Revisions of case criteria for Clinical Description and/or criteria (CD); Confirmed, Probable, Possible, or Suspect cases (CC, PC, PsC, SC); Laboratory Confirmation tests (LC); and Note(s) (N)

- Acute Flaccid Myelitis (CD), (CC), (PC), (SC), (LC)
- Anthrax (N)
- Arbovirus (N)
- Chagas disease, acute (PC), (N)
- Chagas disease, chronic indeterminate (PC), (LC), (N)
- Chagas disease, chronic symptomatic (PC), (LC), (N)
- CRE/CP-CRE
- Cyclosporiasis (CC), (LC)
- Malaria (N)
- Norovirus (CD), (PC), (LC)
- Novel Coronavirus 2019 (CD) effective 11/1/21
- Prion diseases such as Creutzfeldt-Jakob disease (CJD) (CD), (PsC), (LC)
- Rabies, animal (LC)
- Rabies, human (LC)
- Relapsing fever, tickborne (CD)

- Rickettsiosis, unspecified (CD), (PC)
- Shiga Toxin Producing E. Coli / STEC (PC), (LC), (N)
- Spotted fever rickettsiosis (CD), (LC)
- Typhus fever, flea-borne (CD), (LC)

Added Notifiable Conditions

- CP-CRE, subcondition of CRE

Removed Notifiable Conditions

- Multidrug Resistant Acinetobacter (MDR-A)
# TABLE OF CONTENTS

This document provides infectious disease information for surveillance and data entry staff. It contains a table with condition codes, condition names, and case criteria to aid in the classification and coding of conditions. It is organized alphabetically by condition name. Conditions specified as reportable in Title 25, Texas Administrative Code, Chapter 97, Subchapter A, Control of Communicable Diseases are in **bold type**. Click on a condition in the table of contents to go to the text and on the condition code to move back.

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## CASE CRITERIA

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Zika infection, congenital
Zika infection, non-congenital
DEFINITIONS OF TERMS

Clinically compatible case: Medical history and/or signs and symptoms generally compatible with the disease, as described in the clinical description

Confirmed case: A case that is classified as confirmed for reporting purposes

Culture-independent diagnostic testing: The detection of antigen or nucleic acid sequences of the pathogen

Epidemiologically linked case: A case in which a) the patient has had contact with one or more persons who either have/had the disease or have been exposed to a point source of infection (i.e., a single source of infection, such as an event leading to a foodborne-disease outbreak, to which all confirmed case-patients were exposed) and b) transmission of the agent by the usual modes of transmission is plausible
  ▪ A case can be considered epidemiologically linked to a laboratory-confirmed case if at least one case in the chain of transmission is laboratory confirmed.

Laboratory-confirmed case: A case that is confirmed by one or more of the laboratory methods listed in the case definition under Laboratory Confirmation Tests

While other laboratory methods can be used in clinical diagnosis, only those listed are accepted as laboratory confirmation for national and state reporting purposes.

Probable case: A case that is classified as probable for reporting purposes

Supportive or presumptive laboratory results: Specified laboratory results that are consistent with the diagnosis, yet do not meet the criteria for laboratory confirmation

Suspect case: A case that is classified as suspect for reporting purposes

Normally sterile site: Invasive diseases typically cause significant morbidity and mortality. Sterile sites include:
  ▪ Blood (excluding cord blood)
  ▪ Bone or bone marrow
  ▪ Cerebrospinal fluid (CSF)
  ▪ Pericardial fluid
  ▪ Peritoneal fluid
  ▪ Pleural fluid

The following are also considered sterile sites when certain other criteria are met:
  ▪ Internal body sites (brain, heart, liver, spleen, vitreous fluid, kidney, pancreas, lymph node or ovary) when the specimen is collected aseptically during a surgical procedure
  ▪ Joint fluid when the joint surface is intact (no abscess or significant break in the skin)

Although placentas and amniotic fluid from an intact amnion are not considered sterile sites, isolation of Group B streptococci or Listeria from these sites may qualify as invasive disease. Consult the Sterile Site and Invasive Disease Determination flowchart in Appendix A of the EAIDB Investigation Guidelines for more information:

https://www.dshs.texas.gov/IDCU/investigation/Investigation-Guidance/

Normally sterile sites do not include:
  ▪ Anatomical areas of the body that normally harbor either resident or transient flora (bacteria) including mucous membranes (e.g., throat, vagina), sputum, and skin; abscesses; or localized soft tissue infection

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ABBREVIATIONS

LABORATORY TEST ABBREVIATIONS
CF – Complement fixation
CIDT – Culture-independent diagnostic testing
CLSI – Clinical and Laboratory Standards Institute
CSF – Cerebrospinal fluid
DFA – Direct fluorescent antibody
DNA – Deoxyribonucleic acid
EEG – Electroencephalogram
EIA – Enzyme immunoassay
EIISA – Enzyme-linked immunosorbent assay
HA – Hemagglutination
HI – Hemagglutination inhibition
ID – Immunodiffusion
IFA – Indirect fluorescent antibody test
IgG – Immunoglobulin G
IgM – Immunoglobulin M
IHA – Indirect hemagglutination
IHC – Immunohistochemistry
LA – Latex agglutination
MA – Microagglutination
MIC – Minimum inhibitory concentration
MRI – Magnetic resonance imaging
NAT – Nucleic acid testing
PCR – Polymerase chain reaction
PRNT – Plaque reduction neutralization test
RIBA – Recombinant immunoblot assay
RIPA – Radio-immune precipitation assay
rRT-PCR – Real-time reverse transcriptase-polymerase chain reaction
RT-PCR – Reverse transcription polymerase chain reaction
WB – Western blot

HEPATITIS TEST MARKERS
Hepatitis A – HAV
Anti-HAV – hepatitis A antibody
Anti-HAV IgM – hepatitis A IgM antibody
Hepatitis B – HBV
HBcAb or anti-HBc – hepatitis B core antibody
HBc IgM or anti-HBc IgM – hepatitis B core IgM antibody
HBeAb or anti-HBe – hepatitis B e antibody
HBeAg – hepatitis B e antigen
HBsAb or anti-HBs – hepatitis B surface antibody
HBsAg – hepatitis B surface antigen
Hepatitis C – HCV
Anti HCV – hepatitis C antibody
HCV RNA – hepatitis C nucleic acid
HCV NAT – hepatitis C nucleic acid testing
HCV RIBA – hepatitis C recombinant immunoblot assay
Hepatitis D – HDV
Anti-HDV – hepatitis D antibody
Hepatitis E – HEV
Anti-HEV IgM – hepatitis E IgM antibody

OTHER ABBREVIATIONS:
ALT – Alanine transaminase
ARDS – Acute Respiratory Distress Syndrome
AST – Aspartate transaminase
CDC – Centers for Disease Control and Prevention
DHS – Department of State Health Services
EAIDB – Emerging and Acute Infectious Disease Branch
FDA – Food and Drug Administration
HAI- Healthcare Associated Infections
ILI – Influenza-Like Illness
NDM-1 – New Delhi Metallo-beta-lactamase-1
NPDPSC – The National Prion Disease Pathology Surveillance Center
TAC – Texas Administrative Code
VHF – Viral hemorrhagic fever
**Rickettsia Classification**

Rickettsial diseases can be difficult to distinguish between because of overlapping symptomatology and cross-reactivity in serology, which comprises the majority of diagnostic testing for these diseases. The *Rickettsia* are divided into two antigenic groups for surveillance purposes: spotted fever group and typhus group. The condition spotted fever rickettsiosis is defined as infection with spotted fever group *Rickettsia* spread by tick vectors. Flea-borne (murine) typhus, caused primarily by *R. typhi* and spread by fleas, and epidemic typhus, caused by *R. prowazekii* and transmitted by lice, belong to the typhus group. A table classifying Rickettsial species known to cause disease in humans by antigenic group, disease, primary vector, and reservoir occurrence can be found in the CDC’s Traveler’s Health Yellow Book at https://wwwnc.cdc.gov/travel/yellowbook/2020/travel-related-infectious-diseases/rickettsial-including-spotted-fever-and-typhus-fever-rickettsioses-scrub-typhus-anaplasmosis-and-ehr

**Streptococcus Classification**

Streptococci are facultatively anaerobic, gram-positive organisms that often occur as chains or pairs. There are four different classification systems for *Streptococcus* species, clinical (pyogenic, oral, enteric), hemolysis (alpha-hemolysis, beta-hemolysis, gamma-hemolysis), serological (Lancefield: A-H and K-U), and biochemical (physiological).

Lancefield group

Streptococci are subdivided into groups by antibodies that recognize surface antigens. The serologic reactivity of "cell wall" polysaccharide “C” antigens was described by Rebecca Lancefield. Twenty group-specific antigens were established, Lancefield A-H and K-U. Clinically significant Lancefield groups include A, B, C, F, and G. Some streptococci such as *Streptococcus pneumoniae* and the viridans streptococci are Lancefield group nontypeable.

Hemolytic reaction

The type of hemolytic reaction displayed on blood agar has also been used to classify the streptococci. Beta-hemolysis is associated with complete lysis of red cells surrounding the colony, whereas alpha-hemolysis is a partial or "green" hemolysis associated with reduction of red cell hemoglobin. Nonhemolytic colonies have been termed gamma-hemolytic. The property of hemolysis is not very reliable for the absolute identification of streptococci, but it is widely used in rapid screens for identification.

Reportable *Streptococcus*

*Streptococcus pneumoniae* (pneumococcus) - Most strains of *S. pneumoniae* are alpha-hemolytic but can cause β-hemolysis during anaerobic incubation. They are nontypeable by Lancefield group.
### Case Criteria

<table>
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<th>Laboratory Confirmation Tests</th>
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| Acute Flaccid Myelitis 11120 | An illness with onset of acute flaccid limb weakness (low muscle tone, limp, hanging loosely, not spastic or contracted) of one or more limbs. **Confirmed:** A case that meets the clinical symptoms AND confirmatory laboratory/imaging evidence in the absence of a clear alternative diagnosis attributable to a nationally notifiable condition.  
- MRI showing spinal cord lesion with predominant gray matter involvement* and spanning one or more vertebral segments, AND  
- Excluding persons with gray matter lesions in the spinal cord resulting from physician diagnosed malignancy, vascular disease, or anatomic abnormalities. **Probable:** A case that meets the clinical symptoms AND presumptive laboratory/imaging evidence in the absence of a clear alternative diagnosis attributable to a nationally notifiable condition.  
* Presumptive laboratory/imaging evidence:  
- MRI showing spinal cord lesion where gray matter involvement is present, but predominance cannot be determined, AND  
- Excluding persons with gray matter lesions in the spinal cord resulting from physician diagnosed malignancy, vascular disease, or anatomic abnormalities. **Suspect:** A case that meets the clinical symptoms with supportive laboratory/imaging evidence AND available information is insufficient to classify case as probable or confirmed.  
* Supportive laboratory/imaging evidence:  
- MRI showing spinal cord lesion in at least some gray matter and spanning one or more vertebral segments, AND  
- Excluding persons with gray matter lesions in the spinal cord resulting from physician diagnosed malignancy, vascular disease, or anatomic abnormalities. | **Laboratory Confirmation Tests:**  
- A magnetic resonance image (MRI) showing spinal cord lesion with predominant gray matter involvement and spanning one or more vertebral segments,  
- Excluding persons with gray matter lesions in the spinal cord resulting from physician diagnosed malignancy, vascular disease, or anatomic abnormalities.  
* Terms in the spinal cord MRI report such as “affecting mostly gray matter,” “affecting the anterior horn or anterior horn cells,” “affecting the central cord,” “anterior myelitis,” or “poliomyelitis” would all be consistent with this terminology. |

