

Norovirus

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Noroviruses are RNA viruses that cause acute gastroenteritis in humans. The name “Norovirus” was recently approved as the official genus name for the group of viruses previously described as “Norwalk-like viruses”.

Noroviruses are transmitted primarily through the fecal-oral route, either by direct person-to-person contact or by contaminated surface, food, or water. Noroviruses can also spread by droplets from vomitus. In addition, transmission often occurs through hand transfer of the virus to the oral mucosa via contact with materials, fomites, and environmental surfaces that have been contaminated with either feces or vomitus. Contaminated surfaces handled by multiple persons such as doorknobs, sink hardware, or other shared items are the likely source of many infections. Noroviruses are highly contagious, with as few as 10 virus particles probably sufficient to cause infection. These viruses are relatively stable in the environment and can survive freezing and heating to 60 degrees Celsius (140 degrees Fahrenheit).

The average incubation period for norovirus-associated gastroenteritis is 12 to 48 hours, with a median of about 33 hours. Symptoms usually last 24 to 60 hours and include acute-onset of vomiting, watery/non-bloody diarrhea, abdominal cramps, and nausea. In addition, myalgia, malaise, and headache are commonly reported. Low-grade fever may be present. Dehydration is the most common complication and may require intravenous replacement fluids.

Diagnosis of norovirus infection is often made clinically in individual cases, especially when other reasons for gastroenteritis can be ruled out. Laboratory testing for norovirus, using an RT-PCR assay to detect viral RNA in the stool, is available at some reference laboratories and in most state public health laboratories, including the cas-

Laboratory Testing of Shiga-Toxin Producing *Esherichia coli*

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Enterohemorrhagic *Esherichia coli* (EHEC) is recognized as an important cause of diarrhea, hemorrhagic colitis (bloody diarrhea), and hemolytic uremic syndrome (HUS). Perhaps the most notable EHEC is *E.coli* O157:H7, which was recently associated with an outbreak of contaminated spinach that resulted in 199 confirmed cases and three deaths in 26 states. However, more than 100 non-O157:H7 serotypes are also capable of causing similar serious illnesses. The pathogenicity of these organisms is associated with the production of shiga-like toxins, and they are therefore known collectively as Shiga-toxin producing *E. coli* (STEC). Cattle and small ruminants are natural reservoirs of STEC, and domestic animals have been demonstrated to be asymptomatic carriers (1). It is estimated that STEC causes 110,000 human illnesses a year in the United States, and greater than or equal to 30 percent are caused by non-O157 serogroups of *E. coli*.

Two shiga-toxins are produced by STEC: Stx1 and Stx2. A single strain can produce one or both of these toxins. Stx1 and Stx2 share DNA sequence similarity with the shiga-toxin produced by *Shigella dysenteriae*, and their mechanism of action is similar to that of Ricin toxin – they inactivate the 60S ribosomal subunit, blocking protein synthesis. Local action of the cytotoxins Stx1 and/or Stx2 results in hemorrhagic colitis. One or both of the shiga-toxins may also enter the blood and bind to the Gb3 receptor found on kidney endothelial cells. This can lead to a

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cade of events that culminates in DHEC Bureau of Laboratories. Testing should be considered in the event of outbreaks of gastroenteritis in healthcare and other institutional facilities as well as certain other settings. When laboratory testing is indicated, the virus can best be identified from stool specimens taken within 48 to 72 hours after the onset of symptoms, although results can be obtained as long as seven days after the onset of symptom.

Special attention to hand hygiene practices is very important in reducing transmission. Patients with suspected norovirus infection should be managed with Standard Precautions. However, Contact Precautions should be used when caring for diapered or incontinent persons, during outbreaks in a facility, and when there is the possibility of splashes that might lead to contamination of clothing. Persons cleaning areas heavily contaminated with vomitus or feces should wear surgical masks. In an outbreak setting, placing patients with suspected norovirus infection in private rooms or clustering potentially infected patients should be considered.

