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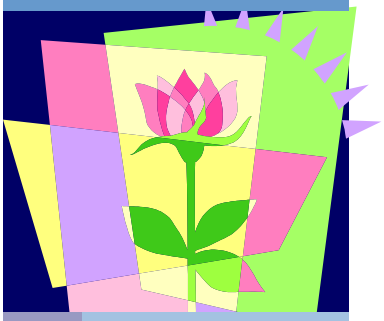


OHIO DEPARTMENT OF HEALTH
BUREAU OF INFECTIOUS DISEASE CONTROL

ODH Infectious Disease Quarterly

Tick-borne Infectious Diseases

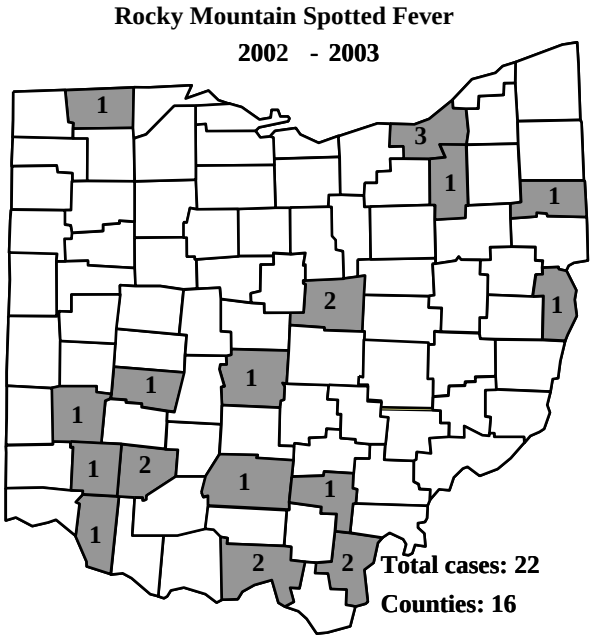
by Bob Restifo, MS, Chief, Vector-borne Disease Program

Now that spring has arrived, ticks are active again. People working and playing outdoors in wooded and weedy areas will be exposed to ticks, and possibly, tick-borne diseases, such as Rocky Mountain spotted fever, Lyme disease and Ehrlichiosis.

Rocky Mountain Spotted Fever

Although Rocky Mountain spotted fever (RMSF) has been reported from 71 Ohio counties, almost half (44 percent) of all cases were from three: Clermont, Franklin and Lucas counties. From 1956-2003, 765 cases were reported in Ohio, including nine deaths since 1964, with one in the past five years. In 2003 there were nine reported cases; this is slightly below Ohio's average of 16 cases per year. The geographical location of RMSF cases in 2002 and 2003 are shown in the figure below. The incidence of RMSF is seasonal, with dates of onset between April and July.

Heightened awareness of tick-borne diseases has probably contributed to a decrease in the case rate over the past several years. However, ticks that transmit RMSF are still abundant in Ohio; therefore the risk of infection has probably not changed. For those with RMSF, the risk of fatal infection is associated with a delay in seeking medical attention after symptoms appear. Late diagnosis can also contribute to this untoward outcome.



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Tick-borne Infectious Diseases—continued

Symptoms

Common symptoms include: sudden onset of fever, headache, and muscle aches, usually followed by a maculopapular rash, first appearing on the arms and legs, and then spreading to the trunk, palms, and soles. The incubation period for RMSF is two to 14 days after tick contact or bite. Early RMSF may be confused with ehrlichiosis, meningococcemia and enteroviral infections, among others.

The Causative Bacterium

RMSF is caused by the bacterium, *Rickettsia rickettsii*. Other species of *Rickettsia* cause various types of typhus.

The Vector

In Ohio, RMSF is transmitted by the American dog tick, *Dermacentor variabilis*. It is the most abundant and widespread Ohio tick species submitted to the Vector-borne Disease Program (VBDP) for identification and testing. *Dermacentor variabilis* constituted 892 of 1,033 (86.3 percent) of the total number of ticks identified in Ohio in 2003; this species was submitted from 74 of Ohio's 88 counties. *Dermacentor variabilis* is not particular with respect to the type of mammal upon which it feeds.

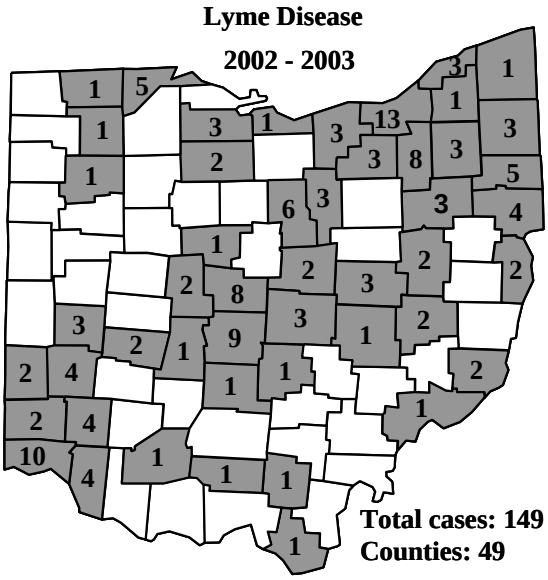
Reporting

Suspected cases of RMSF should be reported by the end of the work week. Individual cases must be reported to the appropriate local health agency. Local health agencies then report cases via the Ohio Disease Reporting System (ODRS) to ODH. *The Tick-borne Rickettsial Disease Case Report Form (CDC 55.1)* must be completed and sent to: Vector-borne Disease Program, 900 Freeway Drive North, Columbus, Ohio 43229 (Attention: Kim Winpisinger). Also, it is important that the geographical location of the tick exposure is recorded.

Lyme Disease

A total of 873 Ohio cases of Lyme disease were reported to the Centers for Disease Control and Prevention (CDC) from 82 Ohio counties during the period of 1984-2003. In 2003, 61 cases were reported. Of the 149 cases reported during 2002-2003, 27 (18.1 percent) involved out-of-state tick exposure, 29 (19.5 percent) involved in-state tick exposure and in 93 cases (62.4 percent) the location of tick exposure was unknown. The geographical locations of reported cases of Lyme disease are shown in the figure at lower right.
In addition, 263 suspect cases reported to VBDP in 2002-2003 were not reported to CDC because: (1) they did not meet the epidemiological case criteria established by CDC; (2) there was insufficient information provided in the case report; or (3) because the physician ultimately chose a different final diagnosis.

Lyme disease has not been proven to be endemic in Ohio because its causative organism has never been isolated or identified in a resident animal or tick. Hundreds of *Ixodes* ticks and Ohio rodents have been tested for the responsible organism; all of these tests have been negative. However, this does NOT mean that Lyme disease cannot be acquired in Ohio. It is noteworthy that 18 blacklegged ticks (*Ixodes scapularis*), the vector of Lyme disease in the eastern United States, have been found in Ohio. However, these ticks were probably brought into the state upon migratory birds or other travelers. The actual number of these vector ticks that are brought into Ohio each year is unknown.



