoking, and eating) in proximity to animals; and, a lack of awareness of the risk; and,
- The layout and maintenance of facilities contributes to the risk for infection, including inadequate handwashing facilities; structural deficiencies associated with temporary food-service facilities; inadequate separation between animal exhibits and food-consumption areas; and, contaminated or inadequately maintained drinking water and sewage/manure disposal systems.

The recommendation to wash hands is the single most important step for reducing the risk of disease transmission. However, other critical recommendations include:

- Proper education of staff, exhibitors, and visitors regarding methods to prevent disease transmission;
- Optimal facility design, including barriers, proper waste disposal, ventilation, and the presence of transition zones for handwashing between animal and non-animal areas;
- Close supervision of children;
- The appropriate care and management of animals; and,
- Warning at-risk populations to take extra care.

While human-animal contact has clear benefits, there are also associated health risks. These risks can be minimized through appropriate prevention strategies. For more detail on the recommendations provided in the 2005 *Compendium* visit www.cdc.gov/mmwr/preview/mmwrhtml/rr5404a1.htm.

Reference

1. Adapted from Disease Control Newsletter, Minnesota Department of Health. 2005. Vol. 33/No. 2.

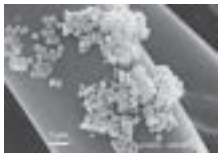
Quick Guide to Important Animal-Pathogen Associations[1]

Animal Reservoir	Pathogens to Consider
Calves	*Cryptosporidium, E. coli* O157:H7, *Campylobacter, Salmonella*
Reptiles	*Salmonella*
Baby Chicks & Ducklings, Adult Poultry	*Salmonella, Campylobacter*
Puppies & Kittens (especially w/diarrhea)	*Campylobacter, Salmonella, Giardia, Cryptosporidium,*

Epidemiology in Virginia

With summer in Virginia comes the seasonal increase in risk for foodborne diseases. The following abstract, illustrating an investigation of a foodborne outbreak in Virginia, was presented during the 2004 Virginia Department of Health Field Epidemiology Seminar in Charlottesville, VA.

COOKING WITH STAPH:



Staphylococcal Intoxication at a Benefit Luncheon

Background – On the evening of July 12, 2003, fourteen persons with symptoms of staphylococcal food intoxication were reported to the local health department by the Wythe County Community Hospital. All fourteen persons had attended a family benefit dinner at a local, private lodge between the hours of 12 noon and 3 pm on that day.

Methods – A case-control study was performed. Forty of approximately 45 attendees of the benefit dinner were interviewed using a telephone questionnaire developed to investigate this outbreak. A case was defined as any person who attended the benefit on July 12 and who had confirmed enterotoxin-producing *Staphylococcus aureus* and/or experienced vomiting in addition to nausea, diarrhea or abdominal cramps. The control group was identified by event coordinators and cases.

Results – Nineteen persons attending the benefit dinner met the case definition. Odds ratios were calculated for all food items and only one was statistically associated with illness; cases were 11.1 times more likely to have eaten ham than the controls (95% CI = 1.2-518.1; p-value = 0.02).

Conclusions – *S. aureus* intoxication occurs after ingesting food contaminated with staphylococcal enterotoxin. The risk is increased for foods that come into contact with food handlers' hands prior to inadequate cooking or refrigeration. It is thought that this outbreak was caused by both time and temperature abuse of approximately 20 pounds of cooked ham: this ham was stacked in a container, transported on ice from North Carolina to Virginia, and then reheated in the same container. Under these conditions, it is likely that bacterial growth was promoted. Proper monitoring of time-temperature procedures during and after food preparation helps to prevent enterotoxin-producing organism growth and associated human illness.

Submitted by: Marlene Peters, BSN

Tick Bites and Prophylaxis for Lyme Disease

Ticks can transmit a variety of diseases, including Lyme disease, Rocky Mountain spotted fever (RMSF), and ehrlichiosis. Although Virginia's temperate climate means that ticks may be active any time during the year, with summer comes the increased risk of exposure as people venture outdoors to enjoy the warm weather. This article reviews some of the current recommendations for managing tick bites, with a particular focus on Lyme disease.

Borrelia burgdorferi, 400x magnification

Lyme Disease

Lyme disease is a bacterial illness caused by *Borrelia burgdorferi*, a spirochete. Although Lyme disease is the most frequently reported tickborne illness in Virginia, with 216 cases reported in 2004, the overall incidence is very low (approximately 2.9 per 100,000 population in 2004). Other regions, such as the Northeastern and the upper Midwestern states, have much higher levels of Lyme disease. For example, Lyme disease incidence in northern Westchester County, NY is 0.5-1 per 1,000 population, approximately 35-70 fold higher than in Virginia.[1] [Note: Meyerhoff (2004) suggests that actual rates may be five times higher than state health department rates.]