The Centers for Disease Control (CDC) recommend either chlorine bleach or U.S. Environmental Protection Agency (EPA) approved disinfectants for use in controlling transmission of norovirus infection. Chlorine bleach should be applied to hard, non-porous, environmental surfaces at a dilution of 1 part household bleach solution to 50 parts water. In areas with high levels of soiling and resistant surfaces, a higher concentration of bleach is recommended. When norovirus contamination is suspected, cleaning procedures that increase the aerosolization of norovirus should not be utilized, such as vacuuming carpets or buffing hard surface floors. Contaminated carpeting should be disinfected with a chemical disinfectant if possible, and then steam cleaned for a minimum 5-minute contact time at a minimum temperature of 170 degrees Fahrenheit. Environmental disinfection recommendations in the literature include the need to disinfect all heavy hand contact surfaces such as food preparation surfaces, self-service utensil handles, faucets, tables, chairs, counters, door handles, push plates, railings, elevator buttons, telephones, keyboards, vending machine keyboards, pens, pencils, casino chips, cards, slot machines, sports equipment, etc. Public restroom surfaces, including faucet handles, soap dispensers, stall doors and latches, toilet seats and handles, and towel dispensers are also important heavy fecal contamination areas that require disinfection.

In South Carolina there has been a recent increase in the number of Norovirus outbreaks reported to DHEC. This increase is consistent with the national trend of increased Norovirus reports. In 2006 a total of 28 outbreaks of laboratory confirmed Norovirus were reported to DHEC. Between January 1, 2007 and March 15, 2007, a total of 29 outbreaks of laboratory confirmed Norovirus were reported to DHEC, representing this recent increase. DHEC requests that facilities contact the regional DHEC public health office if there is an increase in gastroenteritis among persons in the facility.

References:

FDA, CFSAN Risk Profile Norovirus (Draft)
Centers for Disease Control and Prevention -
<http://www.cdc.gov/>
American Academy of Pediatrics, The Red Book – 2006 Report of the Committee on Infectious Diseases, 27th edition.

(LABORATORY TESTING OF SHIGA-TOXIN PRODUCING ESHERICHIA COLI cont'd from Page 1)

obstruction of the renal vasculature and renal failure. Production of shiga-toxin alone isn't enough to produce disease. Other important virulence factors are intimin (eae), which facilitates an intimate attachment to intestinal enterocytes, and hemolysin (e-hyl). Antibiotic treatment may increase the chances of developing HUS – although there is conflicting evidence.

Traditionally, laboratory confirmation of *E. coli* O157:H7 relied on its inability to ferment sorbitol. This has made the organism relatively easy to distinguish from other strains of *E. coli*, including non-O157 isolates. Because sorbitol fermentation is only 50-80 percent sensitive for the detection of O157:H7 and because it is not possible to biochemically differentiate STEC producers from their less virulent counterparts, the only way we can currently screen for STEC is by shiga-toxin detection. This can be done by two methods: enzyme immunoassay (EIA) and polymerase chain reaction (PCR). The STEC EIA detects shiga-toxins 1 and 2 in stool, but does not distinguish which toxin is present or what organism is producing the toxin. *Citrobacter* and *Enterobacter* species and *Shigella dysenteriae* can also cause HUS and produce shiga-toxins that cross react in the EIA. Large commercial laboratories (notably Lab Corp and Quest) are no longer culturing stool specimens for STEC. Instead, they offer only the shiga toxin EIA test to their clients. The STEC EIA can be very sensitive (79–100 percent) and specific (96 – 98 percent); however, physicians must use care in interpreting these tests for their patients. A positive STEC EIA does not confirm a diagnosis of *E. coli* O157:H7.