*Terms in the spinal cord MRI report such as “affecting mostly gray matter,” “affecting the anterior horn or anterior horn cells,” “affecting the central cord,” “anterior myelitis,” or “poliomyelitis” would all be consistent with this terminology.*
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| Amebic meningitis/encephalitis, other 10096 | An infection presenting as meningoencephalitis or encephalitis. Granulomatous amebic encephalitis (GAE) can include general symptoms and signs of encephalitis such as early personality and behavioral changes, depressed mental status, fever, photophobia, seizures, nonspecific cranial nerve dysfunction, and visual loss. GAE neurologic infections are generally fatal within weeks or months; however, a few patients have survived.  
**Confirmed:** A clinically compatible case that is laboratory confirmed  
Note: *Acanthamoeba* species and *Balamuthia mandrillaris* can also cause disseminated disease (affecting multiple organ systems) or cutaneous disease. For *B. mandrillaris* disease, painless skin lesions appearing as plaques a few millimeters thick and one to several centimeters wide have been observed in some patients, especially patients outside the U.S., preceding the onset of neurologic symptoms by 1 month to approximately 2 years. Skin lesions and sinus disease may be seen in *Acanthamoeba* disease. Disseminated disease and cutaneous disease caused by free-living amebae are only voluntarily reportable in Texas unless they progress to meningitis or encephalitis.  
See also [Amebic meningoencephalitis, primary (PAM)](http://www.cdc.gov/parasites/acanthamoeba/) | Detection of *Acanthamoeba, Balamuthia,* or another non-*Naegleria* free-living ameba from a clinical specimen or culture via:  
- Detection of nucleic acid (e.g., PCR),  
  **OR**  
- Detection of antigen (e.g., immunohistochemistry)  
Contact the DSHS epidemiologist for meningitis (amebic) at 800-252-8239 if suspected. DSHS can assist in coordinating specimen and/or electronic images submission to the CDC for verification. Collection & shipping procedures can be found at: [http://www.cdc.gov/parasites/acanthamoeba/](http://www.cdc.gov/parasites/acanthamoeba/) and [http://www.cdc.gov/parasites/balamuthia/](http://www.cdc.gov/parasites/balamuthia/)  
Note: *Acanthamoeba* spp. and *B. mandrillaris* can cause clinically similar illnesses and might be difficult to differentiate using commonly available laboratory procedures. Definitive diagnosis by a reference laboratory might be required. A negative test on CSF does not rule out *Acanthamoeba* or *Balamuthia* infection because these organisms are not commonly present in the CSF. |
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| Amebic meningoencephalitis, primary (PAM) 80750 | An infection presenting as meningoencephalitis or encephalitis. The clinical presentation of PAM is like that of acute meningitis caused by other pathogens and symptoms include headache, nausea, vomiting, anorexia, fever, lethargy, and stiff neck. Disorientation, mental status changes, seizure activity, loss of consciousness, and ataxia may occur within hours of initial presentation. After the onset of symptoms, the disease progresses rapidly and usually results in death within 3 to 7 days. **Confirmed:** A clinically compatible case that is laboratory confirmed  
**Probable:** A clinically compatible case that meets at least one of the supportive laboratory criteria (listed below) and does not meet confirmatory lab criteria  
- Supportive laboratory evidence:  
  - Visualization of motile amebae in a wet mount of CSF  
  - Isolation of *N. fowleri* in culture from a clinical specimen  
See also [Amebic meningitis/encephalitis, other](#). | Detection of *Naegleria fowleri* from a clinical specimen via:  
- Detection of nucleic acid (e.g., PCR),  
  OR  
- Detection of antigen (e.g., immunohistochemistry)  
Notes:  
- When available, molecular characterization [e.g., genotype] should be reported.  
- Contact the DSHS epidemiologist for amebic meningitis at 800-252-8239 if suspected. DSHS can assist in coordinating specimen and/or electronic images submission to the CDC for verification.  
- Collection & shipping procedures can be found at: [http://www.cdc.gov/parasites/naegleria/diagnosis-hcp.html](http://www.cdc.gov/parasites/naegleria/diagnosis-hcp.html)  
*Naegleria fowleri* might cause clinically similar illness to bacterial meningitis, particularly in its early stages. Definitive diagnosis by a reference laboratory is required. Unlike *Balamuthia mandrillaris* and *Acanthamoeba* spp., *N. fowleri* is commonly found in the CSF of patients with PAM. |
| Anaplasmosis (*Anaplasma phagocytophilum* infection) 11090 | Anaplasmosis is a tick-borne illness caused by the bacterium *Anaplasma phagocytophilum*, which is transmitted primarily by blacklegged ticks (*Ixodes* spp.). Initial symptoms may include fever/chills, headache, myalgia, nausea/vomiting and diarrhea. Anaplasmosis may result in severe illness or even death in older or immunocompromised individuals or if treatment is delayed.  
**Clinical evidence:** Fever as reported by patient or provider and one or more of the following: headache, myalgia, anemia, leukopenia, thrombocytopenia, or any hepatic transaminase elevation.  
**Confirmed:** A clinically compatible illness that is laboratory confirmed  
**Probable:** A clinically compatible illness with serological evidence of IgG or IgM antibody reactive (≥1:128) with *A. phagocytophilum* antigen by IFA, OR identification of morulae in the cytoplasm of neutrophils or eosinophils by microscopic examination  
**Suspect:** A case with laboratory evidence of past/present infection with *A. phagocytophilum* (e.g., laboratory report) but no available clinical information |  
- Demonstration of a four-fold change in IgG-specific antibody titer to *A. phagocytophilum* antigen by IFA in paired serum samples (preferably one taken in first week of illness and a second taken 2-4 weeks later),  
  OR  
- Detection of *A. phagocytophilum* DNA in a clinical specimen by PCR,  
  OR  
- Demonstration of anaplasmal antigen in a biopsy/autopsy sample by IHC,  
  OR  
- Isolation of *A. phagocytophilum* from a clinical specimen in cell culture |
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| Anthrax 10350 | An illness or post-mortem examination characterized by several distinct clinical forms, including:  
- **Cutaneous**: A skin lesion evolving during a period of 2-6 days from a papule, through a vesicular stage, to a depressed black eschar. Fever, malaise, and lymphadenopathy can accompany the lesion.  
- **Inhalation**: A prodrome resembling a viral respiratory illness, followed by hypoxia and dyspnea, or acute respiratory distress syndrome (ARDS) with resulting cyanosis and shock. Radiographic evidence of mediastinal widening or pleural effusion is common.  
- **Ingestion** presents as two sub-types:  
  - **Gastrointestinal**: Severe abdominal pain and tenderness, nausea, vomiting, hematemesis, bloody diarrhea, anorexia, fever, and septicemia.  
  - **Oropharyngeal**: Mucosal lesion in the oral cavity or oropharynx, with cervical adenopathy, edema, pharyngitis, fever, and possible septicemia.  
- **Injection**: Severe soft tissue infection manifested as significant edema or bruising after injection. No eschar is apparent and pain is not common. Nonspecific symptoms such as fever, shortness of breath and nausea are sometimes the first indication of illness.  
- **Systemic involvement**: May include fever, convulsions, tachycardia, tachypnea, hypotension, leukocytosis, and/or meningeal signs (anthrax meningitis). These complications may be secondary to the above syndromes.  
  
**Clinical criteria**: A clinically compatible illness with at least one specific OR two nonspecific symptoms and signs that are compatible with cutaneous, ingestion, inhalation, or injection anthrax; systemic involvement; or anthrax meningitis; OR a death of unknown cause AND organ involvement consistent with anthrax.  
  
**Confirmed**: A case that meets clinical criteria AND has confirmatory laboratory test results.  
  
**Probable**: A case that meets clinical criteria AND has a Gram stain demonstrating Gram-positive rods, square-ended, in pairs or short chains; OR a positive result on a test with established performance in a CLIA-accredited laboratory; OR has epidemiologic linkage relating it to anthrax.  
  
**Suspect**: A case that meets the clinical criteria AND for whom an anthrax test was ordered, but with no epidemiologic linkage relating it to anthrax.  
  
Epidemiologic linkage is defined as one or more of the following:  
- Exposure to environment, food, animal, materials, or objects that is suspected or confirmed to be contaminated with *B. anthracis*; OR  
- Exposure to the same environment, food, animal, materials, or objects as another person who has lab-confirmed anthrax; OR  
- Consumption of the same food as another person who has laboratory-confirmed anthrax.  
  
- Culture and identification of *Bacillus anthracis* or *B. cereus* expressing anthrax toxins from clinical specimens by the Laboratory Response Network, OR  
- Demonstration of *B. anthracis* antigens in tissues by IHC using both *B. anthracis* cell wall and capsule monoclonal antibodies, OR  
- Evidence of a four-fold rise in antibodies to protective antigen between acute and convalescent sera or a fourfold change in antibodies to protective antigen in paired convalescent sera using CDC quantitative anti-PA IgG ELISA testing in an unvaccinated person, OR  
- Detection of Lethal Factor (LF) in clinical serum specimens by LF mass spectrometry OR  
- Detection of *B. anthracis* or anthrax toxin genes by the LRN-validated PCR and/or sequencing in clinical specimens collected from a normally sterile site or lesion of other affected tissue.  
  
Note: As required by TAC, all *B. anthracis* isolates must be submitted to the DSHS Laboratory. *B. cereus* expressing anthrax toxin suspect isolates from patients with severe disease should be forwarded for confirmation.
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<th>Disease Category</th>
<th>Description</th>
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<td>Arbovirus, neuroinvasive and non-neuroinvasive</td>
<td>For the purposes of surveillance and reporting, arboviral disease cases are often categorized into two primary groups based on their clinical presentation: neuroinvasive disease and non-neuroinvasive disease. Many arboviruses cause neuroinvasive disease such as aseptic meningitis, encephalitis, or acute flaccid paralysis (AFP). These illnesses are usually characterized by the acute onset of fever with stiff neck, altered mental status, seizures, limb weakness, CSF pleocytosis, and/or abnormal neuroimaging. Less common neurological manifestations, such as cranial nerve palsies, also occur. AFP is characterized by rapid-onset extremity, facial, and/or respiratory weakness and flaccid muscle tone in the affected area; AFP may result from anterior myelitis, peripheral neuritis or post-infectious peripheral demyelinating neuropathy (Guillain-Barré Syndrome). Meningitis is infection or inflammation of the tissues surrounding the brain; symptoms can include fever, headache, photophobia, and nuchal rigidity. Encephalitis is infection or inflammation of the brain tissue itself and may present with fever, altered mental status, seizures, and focal neurologic deficits; meningitis may also be present simultaneously, known as meningoencephalitis. Most arboviruses are capable of causing an acute systemic febrile illness (e.g., West Nile fever) that may include headache, myalgias, arthralgias, rash, and/or gastrointestinal symptoms. Some viruses also can cause more characteristic clinical manifestations, such as severe polyarthritis or arthritis due to chikungunya virus or other alphaviruses.</td>
</tr>
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| Neuroinvasive disorders: | 10058 Cache Valley virus  
10054 California serogroup virus  
10053 Eastern equine encephalitis virus  
10078 Jamestown Canyon virus  
10059 Japanese encephalitis virus  
10081 La Crosse virus  
10057 Powassan virus  
10051 St. Louis encephalitis virus  
10055 Venezuelan equine encephalitis virus  
10056 West Nile virus  
10052 Western equine encephalitis virus |
| Non-neuroinvasive disorders: | 10066 Cache Valley virus  
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10062 Eastern equine encephalitis virus  
10079 Jamestown Canyon virus  
10068 Japanese encephalitis virus  
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10064 St. Louis encephalitis virus  
10067 Venezuelan equine encephalitis virus  
10049 West Nile virus  
10065 Western equine encephalitis virus |
| Other disease categories: | 11718 California encephalitis virus disease  
10073 Chikungunya virus disease  
10093 Colorado tick fever virus disease  
50237 Flavivirus disease, not otherwise specified  
11712 Keystone virus disease  
10072 Other arboviral diseases, not otherwise specified  
11734 Snowshoe hare virus disease  
10074 Tick-borne Encephalitis viruses  
11724 Trivittatus virus disease |

Clinical evidence of neuroinvasive disease:  
- Meningitis, encephalitis, acute flaccid paralysis, or other acute signs of central or peripheral neurologic dysfunction, as documented by a physician, AND  
- Absence of a more likely clinical explanation  

Clinical evidence of non-neuroinvasive disease:  
- Fever or chills as reported by the patient or a health-care provider, AND  
- Absence of neuroinvasive disease, AND  
- Absence of a more likely clinical explanation  

Neuroinvasive:  
- Isolation of virus from, or demonstration of specific viral antigen or nucleic acid in, tissue, blood, CSF, or other body fluid, OR  
- Four-fold or greater change in virus-specific quantitative antibody titers in paired sera, OR  
- Virus-specific IgM antibodies in serum with confirmatory virus-specific neutralizing antibodies in the same or a later specimen, and negative neutralizing antibody results for potentially cross-reactive* arboviruses endemic to the region where exposure occurred, OR  

Non-neuroinvasive:  
- Isolation of virus from, or demonstration of specific viral antigen or nucleic acid in, tissue, blood, or other body fluid, excluding CSF, OR  
- Four-fold or greater change in virus-specific quantitative antibody titers in paired sera, OR  
- Virus-specific IgM antibodies in serum with confirmatory virus-specific neutralizing antibodies in the same or a later specimen and negative neutralizing antibody results for potentially cross-reactive* arboviruses endemic to the region where exposure occurred  