The manifestations of Lyme disease have been divided into three stages: localized, disseminated and persistent (chronic). The primary sign of localized disease is erythema migrans. Symptoms can also include headache, fever, muscle and joint aches, and fatigue.[2,3] The primary signs and symptoms of disseminated disease are intermittent arthritis, cranial nerve palsies and radicular symptoms, atrioventricular (AV) nodal block, and severe malaise and fatigue. The primary signs and symptoms of persistent disease are prolonged arthritis, chronic encephalitis, myelitis and parapareses, and symptoms consistent with fibromyalgia.[2]

Lyme Disease Vector: Ticks

The black legged or deer tick (*Ixodes scapularis*) is the most common carrier of Lyme disease in the eastern United States. Although deer ticks are found in the eastern part of Virginia, they are not as common as American dog and lone star ticks (neither of these ticks transmit Lyme disease). Studies done in Virginia in the mid-1980s on the Eastern Shore and in the Williamsburg/Yorktown area identified *B. burgdorferi* infected rodents and ticks, but the percentages that were infected were much lower than in other parts of the country where more human cases are reported. There have been no recent studies to see

how these infection rates may have changed over time.[3]

The adult deer tick lays eggs that hatch into larvae. During the summer, the larvae feed on small rodents, most commonly the white-footed mouse. If the rodents are carrying *B. burgdorferi*, the tick larvae can become infected. Larvae molt into nymphs, which are dormant during the winter and become active the following spring and summer. If the larvae were infected with *B. burgdorferi*, the nymphs will also contain the spirochete. Transmission usually occurs when the nymph feeds on animals and occasionally on humans. By fall nymphs become adults that may also transmit the disease.

Human infection with *B. burgdorferi* by the nymph or adult tick usually requires that the tick **attach to a person for 1-2 days**. This is related to the life cycle of *B. burgdorferi* in ticks. In previously infected ticks, only small numbers of bacteria are present until the tick feeds. Once feeding begins, the bacteria multiply in the gut of the tick. After 1-2 days, the bacteria travel to the tick's salivary glands where they are injected into the animal as the tick ends its feeding. Until multiplication of the spirochete occurs, ticks are rarely able to pass on the infection.[2]

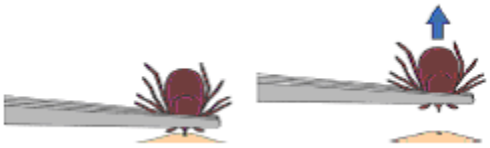
Prevention

The best way to prevent infection with a tickborne disease is to avoid tick-infested areas. However, if exposure to ticks is unavoidable, the risk of infection may be reduced by using light-colored, protective clothing and tick repellants; checking the entire body for ticks every 4-6 hours; and promptly removing attached ticks using fine-toothed forceps.[4]

Tick Removal

Although ticks do not transmit disease until they have been attached for a prolonged period (4-6 hours for RMSF and ehrlichiosis; 1-2 days for Lyme disease), it is very important to remove ticks as soon as they are found. The following is the best way to remove a tick:

1. Grasp the tick with tweezers as close to the skin as possible and gently, but firmly, pull it straight out. Avoid any twisting or jerking motion that may break off the mouth parts in the skin. Mouth parts left in the wound will not transmit the disease, but may cause a minor irritation or infection.



2. If tweezers are not available, use your fingers to remove the tick; however, be sure to protect your fingers with gloves, tissue, or a paper towel. Do not touch the tick with bare fingers. The disease-causing organism could enter the body through a break in the skin and cause disease.
3. After the tick has been removed, wash your hands with soap and water. Apply an antiseptic to the bite site.
4. Dispose of the tick by drowning it in alcohol or flushing it down a drain or toilet.
5. Do not remove ticks using nail polish, petroleum jelly, alcohol or a hot match; these methods are not safe.[3]

Chemoprophylaxis

Chemoprophylaxis using antimicrobials in the general population or high-risk groups **prior** to a tick bite is not recommended. Education will help to minimize the risk of Lyme disease (see the Virginia Department of Health brochure: *Preventing Tickborne Disease in Virginia).*

Despite the concern over Lyme disease following a tick bite, the routine use of antimicrobial prophylaxis or serologic testing **after** a tick bite is not recommended.[4] While Nadelman et al (2001) suggested that treatment with a single dose of 200 mg of doxycycline within 72 hours of removing a tick can prevent the development of Lyme disease, this finding was most applicable when the person was bitten by a tick in an area where the incidence of Lyme disease is high and when the tick is a nymphal deer tick that is at least partially engorged with blood. In the far more common circumstance where the bite occurs in an area where the incidence of Lyme disease is not high (e.g., Virginia), where the tick is not a nymphal deer tick (or either the species or the stage of the tick is unknown), or where the tick is not at least partially engorged, the risk of Lyme disease is likely to be so low that prophylaxis is not indicated.[1] Previous vaccination with the recombinant OspA vaccine preparation (no longer available) reduces the risk of developing Lyme disease associated with tick bites but does not alter the above recommendations.[4]

Persons who remove attached ticks should be monitored closely for signs and symptoms of tickborne diseases for up to 30 days. Healthcare providers should tell patients to watch for the occurrence of a skin lesion at the site of the tick bite that may suggest Lyme disease, or for a temperature of more than 38°C (100.4°F) that may suggest human granulocytic ehrlichiosis or babesiosis. Persons who develop a skin lesion or other illness within one month after removing an attached tick should promptly seek medical attention.[4] Testing of ticks for infectious organisms is not recommended, except in research studies.