Commercial laboratories do not perform follow up cultures to identify the shiga toxin producing bacteria in the toxin positive samples, but because O157:H7 is a reportable disease, enrichment broths are sent to the Bureau of Laboratories (BOL) for isolation of STEC for epidemiologic purposes. Timing of this referral is critical – many are no longer viable when they reach the state lab. Specimens that are non-viable are forwarded to the CDC, where a PCR is performed for four virulence factors: stx1, stx2, eae, and e-hyl. Presence of DNA confirms the diagnosis of STEC. While the EIA is sensitive and specific (100 percent and 98 percent respectively), culture must be performed for serotyping and epidemiological studies. Pulsed-field gel electrophoresis (PFGE) is performed on all STEC isolates to identify potential clusters of food borne illness. All cases of diarrhea investigated by DHEC with no apparent cause (negative for Norovirus or other enteric organisms) are screened for shiga-toxin production. Some laboratories use bloody diarrhea as a criteria for

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**(LABORATORY TESTING OF SHIGA-TOXIN
PRODUCING *ESHERICHIA COLI* cont'd from Page 2)**

testing for *E. coli* O157:H7, however this can be a poor indicator. One study found that 52 percent of cases of STEC would have been missed if only bloody diarrhea were tested (2). Stx2 is strongly associated with diarrhea and eae with HUS (3)

Nationally the most common non-O157 serogroups are: O26 (22 percent), O111 (16 percent), O103 (12 percent), O121 (8 percent), O45 (7 percent), and O145 (5 percent). These 6 serotypes accounted for 71 percent of the isolates recovered from 1983 to 2002 (3). In the last year, South Carolina has seen four serotypes of *E. coli*: O157:H7, O55:H7, O118:H16, and O121:H19. Strain O157:H7 remains most common, but this may be because many laboratories screen for it specifically and are not capable of detecting non-O157 isolates. The BOL has been actively pursuing non-O157 isolates since July 2006 and maintains reagents to serogroup O111 and O26. As additional serogroups are identified in the state, capacity will be increased to detect those that are most common.

A recent case of non-O157 STEC in a Lexington County 18-month-old demonstrated the challenge of daycare exclusion for patients with STEC illness. The child had diarrhea and cramps, but never developed bloody stool or HUS. A STEC EIA performed at a reference laboratory was positive, however broth submitted to the BOL did not grow in culture. The child submitted a second stool sample two weeks after the initial EIA. This sample was also positive by EIA and grew a non-O157, non-O111, non-O26 *E. coli*. Serological testing by the CDC identified the organism as *E. coli* O55:H7, and PCR detected two virulence factors: stx2 and eae. At the suggestion of the physician, the child was excluded from daycare until two consecutive negative cultures were obtained (27 days). The organism may be shed in the stool for several weeks. A study of day-care associated infections in Germany showed that patients with diarrhea or hemorrhagic colitis shed for a median duration of 13 days (range of 2-62 days) and patients with HUS shed for a median duration of 21 days (range of 5-124 days) (4). Other studies reported a median duration of 29 days (5). Duration appears to vary inversely with age, with younger children shedding longer, and studies disagree on whether severity of illness affects duration of shedding (6, 4, 7). Free fecal shiga-toxin may remain measurable for four to six weeks; therefore, isolation of the organism is critical to avoid unnecessary exclusion from daycare. It is unknown how many patients actually follow this rule and return to the doctor for follow-up testing. However the amount of testing required to confirm two negative cultures in an 18-month-old over a four week period (eight stool specimens) suggests that few patients or their parents are as conscientious.

References:

1. Busch et al. EID. 13:348. 2007
2. Gavin et al. Clin Micro Newsletter 26:7. 2004
3. Brooks et al. JID. 2005:192
4. Karch et al., J. Clin Micro. 33:1602
5. Shah et al. Clin Infect Dis. 1996. 23:835-86
6. Belongia, et al. JAMA. 1993. 269:883-88
7. Oai et al. J Infect Dis. 1988. 157:1054-57

Preventing Illnesses and Injuries Associated with Animal Contact Settings

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Venues where humans and animals commonly interact include public stables, petting zoos, traveling photo opportunities, schools, children's parties, livestock shows, and animal rides. These activities normally increase in the summer because of the general increase in outdoor activities and family vacations/outings. Although there is always some risk involved when interacting with animals, awareness of the hazards and careful behavior will decrease the chances of turning a routine activity into a disaster.