*Viruses in the same genus (the majority of pathogenic arboviruses are in the flavivirus, alphavirus, or orthobunyavirus genus) are generally considered potentially cross-reactive. Consider area of exposure, clinical manifestations of each arbovirus, and level of arbovirus activity when assessing which viruses must be ruled out.
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| **Ascariasis**  | A parasitic infection caused by the soil-transmitted helminths *Ascaris lumbricoides* and *Ascaris suum*. Most infections with *Ascaris* spp. are asymptomatic. Live worms, passed in stool or occasionally from the mouth, anus, or nose, are often the first recognized sign of infection. Larval migration may result in pulmonary manifestations such as wheezing, cough, fever, eosinophilia and pulmonary infiltration in some patients. Light infections may result in minor abdominal discomfort, dyspepsia, and loss of appetite. Heavy infections may result in severe abdominal pain, fatigue, vomiting, or weight loss. In children, these symptoms can result in nutrient deficiencies resulting in growth retardation and/or cognitive impairment. Serious complications are rare but can be fatal and include intestinal obstruction by a bolus of worms, or obstruction of bile duct, pancreatic duct or appendix by one or more adult worms.  
**Confirmed:** A case that is laboratory confirmed  
**Probable:** A clinically compatible case with evidence of infection such as  
  - An ultrasound showing *Ascaris* spp. worms in the pancreas or liver, OR  
  - CT scans or MRI showing *Ascaris* spp. worms present in the ducts of the liver or pancreas.  
| • Microscopic identification of *Ascaris* spp. (*A.lumbricoides* or *A. suum*) eggs in stool specimens,  
  OR  
  • Microscopic identification of ascarid larvae in sputum or gastric washings, OR  
  • Identification of *A. lumbricoides* or *A. suum* adult worms passed from the anus, mouth, or nose |
| **Babesiosis**  | Babesiosis is a parasitic disease caused by organisms in the *Babesia* genus. Infection can range from subclinical to life-threatening. Clinical manifestations can include hemolytic anemia and nonspecific influenza-like signs and symptoms (e.g., fever, chills, sweats, headache, myalgia, arthralgia, malaise, fatigue, and generalized weakness), splenomegaly, hepatomegaly, or jaundice. Laboratory findings can include thrombocytopenia, proteinuria, hemoglobinuria, and elevated levels of liver enzymes, blood urea nitrogen, and creatinine. Severe cases can be associated with marked thrombocytopenia, disseminated intravascular coagulation, hemodynamic instability, acute respiratory distress, myocardial infarction, renal failure, hepatic compromise, altered mental status, and death.  
**Objective Clinical Criteria:** fever, anemia, and/or thrombocytopenia  
**Subjective Clinical Criteria:** sweats, headache, myalgia, arthralgia, and/or chills  
**Confirmed:** A case that is laboratory confirmed AND meets at least one objective or subjective clinical criterion  
**Probable:** A case that:  
  - Has at least one supportive laboratory result (criteria listed below) AND meets at least one objective clinical criterion (subjective clinical criteria alone are not sufficient)  
  - IFA total immunoglobulin (Ig) or IgG titer:  
    - *B. microti:* ≥1:256 (≥1:64 in epidemiologically linked blood donors or recipients)  
    - *B. divergens:* ≥1:256  
    - *B. duncanii:* ≥1:512  
  - Immunoblot IgG: *B. microti* positive result, OR  
  - Is a blood donor or recipient epidemiologically linked to a confirmed or probable babesiosis case, AND  
  - Has confirmatory laboratory evidence but does not satisfy objective or subjective clinical criterion, OR  
  - Satisfies the supportive laboratory criteria (same as above)  
**Suspect:** A case that has confirmatory or supportive laboratory results, but insufficient clinical or epidemiological information is available for case classification  
| • Identification of intraerythrocytic *Babesia* organisms by light microscopy in a Giemsa, Wright, or Wright-Giemsa–stained blood smear,  
  OR  
  • Detection of *Babesia* spp. DNA in a whole blood specimen by PCR, OR  
  • Detection of *Babesia* spp. genomic sequences in a whole blood specimen by nucleic acid amplification, OR  
  • Isolation of *Babesia* organisms from a whole blood specimen by animal inoculation |
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| Botulism, foodborne    | **Confirmed:** A clinically compatible case that is laboratory confirmed or that occurs among persons who ate the same food as persons who have laboratory confirmed botulism. **Probable:** A clinically compatible case with a history of ingestion of a food item known to carry a risk for the botulism toxin | • Detection of botulinum toxin in serum, stool/enema gastric aspirate/vomit or patient's food, OR  
  • Isolation of *Clostridium botulinum* from stool/enema or gastric aspirate/vomit  
  Note: As required by *TAC* all *Clostridium botulinum* isolates must be submitted to the DSHS Laboratory. |
| 10530                  | Ingestion of botulinum toxin results in an illness of variable severity. Common symptoms include diplopia, blurred vision, slurred speech, difficulty swallowing, and bulbar weakness. Symmetric descending paralysis can progress rapidly.                                                              |
| Botulism, infant       | **Confirmed:** A clinically compatible case that is laboratory confirmed, occurring in a child aged less than 1 year.                                                                                                                      | • Detection of botulinum toxin in stool/enema or serum, OR  
  • Isolation of *Clostridium botulinum* from stool/enema  
  Note: As required by *TAC* all *Clostridium botulinum* isolates must be submitted to the DSHS Laboratory. |
| 10540                  | An illness of infants, characterized by constipation, poor feeding, altered cry, and “failure to thrive” that can be followed by progressive weakness, impaired respiration, and death.                                                                                         |
| Botulism, other unspecified | Ingestion of botulinum toxin results in an illness of variable severity. Common symptoms include diplopia, blurred vision, slurred speech, difficulty swallowing, and bulbar weakness. Symmetric descending paralysis can progress rapidly.                | • Detection of botulinum toxin in clinical specimen, OR  
  • Isolation of *Clostridium botulinum* from clinical specimen  
  Note: As required by *TAC* all *Clostridium botulinum* isolates must be submitted to the DSHS Laboratory. |
| 10548                  | **Confirmed:** A clinically compatible case that is laboratory confirmed in a patient aged greater than or equal to 1 year who has no history of ingestion of suspect food and has no wounds                                                                                     |
| Botulism, wound        | **Confirmed:** A clinically compatible case that is laboratory confirmed in a patient who has no suspected exposure to contaminated food and who has a history of a fresh, contaminated wound during the 2 weeks before onset of symptoms, or a history of injection drug use within the 2 weeks before onset of symptoms  | • Detection of botulinum toxin in stool/enema or serum, OR  
  • Isolation of *Clostridium botulinum* from wound or stool/enema  
  Note: As required by *TAC* all *Clostridium botulinum* isolates must be submitted to the DSHS Laboratory. |
| 10549                  | An illness resulting from toxin produced by *Clostridium botulinum* that has infected a wound. Common symptoms include diplopia, blurred vision, slurred speech, difficulty swallowing, and bulbar weakness. Symmetric descending paralysis can progress rapidly.           | • Detection of botulinum toxin in stool/enema or serum, OR  
  • Isolation of *Clostridium botulinum* from wound or stool/enema  
  Note: As required by *TAC* all *Clostridium botulinum* isolates must be submitted to the DSHS Laboratory. |
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| **Brucellosis**   | **10020** An illness that can cause a range of clinical signs and symptoms. Initial signs and symptoms may include fever, sweats, malaise, anorexia, headache, myalgia, arthralgia and/or fatigue. Chronic signs and symptoms may include recurrent fevers, arthritis, epididymitis, orchitis, endocarditis, hepatomegaly, splenomegaly, neurologic symptoms, chronic fatigue, and/or depression.  
*Confirmed:* A clinically compatible illness that is laboratory confirmed  
*Probable:* A clinically compatible case with at least one of the following:  
  ▪ Epidemiologically linked to a confirmed human or animal brucellosis case, **OR**  
  ▪ *Brucella* total antibody titer ≥1:160 by standard tube agglutination test (SAT) or by *Brucella* microagglutination test (BMAT) in one or more serum specimens obtained after onset of symptoms, **OR**  
  ▪ Detection of *Brucella* DNA in a clinical specimen by PCR assay | ▪ Culture and identification of *Brucella* spp. from clinical specimens,  
**OR**  
▪ Four-fold or greater rise in *Brucella* agglutination titer between acute- and convalescent-phase serum specimens obtained greater than or equal to 2 weeks apart and tested at the same laboratory  
Note: As required by **TAC**, all *Brucella* spp. isolates must be submitted to the DSHS Laboratory. |
| **Candida auris, Clinical Case** | **50263**  
**Candida auris, Colonization/Screening Cases** | **50264**  
*Candida auris* (*C. auris*) is an emerging multidrug-resistant yeast that can cause invasive infections and is associated with high mortality. Some strains of *C. auris* are resistant to the three major classes of antifungals, severely limiting treatment options. *C. auris* can spread in healthcare settings and cause outbreaks. *C. auris* can colonize patients’ skin and other body sites, perhaps indefinitely, and colonization poses a risk both for invasive infection and transmission. *C. auris* persists in the healthcare environment for weeks, and certain routinely used disinfectants in healthcare settings are not effective against the organism. Past epidemiological investigations have demonstrated that one-third to half of all patients on a given unit, especially in a long-term care setting, can become colonized with *C. auris* within weeks of an index patient entering the facility.  
*Confirmed:* A case that has confirmatory laboratory test.  
*Probable:* A case that has an isolate that is a *Candida haemulonii* or a yeast isolate that was unable to be identified that is from a person who is within same household, same healthcare facility, or in a healthcare facility that commonly shares patients with a facility, with another person with confirmatory laboratory evidence. | ▪ Confirmatory laboratory evidence: Detection of *C. auris* from any body site using either culture or a culture independent diagnostic test (CIDT) (e.g., Polymerase Chain Reaction [PCR]).  
Note: As required by **TAC**, all isolates identified as *Candida auris* must be submitted to the DSHS Laboratory.  
Any yeast isolate identified as *C. haemulonii* or any yeast isolate that had identification attempted without successful identification can be sent to DSHS Laboratory.  
Please contact a DSHS HAI Epidemiologist or the DSHS Laboratory for additional information on available laboratory support. |
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| **Campylobacteriosis**<sup>11020</sup> | An illness of variable severity commonly manifested by diarrhea, abdominal pain, nausea and sometimes vomiting. The organism may also rarely cause extra-intestinal infections such as bacteremia, meningitis or other focal infections.  

**Confirmed:** A case that is laboratory confirmed  

**Probable:**  
- A case with *Campylobacter* spp. detected in a clinical specimen using a culture independent diagnostic test (CIDT)  
- **OR**  
  - A clinically compatible case that is epidemiologically linked to a case that meets the probable or confirmed laboratory criteria for diagnosis  

**Notes:**  
- A case should not be counted as a new case if laboratory results were reported within 30 days of a previously reported infection in the same individual, unless additional information is available indicating a separate infection, e.g., different species. | • Isolation of *Campylobacter* spp. in a clinical specimen |
### Carbenenem-resistant Enterobacterales (CRE) 77924

**Carbenenemase producing CRE (CP-CRE)**

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<td>Carbapenem-resistant Enterobacterales (CRE) 77924</td>
<td>Carbapenem-resistant Enterobacterales (previously known as Enterobacteriaceae), are gram-negative bacilli that are resistant to at least one of the carbapenem antibiotics (ertapenem, meropenem, doripenem, or imipenem) or produce a carbapenemase (an enzyme that can make them resistant to carbapenem antibiotics). CRE can colonize or infect any body site. The most common types of CRE infections include bloodstream infections, ventilator-associated pneumonia, and intra-abdominal abscesses. Common causes of CRE infections in healthcare settings include <em>Klebsiella</em> species, and <em>Escherichia coli</em>. Although <em>Enterobacter</em> species can be resistant to carbapenem antibiotics, <em>Enterobacter</em> species are not included in this CRE definition. <em>Klebsiella aerogenes</em>, previously known as <em>Enterobacter aerogenes</em>, does meet the case definition. CREs are either non-carbapenemase producing (NCP-CRE) or carbapenemase producing (CP-CRE). CRE that produce carbapenemases, enzymes that break down carbapenems and related antimicrobials making them ineffective, are called carbapenemase-producing CRE (CP-CRE). Carbapenemase production by Enterobacterales can occur by many different mechanisms, such as <em>Klebsiella pneumoniae carbapenemase</em> (KPC), New Delhi Metallo-beta-lactamase (NDM), Verona Integron-Encoded Metallo-beta-lactamase (VIM), Imipenemase (IMP) and Oxacillinase-48 (OXA-48). NCP-CRE become resistant to carbapenems through a combination of chromosomal mutations and acquired non-carbapenemase resistance mechanisms. NCP-CRE, by antibiotic sensitivity testing, are resistant to at least one of the carbapenem antibiotics (ertapenem, meropenem, doripenem, or imipenem) in the absence of confirmatory CP-CRE test results. <strong>Confirmed</strong>: A case that is laboratory confirmed</td>
<td>CP-CRE: Any <em>Klebsiella</em> species, <em>E. aerogenes</em> or <em>E. coli</em> from any body site that is confirmed by: Laboratory evidence of carbapenemase production in an isolate:  - Positive phenotypic test:     - Carba NP     - Metallo-β-lactamase testing (e.g., E-test) positive     - Modified Carbenem Inactivation Method (mCIM) positive or indeterminate     - Carbapenem Inactivation Method (CIM) positive     - Modified Hodge Test (MHT) positive     - Positive phenotypic test results (e.g., mCIM, CIM, CarbaNP) but negative molecular method test results.  - Molecular methods:     - PCR positive (for KPC, NDM, OXA-48, IMP, or VIM)     - Xpert Carba-R positive (for KPC, NDM, OXA-48, VIM, IMP)     - PCR or Xpert Carba-R positive for novel carbapenemase  NCP-CRE(CRE): Any <em>Klebsiella</em> species, <em>E. aerogenes</em> or <em>E. coli</em> that does not meet CP-CRE criteria and is:  - Resistant to any carbapenem, including meropenem, imipenem, doripenem, or ertapenem, Note: There is no requirement to submit isolates to the DSHS Laboratory. Please contact a DSHS HAI Epidemiologist or the DSHS lab for additional information on available lab support. If the CRE isolate is sent to the DSHS lab for additional testing, use the submitting lab’s antibiotic susceptibility testing results to meet the epi case criteria.</td>
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- Note: Additional information on CRE can be found at [http://www.cdc.gov/HAI/organisms/cre/index.html](http://www.cdc.gov/HAI/organisms/cre/index.html)