Tick-borne Infectious Diseases—continued

Symptoms

Lyme disease has early and late manifestations; early treatment can usually prevent the progression to late-stage disease. Early Lyme disease can manifest with a "bull's-eye" rash, called erythema migrans. This lesion begins as a red macula or papule that expands over days or weeks, resulting in a large, red, ring-like rash, sometimes with central clearing. The rash often begins at the site of the tick bite. Symptoms of early Lyme disease include fatigue, headache, neck pain, stiffness in muscles or joints, fever and swollen glands. Unfortunately, diagnosis can be hampered by the fact that 20-25 percent of those with early Lyme disease do not develop the characteristic rash. Late Lyme disease can have a wide range of joint, cardiac and neurological manifestations and complications. Some people without early treatment or in whom the diagnosis was not made can develop late disease months or even years after infection. The incubation period for Lyme disease is three to 30 days.

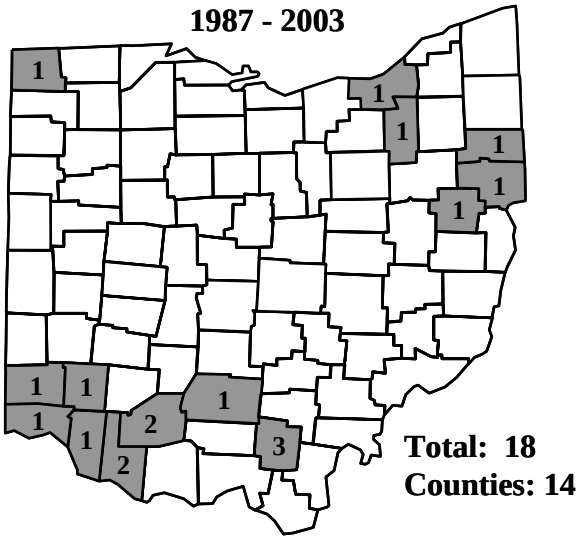
The Bacterium

Borrelia burgdorferi is the causative agent of Lyme disease. It is a spirochete, similar to the causative agent of syphilis, and closely related to the species of *Borrelia* that causes relapsing fever.

The Vector

Lyme disease is transmitted in the eastern United States by the black-legged tick, *Ixodes scapularis*. No other tick species have been proven to be vectors of Lyme disease in humans in the eastern United States. As noted, 18 black-legged ticks have been found in Ohio, but none of these were infected with *Borrelia burgdorferi;* see the figure below. Finally, there is some evidence that the rabbit tick, *Ixodes dentatus*, may be a vector of Lyme disease in rabbits.

Blacklegged tick, *Ixodes scapularis*
1987 - 2003



Total: 18
Counties: 14

Reporting

Lyme disease cases should be reported by the end of the next business day. Individual cases must be reported to the appropriate local health agency. Local health agencies then report cases via Ohio Disease Reporting System (ODRS) to ODH. Local health jurisdictions must complete the signs and symptoms section of the *Lyme Disease Case Report Form* and send the form to: Vector-borne Disease Program, 900 Freeway Drive North, Columbus, Ohio 43229 (Attention: Kim Winpisinger). Again, it is important that the geographic location of tick exposure is recorded.

Ehrlichiosis

Ehrlichiosis is an emerging infectious disease, but it has probably existed for a long time without being recognized. In 1998, deer in four southern Ohio counties—Adams, Gallia, Jackson and Lawrence—were found to be infected with the bacterium that causes Human Monocytic Ehrlichiosis (HME). At that time, a seroprevalence study of 133 deer revealed that 11 (8.3 percent) were positive for the causative organism of HME. In 2002, three human cases of Ehrlichiosis in Ohio were reported to CDC. There was a single case in each of the following counties: Ashtabula, Cuyahoga and Geauga. In 2003, a total of eight human cases were reported to CDC from the following counties: Athens, Clermont, Geauga, Jackson, Montgomery, Portage and Summit.

Tick-borne Infectious Diseases—continued

The Bacterium

HME is caused by the bacterium, *Ehrlichia chaffeensis*. It is a rickettsia-like organism—an extremely small intracellular parasite of selected white blood cells (monocytes and macrophages). The bacterium was first isolated in 1986 from the blood of a US Army reservist at Fort Chaffee, Ark., and was subsequently named *Ehrlichia chaffeensis* after the locality of the first isolate.

The Vector

HME is transmitted by the Lone Star tick, *Amblyomma americanum*. Within the past two years, populations of this tick have been discovered in 28 Ohio counties. The geographical distribution of *Amblyomma americanum* is shown in the figure below.

Symptoms

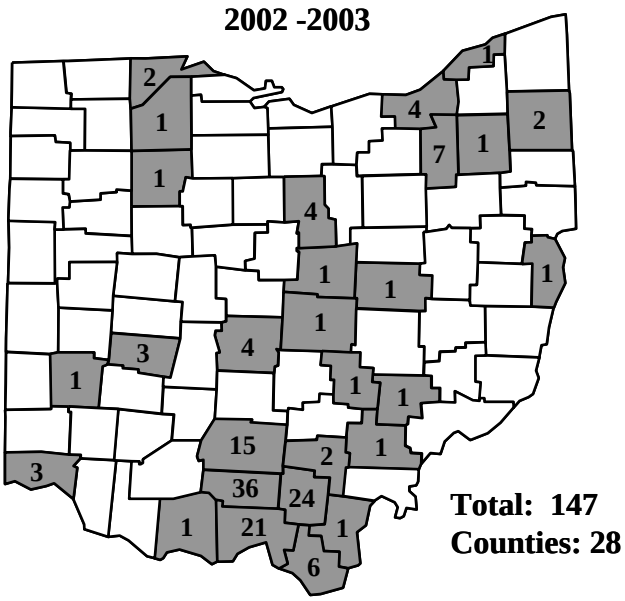
The onset of symptoms of HME occurs between one and 21 days following inoculation and may resemble the symptoms of RMSF. The spectrum of this disease ranges from illness so mild that no medical attention is sought to a severe, life-threatening disease entailing respiratory failure, renal failure and meningoencephalitis. The usual early clinical features include high fever, headache, malaise, muscle aches, vomiting and loss of appetite. A rash similar to that seen in cases of RMSF is present in about 20 percent of cases. The bacterium invades white blood cells and leukopenia as well as thrombocytopenia are common. The disease is most severe in the elderly and in those with immune deficiencies.