Although enteric bacterial illnesses are the most commonly reported health risks associated with animals in public settings, multiple other health risks are of concern. For example, allergies can be associated with animal dander, scales, fur, feathers, urine, and saliva. Additional health concerns include injuries, rabies exposures, and other infections. Both wild and domestic animals are unpredictable and can cause serious injuries, particularly to small children. Also, animals infected with enteric pathogens (e.g., *E. coli* O157:H7, *Salmonella*, and *Campylobacter*) frequently exhibit no signs of illness and may shed pathogens intermittently.

Injuries

Injuries associated with animals in public settings include bites, kicks, falls, scratches, stings, crushing of the hands or feet, and being pinned between the animal and a fixed object. These injuries have been associated with multiple species, including big cats (e.g., tigers), monkeys, domestic animals, and zoo animals.

Infections

Multiple bacterial, viral, fungal, and parasitic agents have been associated with animal contact. These organisms are transmitted through various modes. Exposure to animal feces can result in infection with *E. coli* O157:H7, *Salmonella*, and *Campylobacter*. Infections from animal bites are common and frequently require extensive treatment or hospitalization. Bacterial pathogens that are frequently associated with animal bites include *Pasteurella*, *Staphylococcus*, *Streptococcus*, *Capnocytophaga canimorsus*, *Bartonella henselae* (cat scratch disease), and *Streptobacillus moniliformis* (rat bite fever). Certain monkey species (especially macaques) that are kept as pets or used in public exhibitions can be infected with herpes B virus, either asymptotically or with mild oral lesions. Human exposure through bites or fluids can result in a fatal meningoencephalitis. Because of difficulties with laboratory testing to confirm monkey infection and high herpes B prevalence, monkey bites can require intensive public health and medical follow-up.

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Skin contact with animals in public settings might also result in human infection. Ringworm infection caused by *Trichophyton* species and *Microsporum gypseum* have been documented among pet and livestock owners. Ringworm infection in 23 persons and multiple animal species were traced to a *Microsporum canis* infection in a hand-reared zoo tiger cub. Orf virus infections (contagious ecthyma or sore mouth) have occurred in goats and sheep at a children's petting zoo and in a lamb used for an Easter photo opportunity. In 2003, multiple cases of monkeypox occurred among persons who had had contact with infected prairie dogs either at a child care center or a pet store.

Ecto- and endoparasites pose concerns when humans and exhibit animals interact. *Sarcoptes scabiei* is a skin mite that infests humans and animals, including swine, dogs, cats, foxes, cattle, and coyotes. Although human infestation from animal sources is usually self-limiting, skin irritation and itching may occur for multiple days and be difficult to diagnose. Animal fleas bite humans, which increases the risk for infection or allergic reaction. In addition, fleas are the intermediate host for a tapeworm species that can infect children. Multiple other animal helminths might infect humans through fecal-oral contact or through contact with animals or contaminated earth.

Tuberculosis (TB) is another disease of concern in certain animal settings. Twelve circus elephant handlers at an exotic animal farm were infected with *Mycobacterium tuberculosis*, and one handler had signs consistent with active disease after three elephants died of TB. Medical history and testing of the handlers indicated that the elephants had been a probable source of exposure for the majority of the human infections. At a zoo, seven animal handlers who were previously negative for TB tested positive after a *Mycobacterium bovis* outbreak in rhinoceroses and monkeys.