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| Chagas disease, acute 12041 | Chagas disease is a parasitic infection caused by *Trypanosoma cruzi*. The acute phase is characterized by the first 8 weeks of infection, detectable parasitemia, and asymptomatic (most common) or symptomatic manifestations of disease which can include any of the following: Fever, malaise, rash, body aches, headache, loss of appetite, vomiting, diarrhea, hepatomegaly, splenomegaly, lymphadenopathy, Chagoma (nodular swelling at site of inoculation), Romaña's sign (unilateral swelling of the eyelid), acute myocarditis, and/or meningoencephalitis. | **Confirmed:** A case (asymptomatic or symptomatic) that has confirmatory laboratory testing. Asymptomatic individuals must have evidence of parasitemia based on microscopy or PCR. **Notes:**  
  - *T. cruzi* IgM tests are unreliable and are thus insufficient evidence of infection.  
  - Samples forwarded to CDC for confirmatory testing which test negative cannot be classified as cases.  
  - Congenital infections are considered acute up to 8 weeks of age and can be diagnosed by confirmatory tests. Infants <12 months and epidemiologically-linked need to be retested after 12 months of age.  
  - Please refer to the DSHS website for guidance on Chagas disease testing:  
  - Identification of *T. cruzi* by microscopy including:  
    - Microscopic examination of *T. cruzi* by:  
      - Wet mount – motile trypanosomes  
      - Thick & thin smears - Giemsa stain  
    - Detection of *T. cruzi* DNA by PCR  
    - Positive diagnostic serology confirmed by testing at CDC  
  - Note: No single supportive test has the sensitivity and specificity to be relied on alone, thus two different methods or antibodies specific to *T. cruzi* are used. |
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| Chagas disease, chronic indeterminate | Following the acute phase, most infected people enter into a prolonged, asymptomatic form of disease (called “chronic indeterminate”) during which few or no parasites are found in the blood. During this time, most people are unaware of their infection. Many people remain asymptomatic for life and never develop chronic Chagas-related symptoms.  
  **Confirmed:** An asymptomatic case ≥12 months of age with confirmatory lab results  
  **Probable:** An asymptomatic case ≥12 months of age with a single *T. cruzi* IgG positive assay* with an IV (reference interval) ≥3.5, OR positive blood donor screening and a *T. cruzi* IgG positive assay* with an IV <3.5 (or no IV provided), OR two *T. cruzi* IgG positive assays* that are different test formats or kits (e.g. Lateral Flow Assay, Wiener ELISA, Hemagen ELISA)  
  **Suspect:** An asymptomatic case ≥12 months of age with positive (reactive) blood donor screening OR a single *T. cruzi* IgG positive assay* with an IV <3.5 (or no IV provided)  
  *DSHS or commercial lab  
  Notes:  
  ▪ Samples forwarded to CDC for confirmatory testing which test negative cannot be classified as cases.  
  ▪ Patients with positive diagnostic serology should have confirmatory testing performed at the CDC.  
  ▪ Patients with positive blood donor screening should have *T. cruzi* IgG testing at a commercial or public health lab.  
  ▪ Women with chronic indeterminate disease can transmit infection to their unborn babies. Infants <12 months of age with a mother from an endemic area, in absence of direct detection of the organism, cannot be classified or ruled out due to maternal antibodies; perform serology at 12 months of age and classify based on presence or absence of symptoms as chronic symptomatic or chronic indeterminate case definition.  
  ▪ Please refer to the DSHS website for guidance on Chagas disease testing: [www.dshs.texas.gov/IDCU/disease/Chagas/humans/](http://www.dshs.texas.gov/IDCU/disease/Chagas/humans/) | Detection of antibody specific to *T. cruzi* by TWO distinct diagnostic tests performed at CDC  
  Note: No single supportive test has the sensitivity and specificity to be relied on alone, thus two different methods or antibodies specific to *T. cruzi* are used. |
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<td>Chagas disease, chronic</td>
<td>Much like the chronic indeterminate phase, the chronic symptomatic phase of disease (more than 8 weeks post infection) is characterized by undetectable parasitemia. However, an estimated 20-30% of infected people will develop debilitating and sometimes life-threatening medical problems over the course of their lives. Complications of chronic Chagas disease may include heart rhythm abnormalities that can cause sudden death, a dilated heart that doesn’t pump blood well, and/or a dilated esophagus or colon, leading to difficulties with eating or passing stool. <strong>Confirmed:</strong> A clinically compatible case of physician-diagnosed chronic Chagas disease in a patient ≥12 months of age with confirmatory laboratory results</td>
<td></td>
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<tr>
<td>symptomatic 12042</td>
<td><strong>Probable:</strong> A clinically compatible case of physician-diagnosed chronic Chagas disease in a patient ≥12 months of age with a single <em>T. cruzi</em> IgG positive assay* with an IV (reference interval) ≥3.5, OR positive blood donor screening and a <em>T. cruzi</em> IgG positive assay* with an IV &lt;3.5 (or no IV provided), OR two <em>T. cruzi</em> IgG positive assays* that are different test formats or kits (e.g. Lateral Flow Assay, Wiener ELISA, Hemagen ELISA) <strong>Suspect:</strong> A clinically compatible case of physician-diagnosed chronic Chagas disease in a patient ≥12 months of age with positive (reactive) blood donor screening OR a single <em>T. cruzi</em> IgG positive assay* with an IV &lt;3.5 (or no IV provided)</td>
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|                             | **Notes:**  
|                             | ▪ Samples forwarded to CDC for confirmatory testing which test negative cannot be classified as cases.  
|                             | ▪ Patients with positive diagnostic serology should have confirmatory testing performed at the CDC.  
|                             | ▪ Patients with positive blood donor screening should have *T. cruzi* IgG testing at a commercial or state public health lab.  
|                             | ▪ Women with chronic indeterminate disease can transmit infection to their unborn babies. Infants < 12 months of age with a mother from an endemic area, in absence of direct detection of the organism, cannot be classified or ruled out due to maternal antibodies; perform serology at 12 months of age and classify based on presence or absence of symptoms as chronic symptomatic or chronic indeterminate case definition.  
|                             | ▪ Please refer to the DSHS website for guidance on Chagas disease testing: www.dshs.texas.gov/IDCU/disease/Chagas/humans/  |
|                             | *DSHS or commercial lab  |
|                             | Note: No single supportive test has the sensitivity and specificity to be relied on alone, thus two different methods or antibodies specific to *T. cruzi* are used.  |

**Laboratory Confirmation Tests:**  
- Detection of antibody specific to *T. cruzi* by TWO distinct diagnostic tests performed at CDC.
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<td>Chickenpox - (see Varicella)</td>
<td>See Varicella</td>
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| Cholera (toxigenic *Vibrio cholerae* O1 or O139) 10470 | An illness characterized by profuse watery diarrhea and/or vomiting; severity is variable. **Confirmed:** A clinically compatible illness that is laboratory confirmed  
Note: Illnesses caused by strains of *V. cholerae* other than toxigenic *V. cholerae* O1 or O139 should not be reported as cases of cholera. (See *Vibrio parahaemolyticus*, *Vibrio vulnificus*, and *Vibriosis, other or unspecified*) | • Isolation of toxigenic (i.e., cholera toxin-producing) *Vibrio cholerae* O1 or O139 from stool or vomitus,  
**OR**  
• Serologic evidence of recent infection  
Note: As required by *TAC* all *Vibrio* species isolates must be submitted to the DSHS Laboratory. |
Contaminated sharps injury

A contaminated sharps injury that occurs in a health care setting that is contaminated with human blood or body fluids should be reported per the below guidelines. Contaminated sharps injuries in private facilities must be documented per OSHA guidelines. http://www.osha.gov/SLTC/etools/hospital/hazards/sharps/sharps.html

Contaminated sharps injuries in Texas public facilities (government entities) are reported to DSHS Emerging and Acute Infectious Disease Branch. The facility where the injury occurred should complete the reporting form and submit it to the local health authority where the facility is located. If no local health authority is appointed for this jurisdiction, submit to the regional director of the Texas Department of State Health Services (TDSHS) regional office in which the facility is located. Address information for regional directors can be obtained at http://www.dshs.state.tx.us/regions/default.shtm. The local health authority, acting as an agent for the TDSHS will receive and review the report for completeness, and submit the report to:

Texas Department of State Health Services
Emerging and Acute Infectious Disease Branch
PO Box 149347 (Mail Code 1960), Austin, Texas 78714-9347
Fax number: 512-776-7616

The reporting forms can be found at http://www.dshs.state.tx.us/idcu/health/infection_control/bloodborne_pathogens/reporting/


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| **Cryptosporidiosis**<br>11580 | A gastrointestinal illness characterized by diarrhea and one or more of the following: diarrhea duration of 72 hours or more, abdominal cramping, vomiting, or anorexia.  
**Confirmed:** A case that is laboratory confirmed  
**Probable:**  
- A case with *Cryptosporidium* antigen detected by a screening test method such as, the immunochromatographic card/rapid card test or a laboratory test of unknown method  
  **OR**  
- A clinically compatible case that is epidemiologically linked to a confirmed case by one of the following means:  
  - Household or other close contact to a lab-confirmed case with onset of symptoms within 1 month (before or after), **OR**  
  - Exposure to an outbreak at a body of water or water facility involving at least 2 lab-confirmed cases and onset of symptoms within one month (before or after) of one or more of these cases  
Note: A case should not be counted as a new case if laboratory results were reported within 365 days of a previously reported infection in the same individual, unless additional information is available indicating a separate infection | - Detection of *Cryptosporidium* organisms or DNA in stool, intestinal fluid, tissue samples, biopsy specimens, or other biological sample by certain laboratory methods with a high positive predictive value (PPV):  
  - Direct fluorescent antibody (DFA) test,  
  **OR**  
  - Polymerase chain reaction (PCR),  
  **OR**  
  - Enzyme immunoassay (EIA),  
  **OR**  
  - Light microscopy of stained specimen. |
| **Cyclosporiasis**<br>11575 | An illness of variable severity caused by the protozoan parasite *Cyclospora cayetanensis*. The most common symptom is watery diarrhea. Other symptoms include loss of appetite, weight loss, abdominal cramps/bloating, nausea, body aches, and fatigue. Vomiting and low-grade fever also may occur.  
**Confirmed:** A laboratory-confirmed case with clinical compatibility  
**Probable:** A clinically compatible case that is epidemiologically linked to a confirmed case  
Note: A case should not be counted as a new case if laboratory results were reported within 365 days of a previously reported infection in the same individual, unless additional information is available indicating a separate infection | - Detection of *Cyclospora* organisms by microscopic examination in stool, intestinal fluid/aspirate, or intestinal biopsy specimens,  
**OR**  
- Detection of *Cyclospora* DNA (by PCR) in stool, intestinal fluid/aspirate, or intestinal biopsy specimens |
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| Cysticercosis 12031 | Cysticercosis is a tissue infection caused by the larval form of the pork tapeworm, *Taenia solium*. Infection occurs when the tapeworm eggs are ingested, hatch into larvae, and migrate to tissues where they form cysticerci (cysts). The signs and symptoms of cysticercosis reflect the development of cysticerci in various sites. Subcutaneous cysticerci may be visible or palpable. When cysticerci are found in the brain, the condition is called neurocysticercosis, which can cause diverse manifestations including seizures, mental disturbances, focal neurologic deficits, and signs of space-occupying intracerebral lesions. Death can occur suddenly. Extracerebral cysticercosis can cause ocular, cardiac, or spinal lesions with associated signs and symptoms. Asymptomatic subcutaneous nodules and calcified intramuscular nodules can be encountered. **Confirmed:** Laboratory confirmation of the presence of cysticercus in tissue. Notes:  
- Documentation of biopsy or imaging results is required.  
- Demonstration of *T. solium* eggs and proglottids in the feces are diagnostic of taeniasis (see *Taenia solium and undifferentiated Taeniasis*), not cysticercosis. Persons who are found to have eggs or proglottids in their feces should be evaluated serologically since autoinfection, resulting in cysticercosis, can occur.  
- Blood tests are available to help diagnose an infection but are not always accurate. While suggestive, it does not necessarily prove that cysticercosis is present. | - Diagnosis of neurocysticercosis is usually made by MRI or CT brain scans in order to identify the presence of cysticerci. If surgery is necessary, confirmation of the diagnosis can be made by demonstrating the cysticercus in the tissue involved (biopsy).  
- Radiographs can identify calcified cysticerci in tissues other than the brain. |
<table>
<thead>
<tr>
<th>Dengue–like Illness</th>
<th>Dengue is a potentially fatal febrile illness caused by infection with any of the four dengue viruses (DENV-1, -2, -3 and -4). Dengue is transmitted primarily through the bite of <em>Aedes aegypti</em> and <em>Ae. albopictus</em> mosquitoes. For the purposes of surveillance and reporting, based on their clinical presentation, dengue cases can be categorized into three primary groups: dengue-like illness, dengue, and severe dengue.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical evidence of dengue-like illness:</td>
<td>Fever as reported by the patient or healthcare provider</td>
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<tr>
<td>Clinical evidence of dengue:</td>
<td>Fever as reported by the patient or healthcare provider and the presence of one or more of the following signs and symptoms:</td>
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<tr>
<td>■ nausea/vomiting</td>
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<td>■ rash</td>
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<tr>
<td>■ aches and pains (i.e. headache, retro-orbital pain, arthralgia, myalgia)</td>
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<tr>
<td>■ tourniquet test positive</td>
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<tr>
<td>■ leukopenia (a total white blood cell count of &lt;5,000/mm³)</td>
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<td>■ abdominal pain</td>
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<td>■ persistent vomiting</td>
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<td>■ extravascular fluid accumulation</td>
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<tr>
<td>■ mucosal bleeding</td>
<td></td>
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<tr>
<td>■ liver enlargement &gt;2 centimeters</td>
<td></td>
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<tr>
<td>■ increasing hematocrit concurrent with rapid decrease in platelet count</td>
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<tr>
<td>Clinical evidence of severe dengue:</td>
<td>Dengue with any one or more of the following scenarios:</td>
</tr>
<tr>
<td>■ severe plasma leakage evidenced by hypovolemic shock and/or extravascular fluid accumulation with respiratory distress</td>
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<tr>
<td>■ severe bleeding from the gastrointestinal tract or vagina as defined by requirement for medical intervention including intravenous fluid resuscitation or blood transfusion</td>
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<tr>
<td>■ severe organ involvement, including any of the following:</td>
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<td>■ elevated liver transaminases: aspartate aminotransferase (AST) or alanine aminotransferase (ALT) ≥1,000 units per liter (U/L)</td>
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<td>■ impaired level of consciousness and/or diagnosis of encephalitis, encephalopathy, or meningitis</td>
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<tr>
<td>■ heart or other organ involvement including myocarditis, cholecystitis, and pancreatitis</td>
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<tr>
<td><strong>Confirmed:</strong> A clinically compatible case of dengue-like illness, dengue, or severe dengue with confirmatory laboratory results</td>
<td></td>
</tr>
<tr>
<td><strong>Probable:</strong> A clinically compatible case of dengue-like illness, dengue, or severe dengue AND one of the following:</td>
<td>Detection of DENV nucleic acid in serum, plasma, CSF, other body fluid or tissue by validated RT-PCR, <strong>OR</strong></td>
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<tr>
<td>■ Detection of DENV antigen in tissue, by IHC, <strong>OR</strong></td>
<td>Detection in serum or plasma of DENV NS1 antigen by a validated immunoassay, <strong>OR</strong></td>
</tr>
<tr>
<td>■ Cell culture isolation of DENV from serum, plasma, or CSF specimen, <strong>OR</strong></td>
<td>Detection of IgM anti-DENV in serum or CSF in a traveler returning from a dengue endemic area without ongoing transmission of another flavivirus, clinical evidence of co-infection with a flavivirus or recent vaccination against a flavivirus, <strong>OR</strong></td>
</tr>
<tr>
<td>■ Detection of IgM anti-DENV in serum or CSF in a person living in a dengue endemic or non-endemic area of the US without evidence of other flavivirus transmission, <strong>OR</strong></td>
<td>IgM anti-DENV seroconversion by validated immunoassay in acute (i.e., collected &lt;5 days of illness onset) and convalescent (i.e., collected &gt;5 days after illness onset) serum specimens, <strong>OR</strong></td>
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<tr>
<td>■ IgG anti-DENV seroconversion or ≥4-fold rise in titer in serum specimens collected &gt;2 weeks apart, and confirmed by a normalization test (e.g., plaque reduction neutralization test) with a &gt;4-fold higher endpoint titer as compared to other flaviruses tested</td>
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<tr>
<td>Condition/Code</td>
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<tr>
<td>Diphtheria 10040</td>
<td>An upper respiratory tract illness with an adherent membrane of the nose, pharynx, tonsils, or larynx OR an infection of a non-respiratory anatomical site (e.g., skin, wound, conjunctiva, ear, genital mucosa)</td>
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<td></td>
<td><strong>Confirmed:</strong> A clinically compatible case that is either laboratory confirmed, OR epidemiologically linked to a laboratory-confirmed case OR An infection at a non-respiratory anatomical site (e.g., skin, wound, conjunctiva, ear, genital mucosa) with: • Isolation of toxin-producing <em>Corynebacterium diphtheriae</em> from that site</td>
</tr>
<tr>
<td></td>
<td><strong>Notes:</strong> • PCR and MALDI-TOF (matrix assisted laser desorption/ionization-time of flight mass spectrometry) diagnosis for <em>C. diphtheria</em>, when used alone, do not confirm toxin production. These tests, when used, should always be combined with a test that confirms toxin production, such as the Elek test. • Individuals without evidence of clinical criteria as described by the diphtheria surveillance case definition but for whom toxin-producing <em>C. diphtheria</em> is confirmed via laboratory testing (isolation and toxigenicity testing by modified Elek test or other validated test capable of confirming toxin-production) should not be classified as cases. These individuals are considered carriers of the bacteria and are not reportable.</td>
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<td></td>
<td><strong>Laboratory Confirmation Tests</strong></td>
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<tr>
<td></td>
<td>• Isolation of <em>Corynebacterium diphtheriae</em> from a clinical specimen, <strong>AND</strong> • Confirmation of toxin-production by Elek test or by another validated test capable of confirming toxin-production</td>
</tr>
</tbody>
</table>