Reporting

Cases of HME should be reported by the end of the work week. Individual cases must be reported to the appropriate local health agency. Local health agencies then report cases via the ODRS to ODH. The *Tick-borne Rickettsial Disease Case Report Form (CDC 55.1)* must be completed and sent to Vector-borne Disease Program, 900 Freeway Drive North, Columbus, Ohio 43229 (Attention: Kim Winpisinger).

Amblyomma americanum

2002 -2003



Total: 147
Counties: 28

Removing Ticks

The bacteria that cause RMSF, Lyme disease and Ehrlichiosis are transmitted in the tick's saliva while it is feeding. However, feeding begins a relatively long time after attachment, and the risk of infection prior to feeding is low. For this reason, early detection and correct removal of the tick will help to prevent infection.

Care should be exercised when removing ticks. If squeezed, a tick may involuntarily inject bacteria, like a "mini-syringe." NEVER attempt to "burn off" a tick with a match, as this will cause the tick to burst and inject more bacteria into the feeding site. Secretions and tick feces may also be infective, so it is best to avoid touching the tick.

Grasp the tick as close to the skin as possible using fine-tipped tweezers or with fingers shielded with tissue or rubber gloves. Pull gently but firmly straight out until the tick pulls free. Wash the bite site with soap and water and apply an antiseptic. KEEP THE TICK ALIVE. Place it in a pill vial or film canister and attach a completed tick submission form indicating the county where the tick was picked up and the date it was found. It is also very important to include a small moistened piece of paper towel or napkin (one drop of

water to wet the item is sufficient) in the container with the tick. Without this, or with too much water, the tick may die prior to reaching the VBDP lab for testing.

The VBDP lab can test only live ticks. However, all ticks, dead or alive, will be identified. For identification and testing, send the tick to the Vector-borne Disease Program's Tick Identification and Testing Service at the address below. A tick submission form is provided in this publication. Please make copies as necessary.

Personal Protection

The risk of exposure to ticks can be greatly reduced by practicing basic personal protection. When entering tick habitat (high grass and weeds), wear light-colored long pants and tuck pant legs into sock tops. This may not be fashionable, but it allows for visual exams every half hour or so to remove ticks before they attach to skin. Applying insect repellent to pant legs and sock tops helps. During tick season (April through July), complete a thorough visual exam in the evenings. Examine children and especially dogs that have been running loose and in tick habitat.

Tick Identification & Testing Service

Tick identification and testing is currently available through the Vector-borne Disease Program. The purpose of this service is to supply correct identification of ticks, report the results of pathogen testing and provide information about diseases transmitted by ticks in Ohio. Proper tick identification is essential to determine the potential risk of infection associated with a particular disease. For example, the American dog tick is a vector of RMSF, but NOT Lyme disease. Tick testing at the Vector-borne Disease Program for RMSF and Lyme disease is not diagnostic of human illness but indicates risk if the tick was infected.

For additional information or comments, call or e-mail us at: zoonoses@odh.ohio.gov

Vector-borne Disease Program, ODH,
900 Freeway Drive North
Columbus, Ohio 43229

Phone: 614-752-1029;

FAX: 614-752-1391

Herpes Simplex Virus, Type 2, by Jeff Vasiloff, MD, MPH, Chief HIV/STD Prevention and the STD Prevention Team

Herpes Simplex Virus Type 2 (HSV-2) is the most frequent cause of genital ulcer disease (GUD) worldwide. In the United States an estimated 1.6 million new cases occur each year (1). Further, seroprevalence studies indicate that 22 percent of American adults possess antibodies to HSV-2 confirming past infection (2).

HSV-2 infection occurs most commonly following the inoculation of virus upon the recipient's skin or mucosal surface by sexual contact. After inoculation, the virus replicates in the epithelium and infects sensory nerve endings. Then the virus travels retrograde up the nerves to the dorsal spinal ganglia where it remains for the lifetime of the individual in a latent state punctuated by frequent reactivation of viral replication.

Primary HSV-2 infection can be associated with symptoms or may be asymptomatic. After the resolution of primary infection, recurrent infections can occur by reactivation of the latently infected spinal ganglia. These recurrent infections can be symptomatic or asymptomatic (in the latter case, reactivation is detected by the shedding of HSV-2 virus). It is believed that reactivation of HSV-2, with asymptomatic viral shedding, occurs frequently in most infected individuals, and this is independent of whether or not episodic clinical disease supervenes.

The Manifestations of HSV-2 Infection

The manifestations of HSV-2 infection vary. About 20 percent of infected individuals have classic symptomatic genital herpes; however, another 20 percent have no symptoms. The remaining 60 percent have nonspecific symptoms; these individuals have so-called atypical genital herpes and are often not recognized or diagnosed. In a recent study of more than 5,000 affluent suburbanites in Atlanta, Baltimore, Boston, Chicago, Dallas and Denver, 25.5 percent had serum antibodies to HSV-2 (3). Of the 1,387 seropositive individuals, only 12 percent knew they were infected, which confirms the prevalence of asymptomatic and atypical genital herpes in a population often not thought to be at high risk for sexually transmitted diseases.

Classic herpes genitalis is characterized by painful genital vesicles and ulcers. Other symptoms include dysuria, inguinal lymphadenopathy, urethral discharge and cervical bleeding with vaginal discharge. Among those with classic genital herpes, primary infection can be followed by an average of four to five recurrences during the first year; the frequency of recurrences often gradually decreases with time.

The Spread of HSV-2

The spread of HSV-2, especially among young sexually active populations, is facilitated by two factors. First, the large population of people with no symptoms or only nonspecific symptoms often do not know they are infected. Second, asymptomatic viral shedding occurs frequently between clinical episodes in those with such manifestations and occurs frequently in those who lack clinical disease. In this way, the spread of HSV-2 is similar to that of HIV. Individuals with HIV may have no symptoms or only nonspecific symptoms for the first decade of infection. The status of not feeling ill may lead to the belief that infection is not present, and further, that testing and safer sex precautions are not relevant.

Other HIV-HSV-2 Interrelationships (4)

The risk of having HIV is two- to fourfold higher among those who have HSV-2. It is known that mucosal disruptions caused by HSV-2- mediated genital ulceration increase the susceptibility of animal models exposed to HIV. Further, herpetic ulcerations foster an influx of CD4+ lymphocytes, the target cell of HIV attachment. Thus, HSV-2 infection may increase the risk of HIV acquisition in humans. Similarly, in those with HIV, concurrent HSV-2 infection has been shown in some studies to increase HIV shedding. Thus, HSV-2 co-infection may increase the risk of HIV transmission to an uninfected partner. Finally, HSV-2 infection is an AIDS-defining illness and among those with severe immunodeficiency, HSV-2 infection can be more severe than in immunocompetent individuals.