Zoonotic pathogens may also be transmitted by direct or indirect contact with reproductive fluids, aborted fetuses, or newborns from infected dams. Live-birthing exhibits, usually involving livestock (e.g., cattle, pigs, goats, or sheep), are popular at agricultural fairs. Although the public usually does not have direct contact with animals during birthing, newborns and their dams are frequently available for petting and observation afterward. Q fever (*Coxiella burnetii*), leptospirosis, listeriosis, brucellosis, and chlamydiosis are serious zoonoses that can be associated with contact with reproductive materials. *C. burnetii* is a rickettsial organism that most frequently infects cattle, sheep, and goats. The disease can cause abortion in animals, but more frequently the infection is asymptomatic. During parturition, infected animals shed substantial numbers of organisms that might become aerosolized. The majority of persons exposed to *C. burnetii* develop an asymptomatic infection, but clinical illness can range from an acute influenza-like illness to life-threatening endocarditis. A Q fever outbreak involving 95 confirmed case-patients and 41 hospitalizations was linked to goats and sheep giving birth at petting zoos. These petting zoos

were in indoor shopping malls, indicating that indoor-birthing exhibits might pose an increased risk for Q fever transmission.

Chlamydophila psittaci infections cause respiratory disease (commonly called psittacosis) and are usually acquired from psittacine birds. For example, an outbreak of *C. psittaci* pneumonia occurred among the staff at a Zoo.

Rabies Exposures

Contact with mammals may expose persons to rabies through contamination of mucous membranes, bites, scratches, or other wounds with infected saliva or nervous tissue. Although no human rabies deaths caused by animal contact in public exhibits have been recorded, multiple rabies exposures have occurred, requiring extensive public health investigation and medical follow up. Persons have received rabies postexposure prophylaxis (PEP) after being exposed to rabid or potentially rabid animal species (including cats, goats, bears, sheep, ponies, and dogs) at sites including pet stores, county fairs, petting zoos, schools, and rodeo events. Prompt assessment and treatment are critical for this disease, which is usually fatal.

Brucellosis

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Human cases of brucellosis are listed as an "urgently reportable condition" on the SC Department of Health and Environmental Control (DHEC) List of Reportable Conditions. Suspect or probable cases should be reported to DHEC within 24 hours by phone. Brucellosis is caused by a bacterial pathogen and is often associated with exposure to infected animals or animal products. It is also included on the list of potential bioterrorism agents.

In addition to transmission via infected animals and animal products, *Brucella sp.* may be transmitted via aerosolization in animal kennels or microbiological laboratories. Contact with aborted animal fetal tissues or fluids are an important source of infection for veterinarians, animal producers, and breeders. Ingestion of unpasteurized milk and cheeses made from unpasteurized milk are also risk factors associated with brucella infection.

There have been two confirmed cases of human brucellosis reported to DHEC thus far in 2007. One case was imported (the patient was exposed while traveling abroad). The other case was acquired locally and associated with contact to infected feral swine carcasses during processing.

Although human brucellosis is not common in the US (about 100-200 cases are diagnosed per year according to the Centers for Disease Control and Prevention (CDC),

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South Carolina has some unique sources of potential exposure. The SC Department of Natural Resources (DNR) has conducted studies of feral swine in South Carolina and found that brucellosis is enzootic (endemic) in the wild hog population. Hunting wild hogs is a popular hunting activity in South Carolina since the wild hog population is plentiful and destructive to native flora and fauna. The hunters of these wild hogs and the individuals who process the carcasses for human consumption are at risk of infection with *B. suis*, the *Brucella* species that can be found in both wild and domestic swine (South Carolina commercially raised swine are brucellosis-free). The DNR periodically reminds hunters of this risk in DNR publications, but at-risk patients may fail to link an illness to their hunting activities. Therefore, it is important to query patients with compatible illness regarding any wild hog hunting or processing activities.

International travel is also a risk factor for exposure to *Brucella* sp., particularly in the Mediterranean basin. Another term for brucellosis is Malta Fever, named after the island of Malta in the middle of the Mediterranean Sea. Goats are an important source of infection in the Mediterranean region. *B. melitensis* is the species associated with goats. Many rural communities rely on goat meat and milk as a source of food and income. Soft cheeses produced from unpasteurized goat milk can be purchased by tourists unaware of the risk. *Brucella* sp. are also found in cattle, sheep, deer, elk, dogs, coyotes, and other animals worldwide. Human to human spread is rare, but possible, through sexual transmission or breast-feeding of infants. The incubation period for brucellosis in humans ranges from less than a week to several months.

