

## West Nile Virus Surveillance in Illinois, 2005

By Jonathan Yoder, M.S.W., M.P.H., Linn Haramis, Ph.D. and Mark Dworkin, M.D., M.P.H. & T.M.

During 2005, 252 human cases of West Nile virus (WNV) disease including 12 WNV deaths were reported in Illinois. This is the highest number of human cases and deaths reported in Illinois since 2002 (884 cases, 67 deaths) [1], exceeding the number of cases reported in both 2003 (54 cases, 1 death) and 2004 (60 cases, 4 deaths). In 2005, only the state of California (873 cases) reported more human cases than Illinois. Of the 102 Illinois counties, 21 reported at least one human WNV infection. An examination of mapping of human cases from 2002 to 2005 demonstrates that WNV human disease has been reported from northeastern Illinois, Rock Island, McLean, Sangamon and St. Clair counties in all four of these years, while it has sporadically been reported from other counties [Figure 1a–d].

Among the cases reported in 2005, 134 cases (53 percent) were WNV neuroinvasive disease (i.e., meningitis or encephalitis), 91 cases (36 percent) were West Nile fever, and 27 cases (11 percent) were unspecified illness. The median age of cases was 57 years (range: 9–92 years) [Figure 2]; 55 percent of the cases were female. More than half of the WNV cases (135) were residents of Cook County (suburban Cook County 99; City of Chicago 36), followed by DuPage County (47) and Kane County (17). These three counties accounted for 79 percent of the 2005 cases. The highest incidence of WNV disease occurred in residents of DuPage County (5.1 cases per 100,000 population), Kendall County (4.2 per 100,000), Peoria County (3.8 per 100,000) and Kane County (3.6 per 100,000). In addition to the 21 counties reporting at least

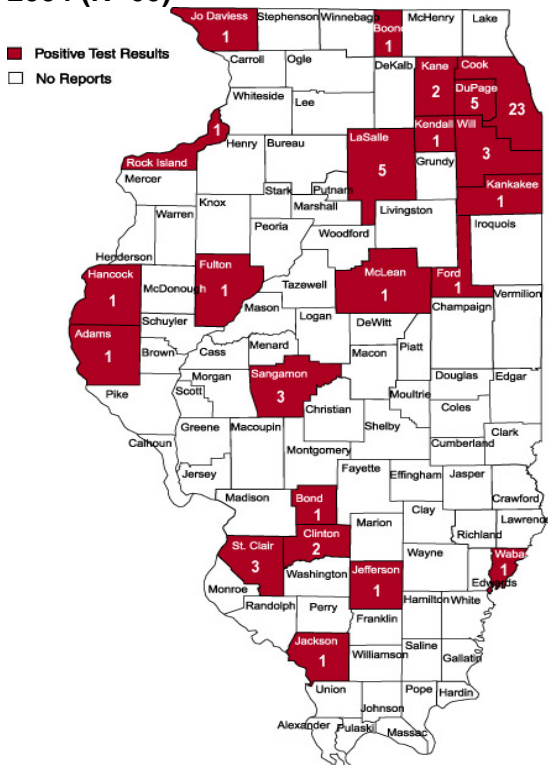
one case in 2005, 34 additional counties had at least one WNV positive bird, mosquito or horse. Statewide mosquito testing revealed 2,465 mosquito batches were WNV positive. Additionally, 227 birds, 16 horses and 1 llama tested WNV positive.

Several possible factors may have contributed to the increased reporting of WNV cases in 2005 compared to 2003 and 2004. Summer temperatures in the northeast part of Illinois were the 3<sup>rd</sup> highest since 1958 and provided favorable conditions for propagation of the house mosquito (*Culex pipiens*) and related species that transmit WNV infection to birds and humans. Above normal temperature has been shown to increase the number of *Culex* mosquitoes as well as increase their rate of WNV infection. However, the summer of 2005 had very little precipitation which decreased the numbers of nuisance “floodwater” mosquitoes. These nuisance mosquitoes are of very minimal importance to transmission of WNV. Nevertheless, *Culex* mosquitoes maintained their population, particularly in urbanized northeastern Illinois, because contrary to conventional wisdom, these vectors are able to thrive despite the lack of rainfall. They establish themselves in urban and suburban areas by utilizing available water in storm water drainage systems (e.g., street catch basins), retention ponds and runoff from sprinkler systems to lay their eggs.