Herpes Simplex Virus Type 1 (HSV-1)

HSV-1 is very closely related to HSV-2. HSV-1 infection usually results in oral cold sores and

fever blisters; however, HSV-1 can cause genital herpes as a result of orogenital contact. Genital infections due to HSV-1 appear to be more frequent among men who have sex with men (MSM) than among heterosexual individuals. In those with genital herpes due to HSV-1, recurrent clinical episodes appear to be less frequent than with HSV-2 infections.

Herpes Associated with Pregnancy

In the United States, as many as 2,000 neonates per year acquire HSV infection through pregnancy and childbirth, in most cases secondary to exposure to infected genital secretions at delivery. The risk of transmission is low among mothers with recurrent HSV infection, higher among mothers who develop primary infection during pregnancy, and highest among those who develop primary infection during the last trimester.

HSV-2 causes about 70 percent of neonatal infections. HSV-1 causes about 30 percent of neonatal cases and in general, results in milder complications and sequellae compared to HSV-2. HSV-infected neonates may present with skin/eye/mouth (SEM) involvement, encephalitis or disseminated disease. The mortality rates for encephalitis and disseminated disease—despite antiviral treatment—remain at 15 percent and 57 percent, respectively. Caesarean section is the primary strategy used to prevent neonatal infection in women with active lesions (5).

Laboratory Diagnosis

If lesions are present, viral culture can be performed; however, false negatives may occur unless sufficient quantities of virus is obtained, which can be difficult in some cases. Serologies have been used for years to detect HSV antibodies; however, until recently, commercial assays could not distinguish between HSV-1 and HSV-2. Newer assays detect type-specific antibodies because of antigenic differences of glycoprotein on the virions' outer envelopes. Just as in HIV antibody testing, HSV serologies may be negative in the weeks immediately following primary infection. Thus, a repeat test after four to six weeks (if the initial test is negative) is warranted. A multiplex polymerase chain reaction (M-PCR) assay is now also available in select centers but only for investigational purposes. This assay identifies the three most common causes of genital ulcers in the United States: HSV, *Treponema pallidum* (syphilis), and *Haemophilus ducreyi* (chancroid) (6).

Treatment

Three antiviral agents have been approved to treat HSV genital infections in immunocompetent hosts: valacyclovir, acyclovir and famicyclovir. Treatment can be episodic—associated with primary or recurrent vesicular eruptions—or suppressive. Suppressive therapy decreases the frequency of recurrences and also decreases subclinical viral shedding that may be important in reducing the transmission of the virus (see below).

Prevention

The Centers for Disease Control and Prevention (CDC) provide the following guidance with respect to prevention messages for individuals with genital herpes (7):

- Patients who have genital herpes should be educated about the natural history of the disease, with emphasis on the potential for recurrent episodes, asymptomatic viral shedding and attendant risks of sexual transmission.

- Patients experiencing a first episode of genital herpes should be advised that suppressive and episodic antiviral therapy is available and is effective in preventing or shortening the duration of recurrent episodes.

- All persons with genital HSV infection should be encouraged to inform their current sex partners that they have genital herpes and to inform future partners before initiating a sexual relationship.

- Persons with genital herpes should be informed that sexual transmission of HSV can occur during asymptomatic periods. Asymptomatic viral shedding is more frequent in genital HSV-2 infection than genital HSV-1 infection and is most frequent in the first 12 months of acquiring HSV-2.

- Patients should be advised to abstain from sexual activity with uninfected partners when lesions or prodromal symptoms are present.

- Latex condoms, when used consistently and correctly, can reduce the risk for genital herpes when the infected areas are covered or protected by the condom. A recent prospective study suggests that condoms have been effective in preventing transmission from men to women.

- Sex partners of infected persons should be advised that they might be infected even if they have no symptoms. Type-specific serologic testing of asymptomatic partners of persons with genital herpes can determine whether risk for HSV acquisition exists.

- The risk for neonatal HSV infection should be explained to all patients, including men. Pregnant women and women of childbearing age who have genital herpes should inform their providers who care for them during pregnancy as well as those who will care for their newborn infant. Pregnant women who are not infected with HSV-2 should be advised to avoid intercourse during the third trimester with men who have genital herpes. Similarly, pregnant women who are not infected with HSV-1 should be counseled to avoid genital exposure to HSV-1 during the third trimester (e.g., cunnilingus with a partner with oral herpes and vaginal intercourse with a partner with genital HSV-1 infection).

Asymptomatic persons diagnosed with HSV-2 infection by type-specific serologic testing should receive the same counseling messages as persons with symptomatic infection. In addition, such persons should be taught about the common manifestations of genital herpes. Antiviral therapy is not recommended for persons who do not have clinical manifestations of infection.

Possible Role of Suppressive Therapy in Prevention

It has been recently reported that suppressive therapy of HSV-2 may decrease sexual transmission (8). In this study, 1,484 heterosexual, monogamous, immunocompetent, serodiscordant couples were enrolled. That is, one partner had serum antibodies to HSV-2 while the other did not and was therefore susceptible to infection. All infected partners not only had antibodies to HSV-2, but also had histories of clinically apparent herpes genitalis.

The couples were randomly divided into two groups. In one group, the infected partner was given a daily placebo while in the other group, the infected partner was given a daily dose of 500 mg of valicyclovir. All couples were followed eight months and each couple had a median of about 50 sexual encounters during the study period. Both groups were counseled on safer sex practices and were given free condoms.

At the end of eight months, acquisition of HSV-2 infection was 3.6 percent in the placebo group and 1.9 percent in the valicylovir group ($p = .04$). The risk of acquiring HSV-2 infection in the treatment group was reduced by 48 percent compared to controls. Viral shedding was also decreased in the treatment group.

While additional research is needed, suppressive antiviral treatment of HSV-2-infected individuals may have a role in sexually transmitted disease prevention and control.

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Summer Fun and Waterborne Disease Outbreaks

by MaryKay Parrish, MS, Bureau of Infectious Disease Control, and Steven Binns, MA, RS Administrator, and Barry Rice BS, both of the Bureau of Environmental Health

It is warm and sunny and summer is here at last. You yawn and stretch, but something is not quite right. Your stomach is rumbling. You have an urgent need to seek out the nearest restroom. You think it might be some sort of food poisoning, but the rest of your family has eaten the same thing as you have all week and no one else is sick…

When you call the doctor, she says, "I've seen several people with the same symptoms this week." Your visit to the doctor is interrupted by no less than four trips to the bathroom. The doctor takes a standard history plus a food and activity history and a stool specimen.

Results of the stool specimen are called to your home the next day. The answering machine picks up, because, yet again, you are indisposed. You listen to your messages. You have *Cryptosporidium*.

You turn to the Internet for information (http://www.cdc.gov), and learn that *Cryptosporidium* is a microscopic parasite that causes diarrhea. It is found in the stool of infected animals and humans. You can become infected after accidentally swallowing the parasite. Food, soil and water are all potential suspects.