Brucellosis in humans is characterized by vague symptoms often associated with the "flu." Signs and symptoms include fever, night sweats, headaches, back pain, fatigue, and arthralgias. Severe illness can affect the central nervous system and heart. Signs and symptoms may diminish and recur resulting in an undulating pattern. (Hence the name Undulant Fever was historically ascribed to brucellosis.)

Most commercial laboratories can conduct testing for *Brucella* sp. In general, the screening tests are fairly sensitive but not extremely specific, so false positives may occur. Confirmatory testing is recommended as well as testing of both acute and convalescent sera collected at least two weeks apart. Samples may be sent to the DHEC Bureau of Laboratories for confirmatory testing.

Treatment of brucellosis with rifampin and doxycycline for six weeks has been successful in humans. Other treatment regimens are also published.

The CDC case definition for human brucellosis is located on the CDC Web site at:
http://www.cdc.gov/epo/dphsi/casedef/brucellosis_current.htm

HPV Disease in the United States and South Carolina and the HPV Vaccine

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Human papilloma viruses (HPV) are a large family of more than 100 types of small DNA viruses that exhibit a high degree of tissue and cellular specificity. Some HPV types infect nongenital skin and cause benign lesions, verrucae, and plantar warts. Other types infect genital skin and mucous membranes. These HPV types are divided into low and high risk, based on their capacity to induce cancer. Of the 15 high-risk HPV types, HPV types 16 and 18 are responsible for approximately 70 percent of squamous cell cancers of the cervix. HPV 16 and 18 can also cause low-grade cervical dysplasia and benign genital warts. Low risk HPV types 6 and 11 cause approximately 90 percent of benign genital warts and have low oncogenic potential.

Genital HPV infection is one of the most common sexually transmitted infections in females and males, with the majority being asymptomatic, unrecognized, or subclinical. Although most young persons can clear HPV infections with no clinical consequences, certain infections persist and result in warts, precancerous changes, and invasive cancers of the anogenital region in both males and females. In 2000, approximately 6.2 million new HPV infections occurred among females and males aged 15–44 years in the United States with 75 percent (4.6 million) of these new infections occurring among 15–24 year old females and males. The most common clinical manifestation of HPV infection is genital warts. In the United States, an estimated 1.4 million people have genital warts, and 500,000 to 1 million new cases occur each year.

The development of cervical cancer is preceded by persistent HPV infection. Infection with HPV 16 or 18 may progress to high-grade cervical dysplasia within three years and to cancer within a 10-year period. Cervical cancer remains a major cause of morbidity and mortality in women in the U.S. In 2002, cervical cancer was the tenth most common cancer in women and the second most common cancer in women in their reproductive years. An estimated 9,710 new cases of cervical cancer and 3,710 cervical cancer deaths occurred in the U.S. in 2006.

South Carolina has one of the highest incidence and mortality rates due to cervical cancer in the United States. The incidence of cervical cancer in 2002 was greater in South Carolina than the United States., and the S.C. age-adjusted mortality rate was the sixth highest in the U.S. for the 2000-2003 period. Racial disparities continue to play a role in the high rates of cervical cancer incidence in South Carolina, with black and Hispanic women having significantly higher rates than white women. Mortality due to cervical cancer in South Carolina is also significantly

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higher for black women than white women. The excess mortality in black women is related to the diagnosis at later stages of cervical cancer.

