West Nile Virus (WNV)

West Nile Virus (WNV) is a flavivirus first isolated in 1937 in Uganda. The CDC believes that WNV has been in the eastern US since 1999, has spread westward and is now permanently established in the Western Hemisphere. In the temperate zones, WN disease occurs primarily in late summer or early fall. In 2002, there were 4156 cases in the US, including 284 deaths. Missouri had 168 cases with 7 deaths and Kansas had 22 cases. It is a reportable disease.

WNV is maintained by continuous transmission between mosquito vectors and bird reservoir hosts. WNV has been detected in 138 bird species. The bird reservoir sustains an infectious viremia for one to four days after exposure followed by life-long immunity. Few die. Although people, dogs, cats, horses, and other mammals can be infected, they rarely develop infectious-level viremias and are therefore considered dead-end hosts. There is no documented evidence of casual person-to-person or animal-to-person transmission of WNV. WNV has been documented to be spread by blood transfusion, organ transplantation, breast-feeding, and from mother to child during pregnancy. Starting in July, transfusion blood will be tested.

Approximately 80 percent of infected people will have no symptoms. Symptoms, if they develop, do so from 3 to 14 days after the mosquito bite. WN fever occurs in ~20 percent of infected individuals. It is a mild disease characterized by flu-like symptoms lasting only a few days and with no long term effects. Fewer then 1 percent of infected people develop a more serious disease of encephalitis, meningitis, or meningoencephalitis. Symptoms include high fever, neck stiffness, stupor, disorientation, coma, tremors, convulsions, muscle weakness, and paralysis. These symptoms may last for several weeks and neurological effects may be permanent.

Diagnosis of WNV infection is based on a clinical index of suspicion, including a history of being in an area with WNV activity, and specific laboratory tests. For patients with meningitis or encephalitis, the best diagnostic approach is to detect IgM antibody in serum or CSF. Although the test may be negative very early in disease, it is usually detectable by the time symptoms appear. It becomes positive in 90 percent of infected people within eight days of onset of symptoms. Since IgM does not cross the blood brain barrier, IgM in CSF, if not a bloody tap, indicates CNS involvement. IgG is usually not detectable until four to five days after symptoms. A PCR test is also available and may be performed as an adjunct to serological testing but does not replace it. Meditech Order Entry codes are given below:

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IgG antibodies develop as part of a complex chain of events when the body’s immune system attempts to inactivate and destroy or remove a foreign substance such as an antigen. Thus, the presence of IgG antibodies specific to a certain antigen is a reliable marker of exposure to that antigen. Furthermore, the level of specific IgG in serum usually reflects the extent of exposure to that antigen. Measuring specific IgG antibodies provides a range of valuable clinical information, especially in the areas of Allergy, Infectious Disease, and Rheumatology.

The Immunology laboratory has the ability to quantify IgG antibodies in human serum directed against specific antigens to select foods, inhalants, and venom using the UniCap instrument. We currently offer determination of IgG antibodies directed against Penicillium notatum, Cladosporium hergarum, Aspergillus fumigatus, Alternaria alternata, and Stachybotrys chartarum molds.

Allergic and other hypersensitivity responses to molds may be immunoglobulin E (IgE)- or immunoglobulin G (IgG)-mediated. Reactions may include immediate hypersensitivity, hypersensitivity pneumonitis, and uncommon allergic syndromes.

The most common form of hypersensitivity to molds is immediate hypersensitivity. This involves an IgE-mediated response to fungal proteins and may lead to allergic asthma or allergic rhinitis that is triggered by breathing in mold spores or hyphal fragments. Circulating IgE antibodies specific to the mold are generally elevated.

An exaggeration of the normal IgG immune response against inhaled foreign proteins, including fungi, is known as hypersensitivity pneumonitis (HP). Most cases of HP result from occupational exposure; however, pet birds, humidifiers, and heating, ventilating, and air conditioning (HVAC) systems may also precipitate the disease. Very high serum levels of specific IgG proteins are observed in HP; however, it is not diagnostic. More than 50 percent of people with occupational exposure to a specific protein have antibodies but do not have clinical disease.

The uncommon allergic syndromes include allergic bronchopulmonary aspergillosis (ABPA) and allergic fungal sinusitis (AFS). These conditions are characterized by unusual variants of allergic (IgE-mediated) reactions in which fungi grow within the patient’s airway or sinuses. Colonization of the fungus does not cause adverse health consequences unless the subject is allergic to the specific fungus that has taken up residence. ABPA is a common complication of cystic fibrosis, occurring in approximately 10 percent of patients and accounts for approximately 10 percent of pulmonary exacerbations.

Exposure to indoor fungi has recently been of concern to the general population. The “black mold” that has been in the news so much lately is usually considered to be Stachybotrys chartarum. It is a fungus with wide geographic distribution and grows well on materials with high cellulose content like sheet rock or jute-backed carpet. Exposure has been associated with human illness in a number of studies. The most controversial linkage is with a series of infant deaths from idiopathic pulmonary hemorrhage in Ohio.

Stachybotrys spores may be in indoor air if there is significant water damage and visible mold growth; however, the level of exposure in undisturbed air is minimal. Only about 49 percent of normal sera tested demonstrated IgG antibodies against Stachybotrys. The presence, or more significantly the absence, of IgG antibodies directed against Stachybotrys can be helpful in determining prior exposure to this fungus and the diagnosis of any subsequent health consequences.

Evaluation of the presence and quantity of human IgG antibodies directed against specific organisms is clinically useful. The clinical laboratory is often requested to make such evaluations. The range of organisms for which these evaluations are offered is ever increasing.