You start thinking, water…you are an avid swimmer, in fact you are the only member of your family to stop regularly at the pool on the way home from work…but your pool is crystal clean, and there is always enough chlorine…

Restless and channel surfing you hear the headlines, "*Cryptosporidium* strikes Ohio pool club members. Details at 11."

It couldn't be YOUR club, or could it?

National Scene

Nationally, the Centers for Disease Control and Prevention (CDC) has seen an increase in waterborne illnesses among swimmers. Between 1999 and 2000, the most recent years with published statistics, 59 outbreaks from 23 states, involving 2,093 persons, were attributed to recreational water exposure. Recreational waterborne disease outbreaks (WBDO) involving gastroenteritis doubled since the previous reporting period (from 18 to 36 outbreaks). These outbreaks were most frequently associated with *Cryptosporidium parvum* (68 percent) from treated water venues (e.g., swimming pools and interactive fountains) and by *Escherichia coli* O157:H7 (21 percent) from freshwater venues. The nationwide increase in the number of outbreaks probably reflects improved surveillance and reporting at the local and state level, as well as a true increase in the number of WBDOs. (1).

The Ohio Scene

Ohio has not seen a clear trend in the number of waterborne illnesses among swimmers. Between 2000 and 2004, Ohio investigated eight recreational WBDOs within six counties. Overall, the responsible organisms in Ohio outbreaks differed from what has been seen nationally. Agents seen in Ohio recreational WBDOs included *Pseudomonas* (n=3), chlorine gas exposures (n=2), *Cryptosporidium* (n=1), *Plesiomonas shigelloides* (n=1) and swimmer's itch (n= 1). Six of the outbreaks were associated with swimming pools or spas while two were associated with swimming in untreated freshwater. A total of 947 Ohioans were affected in the outbreaks, and the number involved per outbreak ranged from two to 844 (2).

The Epidemiologic Approach in WBDOs

The unit of analysis for the WBDO surveillance system is an outbreak, not an individual case of a waterborne disease. Two criteria must be met for an event to be defined as a WBDO. First, two or more persons must have experienced a similar illness after either ingestion of drinking water or exposure to water encountered in recreational or occupational settings. This criterion is waived for single cases of laboratory-confirmed primary amebic meningoencephalitis and for single cases of chemical poisoning if water quality data confirm chemical contamination (1). Second, epidemiologic evidence must implicate water as the probable source of the illness. In Ohio, waterborne outbreaks are Class A(2) reportable diseases under Ohio Administrative Code 3701-3-02. Class A(2)

herpes. New Engl J Med. 2004; 350: 11-20.

means that they are required to be reported by the end of the next business day after the detection of a case, a suspected case or a confirmatory positive laboratory result.

Diseases Caused by WBDOs That Have Occurred in Ohio

There are many agents capable of causing WBDOs. The six agents that have been found in Ohio WBDOs in 2000-2004 are discussed below. Other causes of WBDOs are: *Campylobacter*, *Giardia,* Hepatitis A, Norovirus, Rotavirus, *Salmonella, Shigella*, small round structured viruses, *Vibrio* and carbon monoxide poisoning.

Cryptosporidium parvum

Known colloquially as "crypto," this protozoan causes cryptosporidiosis. While this infection can be asymptomatic, common symptoms include diarrhea, abdominal cramps, fatigue, nausea, vomiting, anorexia and weight loss. Fever and vomiting are common in children and in this population, cryptosporidiosis is confused with viral gastroenteritis. Among immunocompetent individuals, cryptosporidiosis is self-limited; however in those with HIV, severe chronic diarrhea, malnutrition and dehydration can occur and in some, death.

People become infected by swallowing contaminated water or food, or by swallowing the organism after transfer by the fingers and hands to the mouth after touching contaminated surfaces such as lounge chairs, picnic tables, bathroom fixtures or changing tables. Illness usually develops two to 10 days after ingesting the oocysts. *Cryptosporidium* oocysts cannot be seen without a microscope and are highly resistant to chlorine and bromine, disinfectants commonly used in pools.

Cryptosporidium is found in the stool of infected people and animals; cryptosporidiosis is diagnosed through the examination of three or more stool samples. Multiple stools are required because the organism is often shed only intermittently (3). People who have diarrhea are encouraged to contact their physician for diagnosis. In outbreak situations, the Ohio Department of Health Laboratory (ODHL) performs stool specimen testing.

***Escherichia coli* O157:H7**

The O157:H7 strain is one of hundreds of strains of the bacterium *Escherichia coli*. Most of these strains are harmless, while others cause gastrointestinal infections by various mechanisms. *E. coli* O157:H7 releases toxins called Shiga-like toxins. These toxins can cause severe hemorrhagic colitis with severe abdominal pain, although less severe non-bloody diarrhea can alternately occur in some people, and a few have no symptoms. Fever can be absent in milder cases. The illness usually lasts five to 10 days. Children under 5 years of age are at risk for developing Hemolytic Uremic Syndrome (HUS). In HUS, hemolytic anemia, renal failure and death can occur.

E. coli O157:H7 lives in the intestines of healthy cattle and infected people. People can acquire it from ingesting food or water contaminated with the bacterium. *E. coli* O157:H7 infections are diagnosed by culturing the bacterium from stool or detecting the toxin (4). Again, people who have diarrhea are encouraged to contact their physician for diagnosis.

It is important for diagnosing physicians to remember that labs do not routinely perform *E. coli* O157:H7 cultures. These cultures need to be specifically requested. The culturing of organisms that cause gastrointestinal disease is important because it facilitates the linkage of contaminated food and water specimens with clinical isolates. In outbreak situations, ODHL provides stool specimen testing and can "genetically fingerprint" *E. coli* O157:H7 by pulse field gel electrophoresis (PFGE).

Pseudomonas aeruginosa

Pseudomonas aeruginosa is a bacterium that can cause dermatitis (hot tub folliculitis), otitis externa (swimmer's ear) and conjunctivitis in the setting of recreational water exposure. Hot tub folliculitis can become secondarily infected by other organisms, such as *Staphylococcus aureus.* Symptoms may occur as soon as eight hours or as long as five days after exposure. Rashes usually clear spontaneously in two to 10 days, rarely recur and heal without scarring. Potential sequelae include desquamation and hyperpigmentation. *Pseudomonas aeruginosa* can be cultured from fresh pustules, ears or eyes. It can also be cultured from water samples from pools, hot tubs and whirlpools. The warmer temperatures of

hot tubs and spas cause chlorine or other disinfectants to evaporate quickly. This accounts for the increased risk of infection in these venues. *Pseudomonas* is a versatile organism and can be found in soil, sinks, drains, shower-floors, carpeting, filters and even tap water

Nationally, bacterial serotype O:11 is the most commonly reported isolate for water-associated pseudomonas folliculitis (5). ODHL provides speciation and "genetic fingerprinting" of *Pseudomonas* through biochemical testing and PFGE.