The cost of treatment of HPV genital warts as well as screening, follow-up, and treatment of HPV precancerous disease and HPV-induced malignancies makes HPV infection the second most costly sexually transmitted infection after HIV. The annual total direct cost for the treatment of anogenital warts for all age groups in the United States is estimated at \$167.4 million. The annual direct HPV-attributable costs for follow-up of abnormal Pap test results and for treatment of related neoplasia is estimated at \$3.4 billion to \$3.6 billion among women of all ages. In addition, the estimated annual cost of treating the expected 12,800 cases of invasive cervical cancer that occur each year is \$146.4 million.

Two prophylactic HPV vaccines have been developed. The first one is a quadrivalent vaccine that prevents infection with two low-risk HPV types, 6 and 11, and two high-risk types, 16 and 18. This vaccine has received FDA approval. The second is a bivalent vaccine that has been shown in clinical trials to prevent infection with the two high-risk HPV types, 16 and 18. At the time of this writing, FDA approval is pending for this vaccine. Both vaccines are expected to prevent 70 percent of cervical cancers. In addition, the quadrivalent vaccine will also prevent infection with HPV 6 and 11, which cause 90 percent of genital warts. Both HPV vaccines are subunit virus-like particle (VLP) vaccines that do not contain live virus vaccines and thus cannot produce HPV disease.

The Advisory Committee for Immunization Practices (ACIP) and the Centers for Disease Control (CDC) recommend that HPV immunization be administered at the 11-12 year old visit. This recommendation appears in the "2007 Recommended Immunization Schedules for Persons 0-18 Years". However, at the physician's discretion, it can be administered to girls as young as 9 years of age. One of the reasons for choosing the 11-12 year old visit is that HPV vaccine efficacy is based on preventing infection with HPV. Therefore, it is important to administer the vaccine before sexual activity begins. According to 2005 data from the Youth Risk Behavior Surveillance System conducted by CDC, of the female high school students interviewed, 3.7 percent had sexual intercourse before age 13, 62.4 percent had sexual intercourse by grade 12, and 14.3 percent had sexual intercourse with at least four persons by grade 12. In addition, 37.2 percent of sexually active male and female high school students had not used a condom at last sexual intercourse.

Some important points to remember about prophylactic HPV vaccines are the following: 1) vaccination will not prevent infection with the HPV types not contained in the vaccine; 2) vaccination will not cause regression of HPV infection caused by HPV types in the vaccine if the infection is already present at the time of vaccination; 3) individuals with impaired immune defenses (e.g. use of immunosuppressive therapy, a genetic defect, human immunode-

ciency virus [HIV] infection, or other causes) may have reduced antibody response to active immunization and decreased protection from the vaccines; and 4) adolescent girls and women who have been or are sexually active should continue to receive routine Pap testing because the HPV vaccine does not provide protection against several high-risk HPV types that cause 30 percent of cervical cancers.

The series of three immunizations will cost \$360, a significant financial barrier. While several private health insurance plans in South Carolina, including State Health Plan and BlueChoice, will provide coverage for the HPV vaccine, many economically disadvantaged adolescents do not have such coverage. The ACIP has approved a resolution to add the quadrivalent HPV Vaccine to the Vaccine for Children Program (VFC). This resolution is extremely important in providing HPV vaccine to many economically disadvantaged adolescents. However, VFC coverage of vaccines is limited to children 18 and under who are uninsured, underinsured or Medicaid eligible and to children of Native American or Alaskan ancestry. To qualify, the children must receive immunizations through a Federally Qualified Health Center (FQHC) or Rural Health Clinic (RHC). While South Carolina often has additional federal and state funding for vaccines through the Vaccine Assurance for All Children (VAFAC) program, which provides vaccines free of charge for children through the age of 18 who are underinsured (i.e., insurance plan does not cover vaccine), these funds are not available at this time. One alternative for provision of HPV vaccine for underinsured children is referral of these children to FQHCs and Community Health Centers (CHCs). Another alternative is having the patient bear the cost of the immunization. Despite these barriers, the VFC program that is administered through South Carolina will provide funding for the HPV vaccine for a significant number of adolescents. Any health care provider, including those involved in pediatrics, gynecology, family medicine, or internal medicine who would like to be able to administer HPV vaccine to VFC-eligible adolescents may enroll in the S.C. program by contacting the DHEC Immunization Division at (800) 277-4687.