- **Suspect:** A clinically compatible case of dengue-like illness, dengue, or severe dengue with an epidemiologic linkage, defined as:
  - Travel to a dengue endemic country or presence at a location with an ongoing outbreak within two weeks prior to onset of an acute febrile illness or dengue, **OR**
  - Association in time and place with a confirmed or probable dengue case

- **Laboratory Confirmation**

- Detection of IgM anti-DENV by validated immunoassay in serum or CSF in a traveler returning from a dengue endemic area with ongoing transmission of another flavivirus, clinical evidence of co-infection with one of these flaviviruses, or recent vaccination against a flavivirus

- **Suspect:** A clinically compatible case of dengue-like illness, dengue, or severe dengue with an epidemiologic linkage, defined as:
  - Travel to a dengue endemic country or presence at a location with an ongoing outbreak within two weeks prior to onset of an acute febrile illness or dengue, **OR**
  - Association in time and place with a confirmed or probable dengue case

Notes:
- PCR and MALDI-TOF (matrix assisted laser desorption/ionization-time of flight mass spectrometry) diagnosis for *C. diphtheria*, when used alone, do not confirm toxin production. These tests, when used, should always be combined with a test that confirms toxin production, such as the Elek test.
- Individuals without evidence of clinical criteria as described by the diphtheria surveillance case definition but for whom toxin-producing *C. diphtheria* is confirmed via laboratory testing (isolation and toxigenicity testing by modified Elek test or other validated test capable of confirming toxin-production) should not be classified as cases. These individuals are considered carriers of the bacteria and are not reportable.
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<tr>
<td>Ebola (HF) 11630</td>
<td>An illness with an incubation period of 2-21 days (average 8-10 days) characterized by abrupt onset of fever and typically accompanied by one or more of the following symptoms: severe headache, myalgia (muscle pain), maculopapular rash that can desquamate, vomiting, diarrhea, abdominal pain, bleeding not related to injury, or low platelet count (thrombocytopenia). Other symptoms and clinical findings may include chills, malaise, fatigue, weakness, nausea, decreased appetite, arthralgia, conjunctival injection (red eyes), sore throat, hiccups, chest pain, shortness of breath, confusion, seizures, cerebral edema, spontaneous miscarriage, symptoms of impaired kidney and liver function, elevated liver enzymes, or leukopenia frequently with lymphopenia followed later by elevated neutrophils and a left shift.</td>
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<td><strong>Confirmed</strong>: A person that meets laboratory criteria</td>
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<td></td>
<td><strong>Suspect</strong>: A person that meets the clinical criteria AND one or more of the epidemiologic risk factors within 21 days of onset of symptom onset</td>
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<td><strong>Clinical Criteria:</strong></td>
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<td></td>
<td>▪ Fever, AND</td>
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<td>▪ One or more of the following symptoms: severe headache, myalgia (muscle pain), erythematous maculopapular rash on the trunk with fine desquamation 3–4 days after rash onset, vomiting, diarrhea, abdominal pain, bleeding not related to injury, or thrombocytopenia</td>
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<td><strong>Epidemiologic Risk Factor Criteria:</strong></td>
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<td>▪ Direct contact with blood or body fluids of a person who is sick with or has died from Ebola Virus Disease (EVD), OR</td>
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<td></td>
<td>▪ Direct contact with objects (such as clothes, bedding, needles and syringes) contaminated with blood or body fluids from a person who is sick with or has died from EVD, OR</td>
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<td>▪ Work in a laboratory that handles, or direct contact with primates or bats from an Ebola virus endemic area or area with active transmission, OR</td>
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<td>▪ Sexual exposure to semen from a confirmed acute or clinically recovered case of EVD or exposure to breast milk of an individual who had EVD, OR</td>
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<td></td>
<td>▪ Work in a laboratory that handles EVD specimens, OR</td>
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<td></td>
<td>▪ Residence in - or travel to - an EVD endemic area or area of active transmission</td>
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<td></td>
<td><strong>Laboratory Confirmation Tests:</strong></td>
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<tr>
<td></td>
<td>▪ Detection of Ebola virus genetic sequence by real-time RT-PCR from blood or tissues, OR</td>
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<td>▪ Isolation of Ebola virus in cell culture for blood or tissues, OR</td>
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<td>▪ Detection of Ebola virus antigens in blood by ELISA, OR</td>
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<td>▪ Detection of Ebola virus antigens in tissues by immunohistochemistry (IHC)</td>
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<tr>
<td><strong>Echinococcosis</strong>&lt;br&gt;80670</td>
<td>Echinococcosis is an infection caused by the larval stage of tapeworms in the genus <em>Echinococcus</em>, including <em>E. granulosus</em> and <em>E. multilocularis</em>. Transmission occurs through the ingestion of tapeworm eggs in contaminated food, water, soil, dog feces, or on the contaminated coats of dogs and cats. Infection may also occur through the ingestion of cysts in the undercooked internal organs of infected intermediate hosts, such as sheep, goats and swine. Many infections are asymptomatic for years before the growing cysts cause clinical signs and symptoms associated with the affected organs. Liver involvement is associated with abdominal pain, hepatic masses, and biliary duct obstruction. Pulmonary involvement can produce chest pain, cough, and hemoptysis. Other organs, including the brain, bone, and heart, may also be involved with resulting clinical signs and symptoms. Ruptured cysts may cause fever, urticaria (hives), eosinophilia and anaphylactic shock. <strong>Confirmed:</strong> An asymptomatic or symptomatic case that meets one or more confirmatory laboratory criteria. <strong>Probable:</strong> An asymptomatic or symptomatic case with <em>Echinococcus</em>-specific antibodies identified by TWO different types of serological assays.</td>
<td>• Detection of cysts or organ lesions using imaging techniques, including CT, MRI, and ultrasonography AND detection of <em>Echinococcus</em>-specific antibodies, OR • Detection of <em>Echinococcus</em> spp. DNA by PCR in a clinical specimen, OR • Histopathology or parasitology results compatible with <em>Echinococcus</em> spp. (i.e., direct visualization of the protoscolex in cyst fluid)</td>
</tr>
<tr>
<td><strong>Ehrlichiosis (Ehrlichia chaffeensis infection)</strong>&lt;br&gt;11088</td>
<td>Ehrlichiosis is a group of tick-borne diseases caused by <em>Ehrlichia</em> species, obligate intracellular bacteria that infect peripheral blood leukocytes. <em>Ehrlichia chaffeensis</em> is transmitted by the bite of infected lone star ticks. Initial symptoms may include fever/chills, headache, myalgia, nausea/vomiting, confusion, and rash. <em>E. chaffeensis</em> disease may result in severe illness or even death in older or immunocompromised individuals or if treatment is delayed. <strong>Clinical evidence:</strong> Fever as reported by patient or provider and one or more of the following: headache, myalgia, anemia, leukopenia, thrombocytopenia, or any hepatic transaminase elevation. <strong>Confirmed:</strong> A clinically compatible illness that is laboratory confirmed <strong>Probable:</strong> A clinically compatible illness with serological evidence of IgG or IgM antibody reactive (≥1:128) with <em>E. chaffeensis</em> antigen by IFA, OR identification of morulae in the cytoplasm of monocytes or macrophages by microscopic examination <strong>Suspect:</strong> A case with laboratory evidence of past/present infection with <em>E. chaffeensis</em> (e.g., laboratory report) but no available clinical information</td>
<td>• Demonstration of a four-fold change in IgG-specific antibody titer to <em>E. chaffeensis</em> antigen by IFA in paired serum samples (preferably one taken in first week of illness and a second taken 2-4 weeks later), OR • Detection of <em>E. chaffeensis</em> DNA in a clinical specimen by PCR, OR • Demonstration of ehrlichial antigen in a biopsy/autopsy sample by IHC, OR • Isolation of <em>E. chaffeensis</em> from a clinical specimen in cell culture</td>
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<tr>
<td>Condition/Code</td>
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| **Ehrlichiosis** *(Ehrlichia ewingii infection)*  
11089 | Ehrlichiosis is a group of tick-borne diseases caused by *Ehrlichia* species, obligate intracellular bacteria that infect peripheral blood leukocytes. *Ehrlichia ewingii* is transmitted by the bite of infected lone star ticks. Symptoms are similar to that of *E. chaffeensis* disease; however gastrointestinal symptoms are less common, rash is rare, and fewer severe manifestations have been reported.  
*Clinical evidence:* Fever as reported by patient or provider and one or more of the following: headache, myalgia, anemia, leukopenia, thrombocytopenia, or any hepatic transaminase elevation.  
*Confirmed:* A clinically compatible illness that is laboratory confirmed  
*Suspect:* A case with laboratory evidence of past/present infection with *E. ewingii* (e.g., laboratory report) but no available clinical information | Detection of *E. ewingii* DNA in a clinical specimen by PCR  
Note: Because the organism has never been cultured, antigens are not available. Thus, *E. ewingii* infections can only be diagnosed by molecular detection methods. |
| **Ehrlichiosis/Anaplasmosis – undetermined**  
11091 | There are at least three species of intracellular bacteria responsible for ehrlichiosis/anaplasmosis in the US (*Ehrlichia chaffeensis*, *E. ewingii*, and *Anaplasma phagocytophilum*). The clinical signs of disease that result from infection with these bacteria are similar, their geographic ranges overlap, and serologic cross-reactions may occur among tests for these agents.  
*Clinical evidence:* Fever as reported by patient or provider and one or more of the following: headache, myalgia, anemia, leukopenia, thrombocytopenia, or any hepatic transaminase elevation.  
*Probable:* A clinically compatible illness with serological evidence of IgG or IgM antibody reactive (≥1:128) with *Ehrlichia/Anaplasma* spp. by IFA, OR identification of morulae in white cells by microscopic examination in the absence of other supportive lab results  
*Suspect:* A case with laboratory evidence of past/present infection with undetermined *Ehrlichia/Anaplasma* spp. but no available clinical information  
Note: For ehrlichiosis/anaplasmosis, an undetermined case can only be classified as probable. This occurs when a case has compatible clinical criteria with laboratory evidence to support infection, but not with sufficient clarity to identify the organism as *E. chaffeensis*, *A. phagocytophilum*, or *E. ewingii*. This can include the identification of morulae in white cells by microscopic examination in the absence of other supportive laboratory results. | Not applicable - See note |
| *Escherichia coli*, Shiga toxin-producing (STEC) | See [Shiga toxin-producing *Escherichia coli* (STEC)](https://www.cdc.gov/ncidod/dvbid/stec) |  |