Plesiomonas shigelloides

Plesiomonas shigelloides, formerly known as *Aeromonas shigelloides,* is a rod-shaped bacterium that can be found in fresh, temperate waters. This organism has been isolated from the gut of freshwater fish, shellfish, snakes, toads, dogs, cats, pigs, cattle and poultry. Most illnesses attributed to—but not definitively proven to be caused by *Plesiomonas shigelloides* have occurred from 20 to 24 hours after swimming in or ingesting contaminated water (6). Gastroenteritis is the primary infection attributed to *Plesiomonas;* the course of Plesiomonas-attributed gastroenteritis is usually mild and self-limited, although it has also been associated with a more protracted dysentery-like course lasting up to three weeks. As noted above, ODHL provides the identification of organisms responsible for foodborne and waterborne outbreaks.

Swimmer's Itch (Cercarial Dermatitis)

Cercariae, the infective form of a number of blood flukes of birds and nonhuman mammals, can cause a characteristic dermatitis. The etiology of the rash appears to be allergic and the rash develops within minutes to days after swimming in contaminated water. Symptoms include tingling, burning or itching of the skin. Small reddish papules appear within 12 hours of exposure and subside in four to seven days, although severe cases can last for weeks (7).

Chlorine Gas

Chlorine gas is a pulmonary irritant with intermediate water solubility that causes acute damage of the upper and lower respiratory tract. While chlorine gas exposure is not an infectious disease, it is a cause of concern in swimming pool environments. Chlorine-containing compounds are used as disinfectants in various forms in the pool environment. Although the use of gaseous chlorine as a disinfectant in swimming pools in Ohio has been outlawed, gas can be formed due to improper mixing of chlorine-containing compounds with other chemicals commonly found in the pool environment. The effects of exposure to chlorine gas are immediate. These effects include acute inflammation of the conjunctiva, nose, pharynx, larynx, trachea and bronchi. Irritation of the airway mucosa leads to bronchial edema as well as alveolar infiltrates and pulmonary edema. The duration of symptoms and severity of disease depends on level of exposure, age and the cardiorespiratory health of exposed individuals. The resolution of pulmonary symptoms in most individuals occurs over the course of one week to one month following exposure (8).

Public Health Action

CDC and ODH have taken public health action to decrease the number of waterborne outbreaks among swimmers. Nationally, CDC has partnered with a consortium of local and national pool associations to develop a series of health communication materials for those who attend treated recreational water venues and to staff who work at these venues. The public health message is organized into six "P-L-E-As" for healthy swimming. These materials can be found at http://www.cdc.gov/healthyswimming/. CDC has also developed a recreational water outbreak investigation toolkit for public health professionals. The waterborne outbreak investigation toolkit can be found at http://www.cdc.gov/healthyswimming/outbreak.htm.

Within Ohio, public swimming pools and spas are regulated under Chapter 3749 of the Ohio Revised Code (the swimming pool law) and Chapter 3701-31 of the Ohio Administrative Code (the swimming pool rules). Pools and spas are licensed and inspected by the local boards of health. Waterparks and aquatic facilities associated with amusement rides are regulated by the Ohio Department of Agriculture.

The rules specify requirements for the design, construction, installation and operation of pools and spas to protect the health and safety of the bathing public. Before a swimming pool or spa is installed, engineers at ODH review the plans to ensure that the circulation and filtration of water, as well as the basic design of the facility, will protect swimmers from illness and injury.

The swimming pool rules also require the pool or spa operator to test the water on a regular basis for clarity, temperature, disinfectant levels and other chemical parameters such as pH, total alkalinity, cyanuric acid and total dissolved solids.

The Bureau of Environmental Health (BEH) also tests and regulates bathing beaches. During the summer, maps and beach advisories are at http://www.odh.ohio.gov/Alerts/Alertmain.htm . For questions regarding beach monitoring, swimming pool design or operations, contact your local health agency or the BEH at (614) 466-1390.

Courses Available

CDC has developed a *New EH Initiative Swimming Pools and Spas Interactive Training CD-ROM for Environmental Health Pool Program.* This is available to local and state health departments. It has been sent free of charge to 4,000 state and local public health agencies across the United States by the Environmental Health Services Branch of CDC: http://www.cdc.gov/nceh/ehs. Additional copies can be ordered for a fee from http://www.cdc.gov/healthyswimming/swim_cd.htm.

In Ohio, there are a number of public pool operator instruction courses available including the National Pool/Spa Foundation's Certified Pool Operator course. Contact Barry Rice at (614) 466-1390 for additional information.

To investigate waterborne illnesses contact your local health agency, ODH's Bureau of Infectious Disease Control (BIDC) at (614) 466-0265, and the BEH (614) 466-1390.

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Rubella Testing in Pregnancy, by Amy Bashforth, MPA, Immunization Program

The serious consequences of rubella infection during pregnancy raise the importance of routinely verifying immunity of pregnant women. In order to effectively determine immunity of pregnant women and women of childbearing age, it is critical to understand the most effective methods of determining rubella immunity.

Health care providers should routinely determine rubella immunity for women of childbearing age. Proof of immunity may be through a record of vaccination or a positive IgG antibody serologic test (1). In a 2003 survey of American College of Obstetricians and Gynecologists Fellows, 85 percent of respondents indicated that they do routinely screen obstetric patients for rubella IgG antibody. Approximately one-quarter of respondents, however, also reported routinely screening obstetric patients for rubella IgM antibody (2). A rubella IgM antibody test is not recommended as a means of determining immunity to rubella infection and the use of this test for determining immunity may reflect a lack of understanding regarding the type of prenatal screening that should be used.

The rubella IgM test is used to diagnose acute and recent rubella infection and should not be used to determine rubella immune status. Positive IgM cases in pregnant women in a British study illustrated that problems with IgM testing that may arise for a number of reasons, including false positive rubella IgM results, limited experience with rubella diagnosis and its pitfalls (e.g., persistent specific IgM) and misinterpretation of laboratory results (3). It is also known that false positive rubella IgM results have occurred in persons with parvovirus infections, infectious mononucleosis, or rheumatoid arthritis (4).

A rubella IgM test should only be used for a pregnant woman if rubella symptoms or exposure to rubella are reported, in order to avoid problems with interpretation. The positive predictive values of rubella IgM results have declined in countries where rubella seldom occurs (3). From 1993 through 2003, no rubella cases were reported to the Ohio Department of Health. One Congenital Rubella Syndrome case was reported in 2001. The lack of circulating rubella in Ohio suggests that virtually all positive IgM results will be false positives. Few laboratories can perform rubella IgM tests adequately, given that there is little circulating rubella disease, and therefore, the tests are performed infrequently. This increases the likelihood of false positive results.