Conclusions. HPV infection in women and men is very common. HPV infections can lead to the development of cervical neoplasia and cancer. HPV disease is one of the most common and costly sexually transmitted diseases. The HPV vaccine is highly effective in preventing infection with HPV and will prevent 70 percent of cases of cervical cancer in the immunized population. Vaccinating adolescent girls before sexual debut will result in a greater impact of the vaccine on the prevention of cervical cancer as well as on decreasing the spread of HPV types contained in the vaccine within the population of sexually active persons.

Year-to-Date Summary of Selected Reportable Conditions - January 1, 2007 - March 28, 2007

Condition	Confirmed	Probable	Total
Animal Bite— PEP Recommended	74	0	74
Aseptic meningitis	17	1	18
Brucellosis	2	0	2
Campylobacteriosis	46	1	47
Ciguatera fish poisoning	0	0	0
Cryptosporidiosis	10	0	10
Cyclosporiasis	0	0	0
Dengue Fever	0	1	1
Ehrlichiosis- human granulocytic	0	0	0
Ehrlichiosis- human monocytic	0	0	0
Ehrlichiosis- human- other&unspec	0	0	0
Encephalitis- West Nile	0	0	0
Enterohem. E.coli O157:H7	0	0	0
Enterohem.E.coli shigatox+- ?serogrp	0	0	0
Enterohem.E.coli- shigatox+- non-O157	0	0	0
Giardiasis	10	0	10
Group A Streptococcus- invasive	21	0	21
Group B Streptococcus- invasive	2	0	2
Haemophilus influenzae- invasive	12	0	12
Hemolytic uremic synd- postdiarrheal	1	0	1
Hepatitis A- acute	3	0	3
Hepatitis B- acute	20	1	21
Hepatitis B virus infection- chronic	71	36	107
Hepatitis C- acute	0	0	0
Hepatitis C Virus Infection- past or present	638	288	926
Hepatitis Delta co- or super-infection- acute	0	0	0
Hepatitis E- acute	0	0	0
Influenza- human isolates	39	0	39
Kawasaki disease	0	0	0
Legionellosis	3	1	4
Listeriosis	0	0	0
Lyme disease	2	0	2
Malaria	0	0	0
Mumps	0	0	0
Neisseria meningitidis- invasive (Mening. disease)	3	0	3
Pertussis	12	8	20
Rocky Mountain spotted fever	0	4	4
S. aureus- coag+- meth- or oxi- resistant (MRSA)	1	0	1
Salmonellosis	126	2	128
Shiga toxin-producing Escherichia coli (STEC)	0	0	0
Shigellosis	9	0	9
Strep pneumoniae- invasive	96	0	96
Streptococcal disease- invasive- other	5	0	5
Tetanus	0	0	0
Toxic-shock syndrome- staphylococcal	0	0	0
Varicella (Chickenpox)	243	152	395
Vibrio parahaemolyticus	1	0	1
Vibrio spp.- non-toxigenic- other or unspecified	1	0	1
Vibrio vulnificus infection	0	0	0
West Nile Fever	0	0	0
Yersiniosis	2	0	2

Epi-Notes

Division of Acute Disease Epidemiology
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FOR DISEASE REPORTING**

For immediately reportable conditions, call your local county health department or, for after-hours, call 1-888-847-0902. Routine reports may be phoned in to your local health department or mailed on a completed DHEC DISEASE REPORTING CARD (DHEC 1129). Local

county health department numbers are listed on the Official List of Reportable Conditions. For a copy of the current Official List of Reportable Conditions, call 803-898-0861 or visit www.scdhec.gov/health/disease/index.htm

THE EPI NOTES NEWSLETTER IS NOW AVAILABLE ON LINE AT

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Bureau of Disease Control

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