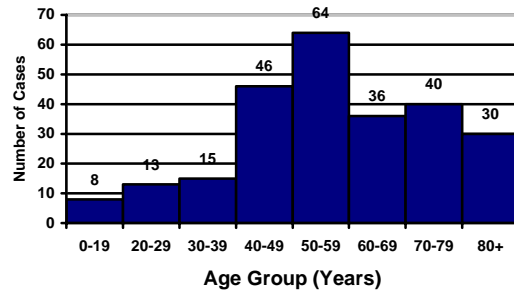
An additional factor that may have contributed to the increased number of human WNV cases was public complacency following low numbers of reported WNV cases in 2003 and 2004. The summer temperatures in 2003 and 2004 were among the coolest recorded which likely led to lower WNV transmission. Public interest in WNV waned as less attention was paid to it during these two low-incidence years. Because nuisance mosquito populations were also



**Figure 1c. Number of Cases of Human West Nile Virus Infection by County—Illinois, 2004 (N=60)**



**Figure 2. Age distribution of case-patients diagnosed with West Nile virus infection—Illinois, 2005 (N=252)**

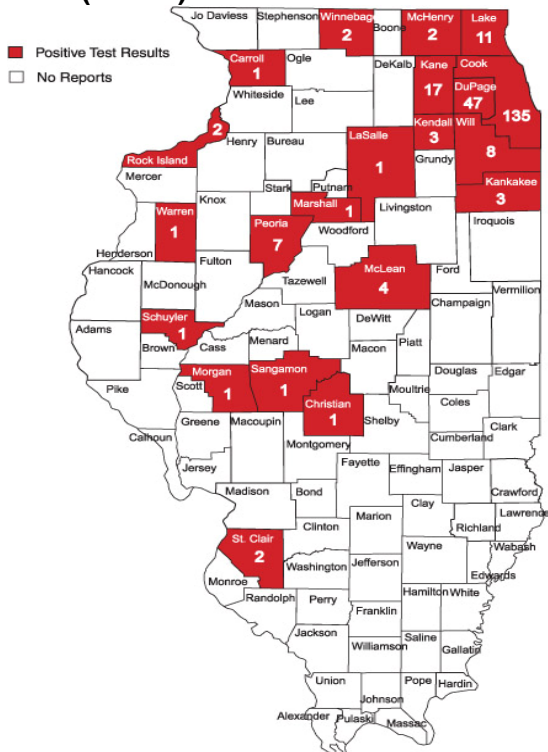


**Leads from the Lab**

**REAL-TIME PCR BASED ASSAY FOR THE DETECTION OF MALARIA PATHOGENS**

By Ira Heimler, Ph.D. Laboratory Research Scientist, Illinois Dept. of Public Health, Chicago Labs

**Figure 1d. Number of Cases of Human West Nile Virus Infection by County—Illinois, 2005 (N=252)**



Malaria pathogens remain a devastating global health concern with an estimated 300-500 million cases occurring annually. The disease in humans is usually caused by any of four species of intraerythrocytic protozoa of the genus *Plasmodium*: *P. falciparum*, *P. vivax*, *P. ovale*, and *P. malariae*. The *Plasmodium* parasites are spread by the bite of an infected *Anopheles* spp. mosquito. The majority of malaria cases in the United States, about 1400 each year, occur among persons who have traveled to areas with ongoing transmission. Other cases in the United States occur through exposure to infected blood products, by congenital transmission, or by local mosquito-borne transmission (1, 2, 3, 4). The signs and symptoms of malaria illness are varied, but the majority of patients experience fever. Other common symptoms include headache, back pain, chills, increased sweating, myalgia, nausea, vomiting, diarrhea, and cough. If untreated, infection with *P. falciparum* can rapidly progress to coma, renal failure, pulmonary edema, and death (4, 5, 6). The IDPH laboratory received 84 specimens for

malaria evaluation in 2005. More than 75% of those specimens were positive.

In the mid 1960s, a rare form of malaria was reported in humans in regions of Malaysia. Recently, a large cluster of human infections caused by this *Plasmodium* was identified in Malaysian Borneo and Thailand. The causative agent is *P. knowlesi*, a simian malaria species closely related to human malarias. *P. knowlesi* is morphologically similar to *P. malariae* when observed in human erythrocytes, but resembles *P. vivax* in non-human primates. Due to morphologic similarities, molecular means of identifying the various species is required (7, 8).

Optimal treatment leading to a cure depends on the proper identification of the infecting *Plasmodium*. The degree of drug resistance, severity of the infection, and personal characteristics (such as drug allergies) must also be taken into consideration (6, 9). Species differentiation of *Plasmodium* is essential for selecting the proper treatment. It is especially critical to differentiate *P. falciparum* from the other human pathogenic species of malaria since this species is responsible for 95% of the deaths caused by malaria (2, 9). In an effort to rapidly identify and detect human pathogenic species of malaria, scientists at the IDPH Division of Laboratories in Chicago began developing a real-time Polymerase Chain Reaction (PCR) assay that may be used on whole blood specimens and blood smears. The test is based on the real-time PCR malaria assay originally developed at the Department of Pathology and Laboratory Medicine, Evanston Northwestern Healthcare and Feinberg School of Medicine. During the development of our test, it was shown that malaria DNA can be isolated from whole blood, thin and thick blood smears, and archived blood smears (even those more than 20 years old), allowing greater flexibility in specimen collection and preparation.