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**Notes:**
- Detection of *E. ewingii* DNA in a clinical specimen by PCR.
- Detection of *E. ewingii* DNA in a clinical specimen by PCR.
- Detection of *E. ewingii* DNA in a clinical specimen by PCR.
- Detection of *E. ewingii* DNA in a clinical specimen by PCR.
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| Fascioliasis 80663 | *Fasciola hepatica* and *Fasciola gigantica* (liver flukes) are transmitted by ingesting raw aquatic plants or water contaminated with immature larvae, usually in locations around domestic and wild ruminants (commonly sheep, cattle and goats). Infection may or may not be symptomatic. In early infection (acute phase), the immature larval flukes migrate through the intestinal wall, the abdominal cavity, and the liver tissue, into the bile ducts, where they develop into mature adult flukes. Symptoms may include fever; gastrointestinal problems such as nausea, vomiting and diarrhea; a swollen liver (hepatomegaly); liver function abnormalities, skin rashes; shortness of breath; and abdominal pain or tenderness. The chronic phase (after the parasite settles in the bile ducts), is marked by inflammation and hyperplasia and thickening of the bile ducts and gall bladder, leading to biliary lithiasis or obstruction. Symptoms of this phase may include: biliary colic, nausea, intolerance to fatty food, right upper quadrant pain, epigastric pain, obstructive jaundice, and pruritus, are the result of a blockade in the biliary tract and inflammation in the gall bladder. Inflammation of the liver, gallbladder, and pancreas can also occur.  
**Confirmed:** A case that is laboratory confirmed  
**Probable:** A clinically compatible case with  
- Detection of *Fasciola* antibodies, **OR**  
- History of ingestion of watercress or freshwater plants and eosinophilia  
- Microscopic identification of *Fasciola* eggs in feces, duodenal contents, or bile, **OR**  
- Microscopic identification of a *Fasciola* adult fluke extracted from a clinical specimen (e.g. bile ducts), **OR**  
- Detection of *Fasciola* coproantigens (antigens found in feces) by ELISA |  |
| Granulomatous amebic encephalitis (GAE) |  
- See Amebic meningitis/encephalitis, other |  |
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| **Haemophilus influenzae, invasive disease**<br>10590 | Invasive *Haemophilus influenzae* may manifest as pneumonia, bacteremia, meningitis, epiglottitis, septic arthritis, cellulitis, or purulent pericarditis; less common infections include endocarditis and osteomyelitis  | • Detection of *Haemophilus influenzae* type b antigen in cerebrospinal fluid [CSF]  
• Detection of *Haemophilus influenzae*-specific nucleic acid in a specimen obtained from a normally sterile body site (e.g., blood or CSF), using a validated polymerase chain reaction (PCR) assay;  
• Isolation of Haemophilus influenzae from a normally sterile body site (e.g., cerebrospinal fluid [CSF], blood, joint fluid, pleural fluid, pericardial fluid)  
See [Normally Sterile Site](#)  
Note: Serotyping of isolates can be performed at the DSHS laboratory. Serotyping is recommended for all *H. influenzae* cases and required by **TAC** on isolates from children under 5 years old. |

*Confirmed:* A case that is laboratory confirmed  
*Probable:* Meningitis with detection of *H. influenzae* type b antigen in cerebrospinal fluid (CSF). (Antigen test results in urine or serum are unreliable for diagnosis of *H. influenzae* disease.)
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| Hantavirus infection, non-HPS 11610 | Hantaviruses are rodent-borne viruses that can be transmitted to humans. Patients with hantavirus infection typically present with nonspecific signs and symptoms including fever, myalgia, headache, and chills. After the prodromal phase, symptoms of hantavirus pulmonary syndrome (HPS) may develop.  
Non-HPS hantavirus infection is a febrile illness with non-specific signs and symptoms including fever, chills, myalgia, headache, and gastrointestinal symptoms, but no cardiopulmonary symptoms. Clinical laboratory findings may include hemoconcentration, left shift in white blood cell count, neutrophilic leukocytosis, thrombocytopenia, and circulating immunoblasts.  
HPS is an acute febrile illness characterized by non-specific viral symptoms including fever, chills, myalgia, headache, and gastrointestinal symptoms, and one or more of the following clinical features:  
- Bilateral diffuse interstitial edema, OR  
- Clinical diagnosis of acute respiratory distress syndrome (ARDS), OR  
- Radiographic evidence of noncardiogenic pulmonary edema, OR  
- Unexplained respiratory illness resulting in death, and includes autopsy examination demonstrating noncardiogenic pulmonary edema without an identifiable cause, OR  
- Healthcare record with a diagnosis of HPS OR  
- Death certificate that lists HPS as a cause of death or a significant condition contributing to death  
**Confirmed:** A clinically compatible case of HPS or non-HPS hantavirus infection with confirmatory laboratory results  
- Detection of hantavirus-specific IgM* or rising titers of hantavirus-specific IgG, OR  
- Detection of hantavirus-specific ribonucleic acid sequence in clinical specimens, OR  
- Detection of hantavirus antigen by IHC in lung biopsy or autopsy tissues  
*Due to the high rate of false positives at commercial labs, a sample should be forwarded to DSHS for confirmatory testing |
| Hantavirus pulmonary syndrome 11590 |                                                                                                                                                                                                                                                |                                                                                                                                                                                                                              |
| Hemolytic uremic syndrome, post-diarrheal (HUS) 11550 | Hemolytic uremic syndrome (HUS) is characterized by the acute onset of microangiopathic hemolytic anemia, renal injury, and low platelet count. Thrombotic thrombocytopenic purpura (TTP) also is characterized by these features but can include central nervous system (CNS) involvement and fever and can have a more gradual onset. Most cases of HUS (but few cases of TTP) occur after an acute gastrointestinal illness (usually diarrhea).  
**Confirmed:** An acute illness diagnosed as HUS or TTP that both meets the laboratory criteria and began within 3 weeks after onset of an episode of acute or bloody diarrhea  
**Probable:**  
- An acute illness diagnosed as HUS or TTP that meets the laboratory criteria in a patient who does not have a clear history of acute or bloody diarrhea in preceding 3 weeks, OR  
- An acute illness diagnosed as HUS or TTP, that a) has onset within 3 weeks after onset of an acute or bloody diarrhea and b) meets the laboratory criteria except that microangiopathic changes are not confirmed  
**Note:** See [Shiga toxin-producing Escherichia coli (STEC)](https://www.cdc.gov/hantavirus/around_the_world/index.html) as cases that meet the HUS case criteria should also be reported as a “Suspect” STEC case, unless other criteria is met for another case definition.  
**The following are both present at some time during the illness:**  
- Anemia (acute onset) with microangiopathic changes (i.e., schistocytes, burr cells, or helmet cells) on peripheral blood smear, AND  
- Renal injury (acute onset) evidenced by either hematuria, proteinuria, or elevated creatinine level (i.e., greater than or equal to 1.0 mg/dL in a child aged less than 13 years or greater than or equal to 1.5 mg/dL in a person aged greater than or equal to 13 years, or greater than or equal to 50% increase over baseline)  
**Note:** A low platelet count can usually, but not always, be detected early in the illness, but it can then become normal or even high. If a platelet count obtained within 7 days after onset of the acute gastrointestinal illness is not less than 150,000/mm3, other diagnoses should be considered. |
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<tr>
<td><strong>Hepatitis A, acute</strong>&lt;br&gt;10110</td>
<td>An acute illness with a discrete onset of any sign or symptom consistent with acute viral hepatitis (e.g., fever, headache, malaise, anorexia, nausea, vomiting, diarrhea, abdominal pain, or dark urine), <strong>AND</strong> a) either jaundice or elevated total bilirubin levels ≥ 3.0 mg/dL, <strong>OR</strong> elevated serum alanine aminotransferase (ALT) levels &gt;200 IU/L, <strong>AND</strong> b) the absence of a more likely diagnosis.&lt;br&gt;&lt;br&gt;<strong>Confirmed:</strong>&lt;br&gt;• A case that meets the clinical case criteria and is IgM anti-HAV positive, <strong>OR</strong>&lt;br&gt;• A case that has hepatitis A virus RNA detected by NAAT (such as PCR or genotyping), <strong>OR</strong>&lt;br&gt;• A case that meets the clinical criteria and occurs in a person who has an epidemiological link with a person who had contact (e.g., household or sexual) with a laboratory-confirmed hepatitis A case 15-50 days prior to the onset of symptoms. <strong>AND</strong>&lt;br&gt;• A case that is not otherwise ruled out by IgM anti-HAV or NAAT for hepatitis A virus testing performed in a public health laboratory.&lt;br&gt;&lt;br&gt;Note: Hepatitis A is usually self-limiting and does not result in chronic infection. However, up to 10% of persons with hepatitis A may experience a relapse during the 6 months after acute illness. Cases of relapsing hepatitis A should not be enumerated as new cases. In addition, a case should not be counted as a hepatitis A case if there is an alternate, more likely diagnosis.</td>
<td>• Immunoglobulin M antibody to hepatitis A virus (anti-HAV IgM) positive, <strong>OR</strong>&lt;br&gt;• Nucleic acid amplification test (NAAT; such as Polymerase Chain Reaction [PCR] or genotyping) for hepatitis A virus RNA positive</td>
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<td><strong>Hepatitis B, acute</strong>&lt;br&gt;10100</td>
<td>An acute illness with a discrete onset of any sign or symptom* consistent with acute viral hepatitis (e.g., fever, headache, malaise, anorexia, nausea, vomiting, diarrhea, and abdominal pain), <strong>AND</strong> either b) jaundice, or c) elevated serum alanine aminotransferase levels (ALT) &gt;100 IU/L.&lt;br&gt;&lt;br&gt;<strong>Confirmed:</strong> A case that meets the clinical case definition, is laboratory confirmed, and is not known to have chronic hepatitis B**&lt;br&gt;&lt;br&gt;*A documented negative hepatitis B surface antigen (HBsAg) laboratory test result within 6 months prior to a positive test result (i.e., HBsAg, hepatitis B “e” antigen [HBeAg], or hepatitis B virus nucleic acid testing [HBV NAT] including genotype) does not require an acute clinical presentation to meet the surveillance case definition. **A person should be considered chronically infected if hepatitis B antigen tests (HBsAg, HBeAg, and/or nucleic acid tests) have been positive for 6 months or longer or if the patient has a history of chronic hepatitis B diagnosis.</td>
<td>• Hepatitis B surface antigen (HBsAg) positive, <strong>AND</strong>&lt;br&gt;• IgM antibody to hepatitis B core antigen (anti-HBc IgM) positive (if done)</td>
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| Hepatitis B virus infection, perinatal 10104 | Perinatal hepatitis B (HBV) in the newborn can range from asymptomatic to fulminant hepatitis.  
**Confirmed:** Child born in the US to a HBV-infected mother and positive for HBsAg at ≥ 1 month of age and ≤ 24 months of age OR positive for HBeAg or HBV DNA ≥9 months of age and ≤ 24 months of age.  
**Probable:** Child born in the US and positive for HBsAg at ≥ 1 month of age and ≤ 24 months of age OR positive for HBeAg or HBV DNA ≥9 months of age and ≤ 24 months of age, but whose mother’s hepatitis B status is unknown (i.e. epidemiologic linkage not present).  
Notes:  
▪ If the mother is known to be NOT infected with HBV, refer to the case definition for acute Hepatitis B.  
These definitions are used for surveillance purposes only, not for perinatal hepatitis B prevention case management purposes. | **Hepatitis B surface antigen (HBsAg) positive, hepatitis B e antigen (HBeAg) positive, or detectable Hepatitis B virus DNA (HBV DNA)**  
Note: HBsAg must be tested more than 4 weeks after last dose of hepatitis B vaccine to be considered confirmatory. |
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| **Hepatitis C, acute**<br>10101 | All hepatitis C virus cases in each classification category should be > 36 months of age, unless known to have been exposed non-perinatally. **Clinical Criteria:**  
  ▪ Jaundice, OR  
  ▪ Peak total bilirubin levels ≥ 3.0 mg/DL, OR  
  ▪ Elevated serum alanine aminotransferase (ALT) level >200 IU/L, AND  
  ▪ The absence of a more likely diagnosis (which may include evidence of acute liver disease due to other causes or advanced liver disease due to pre-existing chronic Hepatitis C virus (HCV) infection or other causes, such as alcohol exposure, other viral hepatitis, hemochromatosis, etc.) **Confirmed:**  
  ▪ A case that meets the clinical criteria and is laboratory confirmed, OR  
  ▪ A documented negative HCV antibody followed within 12 months by a positive HCV antibody test (anti-HCV test conversion) in the absence of a more likely diagnosis, OR  
  ▪ A documented negative HCV antibody OR negative hepatitis C virus detection test (in someone without a prior diagnosis of HCV infection) followed within 12 months by a positive hepatitis C virus detection test (HCV RNA test conversion) in the absence of a more likely diagnosis. **Probable:**  
  ▪ A case that meets clinical criteria and has presumptive laboratory evidence (a positive anti-HCV antibody test), AND  
  ▪ Does not have a hepatitis C virus test reported, AND  
  ▪ Has no documentation of anti-HCV or HCV RNA test conversion within 12 months | **Hepatitis C virus detection test:**  
  ▪ Nucleic acid test (NAT) or PCR test for HCV RNA positive (including qualitative, quantitative or genotype testing) OR  
  ▪ A positive test indicating presence of hepatitis C viral antigen (HCV antigen)*  
  *When and if a test for HCV antigen(s) is approved by FDA and available |
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| Hepatitis E, acute 10103 | Typical clinical signs and symptoms of acute hepatitis E virus (HEV) are similar to those of other types of acute viral hepatitis and include abdominal pain anorexia, dark urine, fever, hepatomegaly, jaundice, malaise, nausea, and vomiting. Other less common symptoms include arthralgia, diarrhea, pruritus, and urticarial rash. The period of infectivity following acute infection has not been determined, but viral excretion in stools has been demonstrated for up to 14 days after illness onset. In most hepatitis E outbreaks, the highest rates of clinically evident disease have been in young to middle-age adults; lower disease rates in younger age groups can be the result of anicteric and/or subclinical HEV infection. No evidence of chronic infection has been detected in long-term follow-up of patients with hepatitis E. The case fatality rate is low except in pregnant women where it can reach 20% among those infected during the third trimester of pregnancy.  

