Hantavirus Infection (including Pulmonary Syndrome)

Hantavirus Infection (including Pulmonary Syndrome) is a Class B Disease and must be reported to the state within one business day.

Hantavirus pulmonary syndrome (HPS) became nationally notifiable in 1995. HPS is a febrile illness characterized by clinical symptoms resembling acute respiratory distress syndrome (ARDS) or bilateral diffuse interstitial edema. Infection is also characterized by a relatively short febrile prodrome, myalgias, headache, chills, dizziness, non-productive cough and gastrointestinal (GI) complaints. In more severe cases, patients may develop severe pulmonary edema and hypotension. Patients may progress, although rarely, to disseminated intravascular coagulation (DIC). Asymptomatic illness is rare.

Non-HPS infection is considered to be a febrile illness with non-specific viral symptoms including fever, chills, myalgia, headache and GI symptoms but no cardio-pulmonary symptoms. Non-HPS infections typically also have clinical laboratory findings including hemoconcentration, left shift in WBC count, neutrophilic leukocytosis, thrombocytopenia and circulating immunoblasts. In 2014, surveillance expanded to include reporting of all laboratory confirmed hantavirus infections (HPS and non-pulmonary hantavirus infection).

Hantavirus pulmonary syndrome can be caused by any of the more than 25 antigenically distinguished viral species, each associated primarily with a single rodent species. The rodent hosts are usually not associated with urban environments, although several may enter human dwellings in rural or suburban areas.

The primary risk factor for hantavirus exposure is rodent infestations in and around the home. Occupational exposures have been recognized, but are rare. Humans acquire infection primarily through inhalation of infectious aerosolized rodent saliva or excreta. Person-to-person transmission has never been associated with HPS cases in the United States. Nevertheless, universal precautions are recommended due to evidence of person-to-person transmission of a related virus in South America.

Hantavirus pulmonary syndrome is often referred to as an emerging infectious disease. The disease was first identified in 1993, but evidence exists of a confirmed Sin Nombre virus (SNV) infection in 1959, with signs compatible with HPS.

Hantaviral infections are rarely reported in Louisiana. Several cases have been caused by a unique hantavirus named Bayou virus. The Bayou virus infection is characterized by Hantavirus pulmonary syndrome with renal insufficiency and intra-alveolar hemorrhage (resembling hemorrhagic fever with renal syndrome (HFRS) associated with a family of Eurasian hantaviruses). The primary reservoir for the virus is thought to be the rice rat, *Oryzomys palustris*. (Table 1)

Table 1: Most Common Hantaviruses Known to Cause HPS and the Associated Rodent Reservoir United States

Virus Name	Rodent Species
Sin Nombre	Deer mouse (*Peromyscus maniculatus*)
New York	White footed mouse (*Peromyscus leucopus*)
Black Creek Canal	Cotton rat (*Sigmodon hispidus*)
Bayou virus	Rice rat (*Oryzomys palustris*)

Several other Hantaviruses associated with specific rodent species also have been identified. HPS is present only in the Americas. Since 2005, there have been very few Hantavirus cases reported in Louisiana. (Table 2)

Table 2: Confirmed Hantavirus Cases – Louisiana, 2005-2018

Reported		Sex	Age	Parish	Severity/ Hospitalized	Deceased	Exposure
Year	Month						
2005	10	M	63	Jeff Davis	ICU	N	Barn at home infested with mice
2007	04	M	48	Lafayette	ICU	Y	
2007	11	M	35	Sabine	ICU	N	Trailer in oil field infested with mice, Houston
2008	02	M	22	Calcasieu	ICU	N	Lodge in CO infested with mice; two friends from same trip suspected
2008	05	F	53	Caddo	ICU	N	
2013	06	M	51	Avoyelles	ICU	N	School teacher. Cleaned out an office area at high school that had a lot of rat/mouse droppings

More than 96% of all nationally reported cases have occurred in states west of the Mississippi River. Four of the six cases reported in Louisiana had exposure outside of the state.

Differences in viruses in the U. S. complicate the use and sensitivity of RT-PCR for the routine diagnosis of hantaviral infections. None of the tests for SNV are licensed by the Food and Drug Administration for diagnostic use with human sera. Acute- and convalescent-phase sera should reflect a four-fold rise in IgG antibody titer or the presence of IgM in acute-phase sera to be diagnostic for hantaviral disease. Note that acute-phase serum sent as an initial diagnostic specimen may not yet have IgG. IgG antibody is long lasting, and sera of patients retrospectively identified appear to have retained antibody for many years. A number of IgM positive (IgG negative) samples may represent false positive reactivity associated with other viral infections.