**The detection of malaria genes by polymerase chain reaction (PCR) is an attractive alternative to immunological based assays (10, 11). The major**

**advantages of using a PCR-based technique are the ability to detect malaria parasites in patients with low levels of parasitemia and to identify them to the species level.** PCR-based assays provide improved sensitivity and specificity over previously used immunological tests for the detection of malaria (10, 11). Reports of conventional PCR methods using amplification of a multi-copy ribosomal gene showed that malaria infection may be detected in samples containing fewer than five parasites per microliter (11, 12, 13). Most recently, a commercially available real-time PCR assay has been developed. This assay, the RealArt Malaria RG PCR Kit (Corbett Research), uses the Artus 3000 and the Rotor-Gene 3000 instrumentation with FAM probes to amplify a 163-bp region of the *Plasmodium* genome. Although the system is reliable, it does not differentiate among the main human malarial pathogens (11).

The newly adapted malaria assay at the IDPH is based on identification of the 18S rRNA gene in malaria pathogens infecting humans. The assay is designed to detect and differentiate the 18S rRNA gene common to all malaria species. It is extremely rapid (results can be obtained in less than 3 hours) and is more sensitive than either the antigen-based assays or microscopic examination. The assay will be used in conjunction with and as an aid to conventional microscopic evaluation in the diagnosis of malaria infection in humans. Our goal is to use the assay to identify and detect all five human malaria pathogens from whole blood samples, thin and thick blood smears, and archived materials. We expect the assay will be implemented by the end of the year, and possibly as early as October 2006, depending on the review process. The assay is currently undergoing verification and validation. Implementation of the PCR-based assay will affect the turn-around time of some malaria diagnoses. If the specimen can be identified by microscopy, the PCR-based test will only aid in confirming the diagnosis. However, in cases of low parasitemia, whereby an identification of malaria cannot be made by microscopic analysis alone, specimens are currently sent to the CDC. In these cases a

diagnosis may be delayed for as long as a month; but by using an in-house PCR-based assay, the turn-around time for malaria diagnosis will be greatly reduced.

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## ***Surveillance Is Information for Action!***

### **Epidemiology of Invasive *Haemophilus influenzae* Disease**

**By Mark S. Dworkin M.D., M.P.H.T.M.  
and Stephanie M. Borchardt, Ph.D., M.P.H.**

Before licensure of the *Haemophilus influenzae* type B conjugate vaccine for young infants in 1990, *H. influenzae* type b was the most common cause of bacterial meningitis in children less than 5 years of age in the United States [1]. Consequently, the incidence of invasive *H. influenzae* disease in children <5 years of age has declined 98% percent [2,3]. In adults, the incidence of invasive *H. influenzae* disease in the prevaccine era was estimated at 1.7 cases per 100,000, mortality was 28 percent, and *H. influenzae* type b was responsible for 50 percent of the cases [4]. Onset of *H. influenzae* disease can be subacute but is usually sudden, including fever, vomiting, lethargy, and meningeal irritation, with bulging fontanelle in infants or stiff neck and back in older children. Bacteremia is a common manifestation. *H. influenzae* are divided into unencapsulated (nontypeable) and encapsulated strains. The encapsulated strains are further classified into serotypes a through f.

During January 1996 through December 2004, 770 cases of invasive *H. influenzae* were

reported to the Illinois Department of Public Health. During 1996 to 2004, the number of cases rose from 49 to 132 (an increase of 269 percent) (Table 1, Figure 1). In adults ages 18

to 64 years, the number of cases rose from 13 to 37 (285%), while in adults age  $\geq 65$  years, the number of cases rose from 16 to 58 (363%). The number of cases in children aged  $<5$  years also rose from 1999 to 2004 (Figure 1). Persons at the extremes of age,  $\geq 65$  years and  $<5$  years, accounted for 310 (44.0%) and 148 (21.0%) cases, respectively. The average number of cases in the winter months (December – February) was 128% higher than in the summer months (June – August) (74.0 cases versus 57.7 cases, respectively).

Among 522 isolates submitted to the Illinois Department of Public Health laboratory, overall, type b accounted for 78 cases (14.9%), non-b (a, c, d, e, or f) for 161 cases (30.8%), and nontypeable for 283 cases (54.0%). Most of the 161 non-b isolates were type e (44 cases, 27.3%) and type f (96 cases, 59.6%). Nontypeable *H. influenzae* accounted for the greatest proportion of cases (47.3% to 61.5%) in all age groups, except in persons 50 to 64 years of age, where non-b types accounted for the greatest proportion of cases (46.3%).