**Confirmed:** A case that meets the clinical case description and is laboratory confirmed  
**Probable:** A case that meets the clinical case description with supportive laboratory evidence (positive IgM antibody from labs other than CDC), OR negative tests for other acute hepatitis markers and an epidemiological link to other confirmed cases or travel history to an endemic area during exposure period | • IgM anti-HEV from CDC laboratory or PCR positive from reference laboratory  
Note: No FDA approved tests to diagnose HEV infection are available in the United States. |
| Hookworm (Ancylostomiasis) 80760 | A parasitic infection caused by the soil-transmitted helminths *Necator americanus* and *Ancylostoma duodenale* (rarely by other *Ancylostoma* species, e.g. *A.ceylanicum*). Itching and localized rash are often the first signs of infection. Other symptoms may include cough, abdominal discomfort, diarrhea, blood in the stool, loss of appetite, nausea, fatigue, or pale skin. Light hookworm infections generally produce few or no clinical effects. In heavy infections, symptoms may include abdominal pain, nausea and anorexia. Chronic blood loss at the site of the intestinal attachment of adult worms can lead to anemia. Children with heavy long-term infection may have impaired growth and delayed mental development.  

**Confirmed:** A case that is laboratory confirmed | • Microscopic identification of *Ancylostoma* or *Necator* (Hookworm) eggs in feces,  
OR • Microscopic identification of *Ancylostoma* or *Necator* species larvae cultured from feces,  
OR • Identification of *Ancylostoma* or *Necator* species adult worms expelled after treatment or removed during endoscopy |
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| Influenza, human isolates - [outbreaks only] 11060 | The flu is a contagious respiratory illness caused by influenza viruses. It can cause mild to severe illness and at times can lead to death. Symptoms of flu may include fever, headache, extreme tiredness, dry cough, sore throat, runny or stuffy nose, and muscle aches. Stomach symptoms (nausea, vomiting, and diarrhea) can occur but are more common in children than adults. Complications of flu can include bacterial pneumonia, ear infections, sinus infections, dehydration, and worsening of chronic medical conditions, such as congestive heart failure, asthma, or diabetes.  
**Confirmed:** Case that is clinically compatible and laboratory confirmed  
Outbreak: See the Texas Influenza Surveillance Handbook for more information on influenza (flu)-associated outbreaks including operational influenza-like illness (ILI) and flu-associated outbreak definitions.  
Note: Influenza is not a reportable condition in Texas. See Influenza A, novel/variant infection for reporting of novel/variant strains. See Influenza-associated pediatric mortality for reporting of influenza-associated deaths in all persons aged <18 years. | Influenza virus isolation in tissue cell culture from respiratory specimens,  
OR  
Reverse-transcriptase polymerase chain reaction (RT-PCR) testing of respiratory specimens,  
OR  
Immunofluorescent antibody staining (direct or indirect) of respiratory specimens,  
OR  
Rapid influenza diagnostic testing of respiratory specimens,  
OR  
Immunohistochemical (IHC) staining for influenza viral antigens in respiratory tract tissue from autopsy specimens,  
OR  
Four-fold rise in influenza hemagglutination inhibition (HI) antibody titer in paired acute and convalescent sera |
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<td>Influenza A, novel/variant 11062</td>
<td>An illness compatible with influenza virus infection (fever &gt;100 degrees Fahrenheit, with cough and/or sore throat)</td>
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<td><strong>Confirmed:</strong> A case of human infection with a laboratory confirmed novel/variant influenza A virus</td>
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<td><strong>Probable:</strong> A case meeting the clinical criteria and epidemiologically linked* to a confirmed case, but for which no confirmatory laboratory testing for novel/variant influenza virus infection has been performed or test results are inconclusive for a novel/variant influenza A virus infection</td>
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<td>Epidemiologic linkage criteria: a) the patient has had contact with one or more persons who either have or had the disease and b) transmission of the agent by the usual modes of transmission is plausible. A case can be considered epidemiologically linked to a laboratory-confirmed case if at least one case in the chain of transmission is laboratory confirmed.</td>
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<td><strong>Suspect:</strong> A case meeting the clinical criteria in which influenza A has been detected but is pending laboratory confirmation. Any case of human infection with an influenza A virus that is different from currently circulating human influenza H1 and H3 viruses is classified as a suspect case until the confirmation process is complete.</td>
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<td>Note: Typically, sporadic novel/variant influenza cases will have a history of either close contact with ill animals known to transmit novel subtypes of influenza A (such as wild birds or poultry, swine, or other mammals) OR travel, within 14 days, to any country where a novel influenza A virus (such as highly pathogenic avian influenza A H5N1) has been recently identified in animals or people.</td>
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<td>Identification of an influenza A virus subtype or strain that is different from currently circulating human influenza H1 and H3 strains as confirmed by CDC’s influenza laboratory, by public health laboratories using CDC-approved protocols for that specific strain, or by labs using FDA-authorized tests for specific strains.</td>
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<td>- Novel/variant subtypes include, but are not limited to, H2, H5, H7, and H9 subtypes.</td>
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<td>- Influenza H1 and H3 subtypes originating from a non-human species or from genetic reassortment between animal and human viruses are also novel/variant subtypes or strains.</td>
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<td>- Methods available for detection of currently circulating human influenza viruses at public health laboratories (e.g., rRT-PCR) will also detect suspected novel/variant subtypes and strains.</td>
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<td>- Initial confirmation that a specific influenza A virus represents a novel/variant virus will be performed by CDC’s influenza laboratory.</td>
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<td>- Currently, only viral isolation, RT-PCR, gene sequencing, or a 4-fold rise in strain-specific serum antibody titers are considered confirmatory for case classification purposes.</td>
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<td>Influenza-associated pediatric mortality 11061</td>
<td>An influenza-associated death is defined for surveillance purposes as a death resulting from a clinically compatible illness that was confirmed to be influenza by an appropriate laboratory or rapid diagnostic test. There should be no period of complete recovery between the illness and death. Influenza-associated deaths in all persons aged &lt;18 years should be reported. A death should not be reported if there is no laboratory confirmation of influenza virus infection, the influenza illness is followed by full recovery to baseline health status prior to death, the death occurs in a person 18 years of age or older, or after review and consultation there is an alternative agreed upon cause of death which is unrelated to an infectious process (For example, a child with a positive influenza test whose death clearly resulted from trauma after a car accident would not qualify as a case. However, a child with a respiratory illness and a positive influenza test whose death is attributed to another infectious cause such as staphylococcal pneumonia would still qualify as a case.). <strong>Confirmed:</strong> A death meeting the clinical case definition that is laboratory confirmed</td>
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| **Legionellosis 10490** | Legionellosis is associated with three clinically and epidemiologically distinct illnesses: Legionnaires’ disease, which is characterized by fever, myalgia, cough, and clinical or radiological pneumonia; Pontiac fever, a milder illness without pneumonia; and extrapulmonary legionellosis, a rare manifestation in which *Legionella* can cause disease at sites outside the lungs (e.g., endocarditis, wound infection, joint infection, graft infection). **Confirmed:** A clinically compatible case that meets at least one of the confirmatory laboratory criteria **Probable:** A clinically compatible case with an epidemiologic linkage* during the incubation period  
*Epidemiologic linkage criteria:  
1) Linkage to a setting with a confirmed source of *Legionella*  
OR  
2) Linkage to a setting with a suspected source of *Legionella* that is associated with at least one confirmed case | • Isolation (culture) of *any* *Legionella* organism from respiratory secretions, lung tissue, pleural fluid, or other normally sterile fluid, **OR**  
• Detection of *any* *Legionella* species from lower respiratory secretions, lung tissue, or pleural fluid by a validated nucleic acid amplification test (e.g. PCR), **OR**  
• Detection of *Legionella pneumophila* serogroup 1 antigen in urine using validated reagents, **OR**  
• Demonstration of seroconversion by a fourfold or greater rise in specific serum antibody titer between paired acute and convalescent phase serum specimens to *Legionella pneumophila* serogroup 1 using validated reagents |
| **Leishmaniasis 80550** | Leishmaniasis is a parasitic disease that is present primarily in South and Central America, Africa, Asia, and southern Europe. The *Leishmania* parasite is transmitted via the bite of phlebotomine sand flies. There are several forms of the disease in humans: cutaneous, the most common, which causes skin lesions; visceral, which may affect multiple internal organs, including the liver, spleen, and bone marrow; and mucosal, a less common form that affects mucous membranes of the nose, mouth, or throat.  
Most leishmaniasis cases reported in Texas are the cutaneous form and are travel-associated, albeit autochthonous cases occur occasionally. Cutaneous leishmaniasis infection can present as one or more skin sores weeks or months after a sand fly bite. Over time, the sores may change in size and appearance—they may start out as papules or nodules and may end up as ulcers which might scab over. Lesions can heal spontaneously within weeks to months, or last for a year or more. Some *Leishmania* strains can disseminate to cause mucosal lesions (espundia) years after the primary cutaneous lesion has healed. Without treatment, this sequela can progress and lead to destruction of the naso-oropharyngeal mucosa, which can be severely disfiguring. Visceral leishmaniasis infection can be asymptomatic or result in manifestations such as fever, weight loss, hepatosplenomegaly, and pancytopenia. Severe cases of visceral leishmaniasis are often fatal without treatment. **Confirmed:** A clinically compatible case that is laboratory confirmed | • Microscopic identification of the nonmotile, intracellular form (amastigote) in stained specimens from lesions, **OR**  
• Culture of the motile, extracellular form (promastigote) on suitable media, **OR**  
• An intradermal (Montenegro) test with leishmanin, an antigen derived from the promastigotes, is usually positive in established disease, **OR**  
• Positive *Leishmania* Real-Time PCR or *Leishmania* PCR and DNA sequencing at CDC |
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<td>Listeriosis 10640</td>
<td>In adults, invasive disease caused by <em>Listeria monocytogenes</em> manifests most commonly as meningitis or bacteremia; infection during pregnancy can result in fetal loss through miscarriage or stillbirth, or neonatal meningitis or bacteremia. Other manifestations can also be observed. <strong>Confirmed:</strong> A clinically compatible case that is laboratory confirmed. <strong>Probable:</strong> The mother of a neonate with confirmed or probable listeriosis, even if the laboratory criteria are not met for the mother; a neonate born to a mother with confirmed or probable listeriosis, even if laboratory criteria are not met for the neonate; or a clinically compatible case detected through use of a culture independent laboratory testing method. <strong>Suspect:</strong> Isolation of <em>L. monocytogenes</em> from a non-invasive clinical specimen, e.g., stool, urine, wound. Notes: ▪ Pregnancy loss and intrauterine fetal demise are considered maternal outcomes and would be counted as a single case in the mother. ▪ Cases in neonates and mothers should be reported separately when each meets the case definition. A case in a neonate is counted if live-born. A case should not be counted as a new case if laboratory results were reported within 365 days of a previously reported infection in the same individual, unless additional information is available indicating a separate infection.</td>
<td>• Isolation of <em>L. monocytogenes</em> from a normally sterile site, e.g., blood, cerebrospinal fluid (CSF), or less commonly, joint, pleural, or pericardial fluid, <strong>OR</strong> • Isolation of <em>L. monocytogenes</em> from products of conception at time of delivery and non-sterile sites of neonates obtained within 48 hours of delivery, <strong>OR</strong> • In the setting of miscarriage or stillbirth, isolation of <em>L. monocytogenes</em> from placental or fetal tissue, <strong>OR</strong> • In the setting of pregnancy or live birth, isolation of <em>L. monocytogenes</em> from mother’s or neonate’s blood or other sterile site, or from placental or amniotic fluid. <strong>See Normally Sterile Site</strong></td>
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| Lyme disease 11080 | A systemic, tickborne disease with protean manifestations, including dermatologic, rheumatologic, neurologic, and cardiac abnormalities. The most common clinical marker for the disease is erythema migrans (EM), the initial skin lesion that occurs in 60-80% of patients. For purposes of surveillance, EM is defined as a skin lesion that typically begins as a red macule or papule and expands over a period of days to weeks to form a large round lesion, often with partial central clearing. Secondary lesions also may occur. Annular erythematous lesions occurring within several hours of a tick bite represent hypersensitivity reactions and do not qualify as EM. For most patients, the expanding EM lesion is accompanied by other acute symptoms, particularly fatigue, fever, headache, mildly stiff neck, arthralgia, or myalgia. These symptoms are typically intermittent. Texas is a low-incidence jurisdiction for Lyme disease (has had <10 confirmed cases/100,000 population for a period of three consecutive years) and thus follows the recommended reporting criteria for low-incidence jurisdictions. Clinical criteria: An illness characterized by one of the following early or late-stage manifestations, as reported by a healthcare provider, and in the absence of another known etiology:  
  - Erythema migrans (EM) rash ≥ 5 cm in diameter  
  - Recurrent, brief attacks (weeks or months) of objective joint swelling in one or a few joints  
  - Any of the following signs that cannot be explained by any other etiology, alone or in combination: lymphocytic meningitis; cranial neuritis, particularly facial palsy (unilateral or bilateral); radiculoneuropathy; or, rarely, encephalomyelitis  
  - Acute onset of high-grade (2nd-degree or 3rd-degree) atrioventricular conduction defects that resolve in days to weeks  
  