Health care providers should verify rubella immunity for all pregnant women at the earliest possible prenatal visit. A positive IgG antibody test indicates rubella immunity and it is generally assumed that the immunity was acquired before pregnancy (1). Any susceptible pregnant women should be monitored for signs of rubella, advised to avoid contact with persons with rash illnesses, and vaccinated postpartum.

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Mosquito-borne Infectious Diseases, by Bob Restifo, MS, Chief, Vector-borne Disease Program

Even after a May overnight low of 20, the mosquito larvae were happily wriggling around their water holes, munching on bacteria, algae or whatever organic material they can find, slowly growing and getting closer to the time when they will pupate and then emerge as fully functional purveyors of nuisance and disease.

Mosquitoes are a ubiquitous part of life and there is almost nowhere that they can be avoided. They are present in every habitat in which people want to be. Usually they are an annoyance, severe at times, but still just a nuisance. However, under certain circumstances, some species become more than that – they can transmit pathogens that can cause mild to severe illness, including death, to a wide variety of animals (reptiles, birds and mammals). Many parts of Ohio, and indeed the world, would be uninhabitable without some form of mosquito control. Malaria is still a serious illness in many parts of the world, affecting millions of people annually. Yellow fever, dengue and other scourges of the Americas are still around and inflicting suffering.

Mosquitoes in Ohio

Of the 163 species of mosquitoes known to occur in the state, the "spring species" bite in April and May, then the "flood water species" produce huge numbers after summer rains fill all the low spots. But the ones that produce the big numbers may not be the ones that do the real damage – the vector species that can transmit pathogens like the viruses that cause West Nile neuroinvasive disease and La Crosse and Eastern Equine encephalitides.

Mosquito Life Stages

Mosquitoes have four distinct life stages – egg, larva, pupa and adult. Eggs are deposited on a surface that will flood or directly onto standing water, either singly or in "rafts" on the surface of the water, or attached to the underside of floating vegetation. Most mosquito eggs can withstand freezing and drying, so this is the stage that "overwinters" in most species. The incubation period of eggs can be anywhere from a couple of days to several months. During the mosquito season, eggs hatch in two to three days. The first larval "instar" is so small that most people cannot see them. They feed and molt three more times into second, third and fourth instars, getting bigger with each molt. Larvae feed on organic material in the water, usually bacteria, algae and diatoms.

Although larvae have gills, they are air breathers, coming to the surface for passive gas exchange with the atmosphere. The gills are used for ion regulation within their bodies. When disturbed, they move through the water with a wriggling motion. After five or six days, the larvae molt into pupae. Pupae are usually dark colored, comma-shaped and very mobile. They do not feed. They also breathe at the surface of the water. In this stage, tissues are reorganized from those of the larva to those of the adult mosquito. In two to three days an adult mosquito emerges.

Adult mosquitoes can be differentiated from any other insect because they have two wings—not four like most insects—and long, straw-like mouths, perfectly fitted to suck fluids like plant juice or blood. They usually live two to four weeks, however some species overwinter as adults; so they can live several months. Most species do not fly more than a quarter to half a mile from their breeding sites, but some can go as far as 20 miles during their lives. Both males and females get their daily energy from sugar solutions like sap and plant juices. Only female mosquitoes require blood; they need a protein source to develop eggs. It is this activity that makes them such a nuisance and also vectors of disease. Some species prefer to feed on birds, a few feed only on amphibians and reptiles. Most species in Ohio prefer mammals. Some species do not care what they feed on – reptile, bird or mammal.

Mosquitoes That Are Vectors of Disease

Of the 163 species in Ohio, less than a dozen are vectors, that is, capable of transmitting pathogens. When female mosquitoes take a blood meal, the first thing they do after inserting their mouth parts into a capillary is to inject some saliva. This prevents the blood from coagulating and ensures a full meal. The itchy welt that results is a response to the proteins in the saliva. Pathogens can be transmitted through the saliva. In most species of mosquitoes, pathogens can be ingested with a blood meal but then do not get into the body cavity, and thus, do not get into the salivary glands. In this case, ingested pathogens cannot be passed to the next blood host. But some species are very good at passing pathogens. One cannot tell by looking at a mosquito if it is infected or infective.

Prevention of Mosquito Bites

There are some simple things people can do to reduce the risk of getting bitten. If it is not necessary to be outdoors during peak mosquito activity periods (several hours before and after dark), staying indoors is advisable. When outside during mosquito activity periods, one should wear long pants and a long-sleeved, light-colored shirt and avoid using colognes and after-shaves, as many contain chemicals that attract mosquitoes. Use repellents according to label instructions. Repellents containing 30 percent active ingredient or less on adults and less than 10 percent on children are recommended. Those containing a chemical called DEET remain effective the longest. Some of the herbal-based repellents last only a few minutes and need to be reapplied often. Apply repellents to shirt cuffs and collars, hats, pant legs and sock tops, thus reducing the amount applied directly to the skin. Adults should apply repellents to children to avoid children getting repellent into their mouths.

People should get rid of any standing water around their yard and fill in low spots so they do not hold water. Birdbaths must be emptied and refilled weekly. Keep gutters clear of debris. Remove any container that holds water. Prevent tarps covering cars, boats and other equipment from forming pockets that hold water. Put goldfish in ornamental ponds. There are some good, environmentally safe products available for homeowner use. One is a bacterium called *Bacillus thuringiensis israeliensis*, or Bti, which is available in granules, called Quick Kill, or donuts called Mosquito Dunks. Another product is an insect growth regulator called Altocid. This is actually a mosquito hormone that prevents the maturation of larvae. Finally a monomolecular surface film called Agnique is also available. This film prevents larvae and pupae from reaching the surface to breathe, thus drowning them.

Mosquito-borne Diseases in Ohio

The mosquito-borne diseases that are monitored in Ohio are called arboviruses (**ar**thropod-**bor**ne **viruses**). They all have a similar cycle. The virus passes between a vector mosquito and a vertebrate host, called a reservoir. The species of mosquito and reservoir host are different for each disease. These viruses attack the brain and spinal cord, causing neurological symptoms. They cannot be differentiated clinically. The definitive way to confirm a case is to collect an acute and convalescent blood sample and look for differences in specific antibody titers. By case definition, a confirmed case is one in which there is a fourfold change in titer between the acute and convalescent samples.

La Cross Encephalitis (LAC)

The LAC virus is a member of the California serogroup viruses, genus: *Bunyavirus*, and is endemic in Ohio; that is, it naturally occurs here. Ohio is part of the La Crosse belt that runs from Virginia and Tennessee through Ohio, Indiana, Illinois, and Michigan into Wisconsin and Minnesota. For many years, Ohio reported more cases of LAC encephalitis than any other state. In the past several years, West Virginia has led the nation in reported cases.