Meningitis, bacteremia, and all other diagnoses accounted for 105 (13.6%), 620 (80.5%), and 45 (5.8%) of cases, respectively. Among the 503 cases with survival information, overall, the case fatality rate was 12.7%. Meningitis accounted for only one death during 1996 to 2004. However, the case fatality rate from bacteremia and other illness was 14.9% (59 deaths) and 11.4% (4 deaths), respectively. Overall, the case fatality rate increased with age (Table 1). The case fatality rate for persons  $\geq 60$  years of age was similar to that for persons  $\geq 65$  years of age (19.4%). Mortality was highest during the autumn months of October 8/34 (22.2%) and November 10/44 (22.7%).

According to the Illinois Department of Public Health Rules and Regulations for the Control of Communicable Disease, a case of *H. influenzae* meningitis or other invasive manifestation must be reported by telephone to

the local health authority within 24 hours. Cases must be in respiratory isolation for 24 hours after initiation of chemotherapy. Contacts of a case under 6 years of age,

infants in particular, should be observed for clinical symptoms consistent with *H. influenzae* infection.

Chemoprophylaxis may be administered to household contacts in households in which there are children less than 12 months of age or children 1 to 3 years of age who are inadequately immunized against *H. influenzae* type b. Chemoprophylaxis is also recommended in daycare center classrooms where a case has occurred and children under 12 months of age have been exposed or children 12 to 24 months of age have been exposed and are inadequately immunized.

## Kudos

The Illinois Department of Public Health was fortunate to have matched with three excellent young bright professionals through CDC training programs in 2004. They are completing their 2-year fellowships in the summer of 2006.

**Kathy Ritger, M.D., M.P.H.** has served IDPH as a CDC Epidemic Intelligence Service (EIS) Officer. In that role, she has led a variety of outbreaks and investigations including outbreaks of pertussis, *Pseudomonas aeruginosa*, and *E. coli* O157:H7, and investigations of invasive pneumococcal disease, *Chlamydia*, laboratory exposure to *Brucella*, antimicrobial resistant bacteria, and Creutzfeldt Jakob disease. She has also assisted with a large number of other issues and investigations demonstrating a high level of ability and flexibility.

**Stephanie Borchardt, Ph.D., M.P.H.** has served IDPH as a CDC CSTE Applied Epidemiology Fellow. In that role, she has led outbreak investigations of methicillin-resistant *Staphylococcus aureus* and investigated varicella, pertussis, blastomycosis, HIV (including deaths in women with AIDS and non-named reporting), and the molecular epidemiology of meningococcal disease. Her epidemiological knowledge has allowed for

more technically challenging methods to be used in some investigations.

**Jonathan Yoder, M.S.W., M.P.H.** has served IDPH as a CDC Public Health Prevention Service Fellow. In that role, he has worked on or led outbreaks of cryptosporidiosis, pertussis, and Enterotoxigenic *E. coli*, surveyed an Amish community on immunization knowledge and beliefs, assisted the STD section with hepatitis C prevention planning activities and the TB section with an evaluation, summarized West Nile virus data, and worked in India to eradicate polio. His assistance has allowed for investigations to be carried out that might otherwise have not been possible due to limited resources.

Each of these fellows have been highly productive, participated in each others projects, served on the Rapid Response Team, given many educational presentations throughout the state, and enhanced efforts of the Department. They have done an excellent job in their 2 years in Illinois. They are wished the best of luck with their bright careers ahead!

The Division of Infectious Diseases also recognizes **Michele McGee** who fulfilled the role of Managing Editor of this newsletter since its inception. In early 2006, Michele took a job with a different State of Illinois agency but her hard work and creativity helped make the Illinois Infectious Disease Report a success. Thanks Michele and best of luck to you!

### Factoid

The rash of chickenpox erupts with new spots for a period typically of 2 to 3 days. New vesicles may lie side by side with pustules that may lie near scabs. This is different from smallpox where the spots are generally of the same age in any one area of rash. An exception to this is modified smallpox where the spots are of the same age but they vary in depth so that they appear to be at different stages of development.

## Upcoming Events

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August 7-9, 2006 – Illinois Bioterrorism Summit: Partners Sustaining the Heartland. Oak Brook Hills Marriott Resort, Oakbrook, IL

September 20-21, 2006 – Seventh Annual Illinois Food Safety Symposium: “Tools You Can Use.” Hotel P re Marquette, Peoria, IL

October 31-November 2, 2006 – Illinois HIV and STD Conference. Hilton Hotel, Springfield, IL

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