For further information on ways to minimize the risk of exposure to ticks, please see the Division of Zoonotic and Environmental Epidemiology (DZEE) website (www.vdh.virginia.gov/whc/external_whc/ZEEpageExternal.asp) or the Centers for Disease Control and Prevention website (www.cdc.gov/ncidod/ticktips2005/).

References

1. Shapiro ED. 2001. *N Engl J Med.* 345: 133-134.
2. Meyerhoff J. 2004. *Emedicine.* www.emedicine.com/med/topic1346.htm. Accessed: June 13, 2005.
3. Virginia Department of Health. Preventing Tickborne Disease in Virginia. www.vdh.virginia.gov/whc/external_whc/Tick%20Brochure.pdf. Accessed: June 13, 2005.
4. Preboth M. 2001. *Am Fam Physician.* Vol. 63/No. 10.
5. Nadelman RB, Nowakowski J, Fish D, et al. 2001. *N Engl J Med.* 345:79-84.

Cases of Selected Notifiable Diseases Reported in Virginia*

Disease	Total Cases Reported, May 2005: State	Regions: NW	N	SW	C	E	Total Cases Reported Statewide, January - May: This Year	Last Year	5 Yr Avg
AIDS	45	4	17	4	13	7	255	284	309
Campylobacteriosis	36	6	9	9	2	10	150	178	157
E. coli O157:H7	3	0	1	2	0	0	7	1	10
Giardiasis	30	10	7	6	2	5	211	141	126
Gonorrhea	550	41	33	49	182	245	3,332	3,595	3,590
Hepatitis, Viral									
A	10	0	3	1	1	5	39	33	44
B, acute	9	0	1	2	2	4	82	80	68
C, acute	1	0	1	0	0	0	7	7	2
HIV Infection	72	4	14	2	24	28	312	350	341
Lead in Children†	43	3	1	13	18	8	169	234	219
Legionellosis	5	0	1	2	2	0	10	8	6
Lyme Disease	5	0	4	0	1	0	33	13	16
Measles	0	0	0	0	0	0	0	0	0
Meningococcal Infection	3	0	1	1	0	1	14	8	17
Mumps	0	0	0	0	0	0	0	2	2
Pertussis	11	3	0	5	2	1	74	59	37
Rabies in Animals	49	8	5	14	10	12	196	199	201
Rocky Mountain Spotted Fever	2	0	0	0	1	1	6	1	1
Rubella	0	0	0	0	0	0	0	0	0
Salmonellosis	89	9	19	28	21	12	299	251	282
Shigellosis	9	1	4	2	0	2	36	36	128
Syphilis, Early§	34	0	8	1	5	20	98	69	79
Tuberculosis	33	1	21	1	3	7	108	84	85

Localities Reporting Animal Rabies This Month: Accomack 1 fox, 1 raccoon; Albemarle 1 raccoon; Alleghany 1 raccoon; Arlington 1 raccoon; Bath 1 skunk; Campbell 1 fox; Carroll 2 raccoons; Chesterfield 1 bat, 1 raccoon; Clarke 1 cat; Cumberland 1 raccoon; Fairfax 1 bat, 1 raccoon; Fauquier 1 raccoon; Floyd 1 cow, 1 raccoon; Franklin 1 cow; Gloucester 1 fox; Hampton 1 raccoon; Hanover 3 raccoons; Henry 1 raccoon; Isle of Wight 1 raccoon; King William 1 skunk; Loudoun 1 fox; Lunenburg 1 raccoon; Montgomery 1 raccoon; Northampton 2 raccoons; Patrick 3 raccoons; Powhatan 1 raccoon; Prince George 1 raccoon; Prince William 1 cat; Richmond City 1 raccoon; Roanoke 1 fox; Rockbridge 1 skunk; Rockingham 1 raccoon; Shenandoah 1 fox, 1 raccoon; Suffolk 1 fox; Westmoreland 1 cat, 1 raccoon; Wythe 1 raccoon; York 1 raccoon.

Toxic Substance-related Illnesses: Adult Lead Exposure 2; Asbestosis 2; Pneumoconiosis 4.

*Data for 2005 are provisional.

†Elevated blood lead levels ≥10µg/dL. §Includes primary, secondary, and early latent.

Published monthly by the
VIRGINIA DEPARTMENT OF HEALTH
Office of Epidemiology
P.O. Box 2448
Richmond, Virginia 23218
http://www.vdh.virginia.gov
Telephone: (804) 864-8141

VDH VIRGINIA DEPARTMENT OF HEALTH
Protecting You and Your Environment

PRESORTED STANDARD U.S. POSTAGE PAID Richmond, Va. Permit No. 591