LAC virus can attack anyone but disease is most severe in children; the average case age is 6-7 years. The severity of disease ranges from subclinical (detected only by the presence of specific antibodies) to stupor and coma in 50 percent, leaving seizures as a residua in at least 10 percent. The mortality rate is <1 percent, and the case-to-infection rate is < 1:1,000. LAC infection can occur in both suburban and rural settings.

The principal vector of LAC encephalitis is the eastern tree hole mosquito, which breeds in rot cavities in trees and any water-holding container found in shaded areas, especially tires. In fact, the VBDP retrieved a tire from a case residence and reared more than 10,000 mosquitoes from it! The virus is amplified in small woodland mammals, especially chipmunks. Further, the virus can be passed from one generation of mosquitoes to the next through the eggs. This transovarial or vertical transmission is unique to this virus. In laboratory experiments, Dr. Richard Berry, retired chief of the VBDP, passed LAC virus transovarially through 18 generations of *Ochlerotatus triseriatus*, and found that 68 percent of the female mosquitoes were capable of transmitting the virus. This makes container clean-up extremely important for reducing the risk from this disease.

St. Louis Encephalitis (SLE)

In 1975, the upper Midwest experienced an epidemic of SLE. Ohio reported 416 cases and 29 fatalities. This event expanded the focus of the VBDP from surveillance for LAC virus to include other arboviruses as well. SLE is a *Flavivirus* and its life cycle involves birds and bird-feeding mosquitoes, principally of the genus *Culex*. The species pri-

marily involved in maintaining the bird-to-bird cycle are *Culex pipiens,* the Northern house mosquito, and *Culex restuans*. These are very common species which breed in a variety of habitats but are rarely seen, because they prefer to feed on birds. SLE is an urban, epidemic disease with a fatality rate of about 10 percent; the case-to-infection ratio is < 1:200.

SLE attacks all age groups but is more severe in people over 50 years of age. The last documented case of SLE in Ohio occurred in 1985. The virus has been detected sporadically in birds over the years, but not consistently.

Eastern Equine Encephalitis (EEE)

EEE is a very severe disease in both humans and equines. There had been sporadic case reports in horses, but virus was not confirmed in any follow-up investigations until 1991, when a small outbreak occurred in horses around the Killbuck Marsh Wildlife Area in Wayne and Holmes counties. The VBDP detected EEE virus in brain tissue and/or specific EEE antibodies in the blood samples in 19 horses. More than 40 equine deaths were reported but samples were not available for testing.

EEE virus is an *alphavirus* and it cycles between birds and mosquitoes; however, its cycle is unique in that the mosquito that maintains the bird-to-bird transmission is very rare in Ohio because of its habitat: acid bogs and hardwood swamps. Nevertheless, there is a "bridge vector" that passes the virus from birds to mammals. In the Midwest, the bridge vector is the cattail marsh mosquito, *Coquillettidia perturbans*.

EEE virus attacks all age groups and has a mortality rate of more than 70 percent in horses and more than 30 percent in humans. Another 30 percent of human cases are left with severe neurological residua. The case-to-infection ratio is 1:40 in adults and 1:17 in children. Fortunately there has not been a human case of EEE in Ohio; there is an average of five cases per year in the United States. EEE is a rural disease that usually occurs in states along the Gulf Coast, East Coast and in focal areas of upstate New York, northeast Indiana, southwest Michigan and Wisconsin and Minnesota. It usually occurs in very wet years, probably because of the expanded habitat for the bird-to-bird vector.

West Nile Virus (WNV) Infection

West Nile virus (WNV) appeared in the United States in 1999 in New York City and spread across the country. It was first detected in Ohio in 2001 in birds and mosquitoes but not in horses or humans. In 2002 - a drought year- a large outbreak occurred throughout the eastern United States, in which Ohio reported the third-largest number of human cases: 441. The year of 2003 was a very wet year in the eastern United States; this was associated in a reduction in the number of cases in Ohio, as there were 108 Ohio human cases in 2003.

Like SLE virus, the WNV is of the genus: *flavivirus,* and it has a very similar cycle.

It differs in that WNV kills the reservoir birds. WNV is now endemic in the United States and will require continual surveillance to keep the case occurrence as low as possible.

WNV is spread primarily by *Culex* mosquitoes; these mosquitoes are very abundant but are, in general, not noticed by people because mosquitoes of this genus prefer to feed on birds. It is known from years of experience across the United States that mosquito arboviral infection rates vary from season to season. In a normal, non-epidemic season, mosquito infection rates expected are 1:2,000 or so—that is, a positive test for every 2,000 mosquitoes collected in a specific area over a one week time period. An infection rate of 1:400 indicates increased virus activity and should stimulate increased surveillance and preventive measures. An infection rate of 1:200 indicates an epidemic in progress. In 2002, WNV mosquito infection rates in some locations in Ohio reached very high levels, rates as high as 1:50.

Besides monitoring the mosquito populations around the state, the VBDP also tests blood and tissue samples from birds. This gives ODH about a two-month warning with respect to the likely onset of human disease. Positive viral activity in birds triggers increased surveillance in mosquitoes and increased prevention activities. Some birds, especially crows and blue jays, are very susceptible to WNV and develop high levels of viremia followed by high mortality rates. Other birds are less susceptible. For example, exposed sparrows, chickens and pigeons often produce antibodies but do not develop high levels of viremia. Thus, these species are probably not involved in the disease amplification cycle; however, they remain as good sentinel animals.

Horses and other equines are also susceptible to WNV. Horses

are not, however, good surveillance/sentinel animals because they develop WNV infection about the same time as exposed humans; that is, the appearance of infection in horses provides no lead time to prevent human disease. Mortality rates in horses are difficult to calculate because race and event horse owners frequently euthanize clinically infected animals. For the past two years, there have been about 15,000 horse cases in the United States per year. However, there are two vaccines available for use in horses with protection rates above 90 percent.

Of people infected with WNV, about one in five will develop a mild febrile illness (West Nile Fever) (1). About one in 150 will develop meningitis, encephalitis or both. Advanced age is the biggest risk factor for severe neurological disease, long-term morbidity and death (1). Encephalitis or meningoencephalitis is more common than meningitis among hospitalized patients, and common symptoms in this population include fever, weakness (including complete flaccid paralysis), gastrointestinal symptoms, headache and changes in mental status. Among patients hospitalized for WNV infection, the mortality rate is more than 10 percent. There is no available vaccine for human use at this time; prevention is fundamental.

For more information about mosquito-borne diseases, look at the ODH Web site: http://www.odh.ohio.gov/ODHPrograms/ZOODIS/ZooMain1.htm and search for zoonotic diseases. The information is updated annually for all diseases. During the mosquito season, WNV data is updated weekly. The Centers for Disease Control and Prevention (CDC) maintains an excellent Web site at http://www.cdc.gov.

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