  Confirmed: A clinically compatible case that has confirmatory laboratory evidence.  
  
  Probable: A clinically compatible case that has presumptive laboratory evidence:  
  - Positive IgG immunoblot without positive or equivocal first-tier screening assay.  
  
  Suspect: A case of EM rash with no laboratory evidence of infection OR a case that meets confirmatory or presumptive laboratory criteria, but no clinical information is available  
  
  Notes:  
  - A new case is one that has not been reported within the same calendar year  
  - While a single IgG immunoblot is adequate for surveillance purposes, a two-tier test is still recommended for patient diagnosis; a positive IgG immunoblot preceded by a negative screen is considered a false positive  
  - There is no validated Lyme disease test for CSF; positive tests on CSF are not confirmatory  |  
  - Isolation of *B. burgdorferi* sensu stricto or *B. mayonii* in culture, OR  
  - Detection of *B. burgdorferi* sensu stricto or *B. mayonii* in a clinical specimen by a *B. burgdorferi* group-specific NAAT assay, OR  
  - Detection of *B. burgdorferi* group-specific antigens by immunohistochemical assay (IHC) on biopsy or autopsy tissues, OR  
  - Standard two-tier test (STTT): positive or equivocal EIA or IFA test, followed by a positive IgM or IgG immunoblot OR  
  - Modified two-tier test (MTTT): positive or equivocal EIA or IFA test, followed by a different, sequential positive or equivocal EIA  
  1IgM WB is considered positive when at least two of the following three bands are present: 24 kilodalton (kDa) outer surface protein C (OspC)*, 39 kDa basic membrane protein A (BmpA), and 41 kDa (Fla). Disregard IgM results for specimens collected >30 days after symptom onset.  
  2IgG WB is considered positive when at least five of the following 10 bands are present: 18 kDa, 24 kDa (OspC)*, 28 kDa, 30 kDa, 39 kDa (BmpA), 41 kDa flagellin (Fla), 45 kDa, 58 kDa (not GroEL), 66 kDa, and 93 kDa.  
  *Depending upon the assay, OspC could be indicated by a band of 21, 22, 23, 24 or 25 kDa.
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| **Malaria 10130** | Initial symptoms of malaria are non-specific and include fever, chills, sweats, headaches, muscle pains, nausea and vomiting. In severe cases of malaria (usually caused by *Plasmodium falciparum*), clinical findings can also include confusion, coma, neurologic focal signs, severe anemia, and respiratory difficulties.  
*Confirmed:* A case that is laboratory confirmed in any person (symptomatic or asymptomatic) diagnosed in the United States, regardless of whether the person experienced previous episodes of malaria while outside the country  
*Suspect:* Detection of *Plasmodium* species by rapid diagnostic antigen testing (RDT) without confirmation by microscopy or nucleic acid testing in any person (symptomatic or asymptomatic) diagnosed in the United States, regardless of whether the person experienced previous episodes of malaria while outside the country  
• Note: A subsequent episode of malaria is counted as an additional case, regardless of the detected Plasmodium species, unless the case is indicated as a treatment failure within 4 weeks of initial presentation. | • Detection and specific identification of malaria parasite species by microscopy on blood films in a laboratory with appropriate expertise  
OR  
• Detection of *Plasmodium* species by nucleic acid test*  
OR  
• Detection of unspeciated malaria parasite by microscopy on blood films in a laboratory with appropriate expertise  
*Laboratory-developed malaria PCR tests must fulfill CLIA requirements, including validation studies. |
| **Measles (Rubeola) 10140** | An illness characterized by all of the following: a generalized maculopapular rash lasting at least 3 days; a temperature ≥ 101.0°F (>38.3°C); and cough, coryza, or conjunctivitis.  
*Confirmed:* An acute, febrile rash illness (temperature can be lower than 101°F and rash < 3 days) that is:  
• Laboratory confirmed  
OR  
• Epidemiologically linked to a laboratory confirmed measles case | • IgG seroconversion or a significant rise in measles immunoglobulin G antibody level by any standard serologic assay*,  
OR  
• Isolation of measles virus from a clinical specimen*,  
OR  
• Detection of measles-virus-specific nucleic acid by PCR*,  
OR  
• A positive serological test for measles immunoglobulin M antibody* not otherwise ruled out by other confirmatory testing or more specific measles testing in a public health laboratory  
*Not explained by MMR vaccination during the previous 6-45 days |
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| **Meningococcal infection, invasive (Neisseria meningitidis)** 10150 | Invasive meningococcal disease manifests most commonly as meningitis and/or meningococcemia that can progress rapidly to purpura fulminans, shock, and death. However, other manifestations (e.g., pneumonia, myocarditis, endocarditis or pericarditis, arthritis, cervicitis) might be observed.  
**Confirmed:** A case that is laboratory confirmed  
**Probable:** A case that has one of the following:  
- *N. meningitidis* antigen detection by immunohistochemistry (IHC) on formalin-fixed tissue  
- *N. meningitidis* antigen detection by latex agglutination of CSF  
**Suspect:** A case that has one of the following:  
- Clinical purpura fulminans in the absence of a positive blood culture  
- Gram-negative diplococci, not yet identified, isolated from a normally sterile site (e.g., blood or CSF) | - Isolation of *Neisseria meningitidis* from a normally sterile site,  
| | | OR  
| | | Isolation of *N. meningitidis* from purpuric lesions,  
| | | OR  
| | | Detection of *N. meningitidis*-specific nucleic acid in a specimen obtained from a normally sterile site, using a validated polymerase chain reason (PCR) assay  
| | | See [Normally Sterile Site](#)  
| | | Note: As required by TAC all *Neisseria meningitidis* isolates from normally sterile sites and/or purpuric lesions must be submitted to the DSHS Laboratory for typing and molecular analysis. |
| **Mumps** 10180 | Acute parotitis or other salivary gland swelling lasting at least 2 days, or orchitis or oophoritis unexplained by another more likely diagnosis  
**Confirmed:** A case that has a positive mumps PCR result, OR positive mumps culture, AND either meets the clinical case definition, OR has aseptic meningitis, encephalitis, hearing loss, mastitis, or pancreatitis  
**Probable:** A case that meets the clinical case definition, AND  
- Has a positive test for serum anti-mumps immunoglobulin M (IgM) antibody, OR  
- Has an epidemiologic link to another probable or confirmed case or linkage to a group/community defined by public health during an outbreak of mumps  
**Suspect:** A case that has parotitis, acute salivary gland swelling, orchitis, or oophoritis unexplained by another more likely diagnosis, OR a has a positive lab result with no mumps clinical symptoms (with or without an epidemiologic link to a confirmed or probable case). | - Isolation of mumps virus from a clinical specimen,  
| | | OR  
| | | Detection of mumps-virus-specific nucleic acid by PCR  
<p>| | | Note: An elevated serum amylase is not confirmatory for mumps. |</p>
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</table>
| Norovirus - [outbreaks only] 10996 | Acute onset vomiting, watery, non-bloody diarrhea, abdominal cramps, and nausea. Low-grade fever may also occasionally occur, and vomiting is more common in children. **Confirmed:** A clinically compatible case that is laboratory confirmed  
  - **Probable:** A clinically compatible case that is epidemiologically linked to a confirmed case | - Detection of norovirus DNA (PCR) in stool or vomitus  
  - Identification of norovirus can best be made from stool specimens taken within 48 to 72 hours after onset of symptoms. Virus can sometimes be found in stool samples taken as late as 2 weeks after recovery.)  
  OR  
  - Detection of norovirus antigen in stool,  
  Note: The etiology of gastrointestinal outbreaks should be confirmed by submitting specimens to the DSHS Laboratory. Sequencing for norovirus strains is available. |
**Condition/Code** | **Case Definition/Case Classification** | **Laboratory Evidence**
---|---|---
**Novel Coronavirus 2019 11065** | A novel coronavirus is a newly identified coronavirus that has not been previously identified in the human population and it is assumed there is no existing immunity to the virus. The virus (SARS-CoV-2) causing coronavirus disease 2019 (COVID-19), first identified in Wuhan, China in 2019 is not the same as coronaviruses that commonly circulate among humans and cause mild illness, like the common cold. The virus is distinct from although closely related to both SARS-CoV and MERS-CoV. Epidemiologic findings indicate COVID-19 may be less severe than SARS or MERS, but evidence suggests that the virus is more contagious than its predecessors†. SARS-CoV-2 is a newly identified pathogen and it is assumed there was no pre-existing human immunity to the virus. There are risk factors that increase an individual’s illness severity. Those at highest risk for severe disease and death include people aged over 60 years (especially those 85 years and older) and those with underlying conditions, including but not limited to obesity, hypertension, diabetes, cardiovascular disease, chronic respiratory or kidney disease, immunosuppression from solid organ transplant, and sickle cell disease. A complete list can be found at: https://www.cdc.gov/coronavirus/2019-ncov/need-extra-precautions/people-with-medical-conditions.html. Disease in children mostly appears to be relatively mild, and there is evidence that a significant proportion of infections across all age groups are asymptomatic, or presymptomatic at the time of testing. Symptoms of COVID-19 are non-specific and the disease presentation can range from no symptoms (asymptomatic) to severe pneumonia and death. People with COVID-19 generally develop signs and symptoms, including mild respiratory symptoms and fever ~5 days after infection (mean incubation period 5-6 days, range 1-14 days). In accordance with The Council of State and Territorial Epidemiologists (CSTE) Update to the standardized surveillance case definition and national notification for 2019 novel coronavirus disease (COVID-19) Interim-20-ID-02, DSHS has adopted the following case classification strategy effective November 1, 2021;**

*Laboratory evidence using a method approved or authorized by the FDA¹ or designated authority²:

**Confirmatory³ laboratory evidence:**
- Detection of SARS-CoV-2 RNA in a post-mortem respiratory swab or clinical specimen using a diagnostic molecular amplification test performed by a CLIA-certified provider,
  
  OR
- Detection of SARS-CoV-2 by genomic sequencing⁴.

**Presumptive³ laboratory evidence:**
- Detection of SARS-CoV-2 specific antigen in a post-mortem obtained respiratory swab or clinical specimen using a diagnostic test performed by a CLIA-certified provider.

**Supportive³ laboratory evidence:**
- Detection of antibody in serum, plasma, or whole blood specific to natural infection with SARS-CoV-2 (antibody to nucleocapsid protein),

OR

**Confirmed:** A case that meets confirmatory laboratory evidence*

**Probable:** A case that:

- Meets clinical criteria AND epidemiologic linkage criteria with no confirmatory laboratory testing performed for SARS-CoV-2,

  \[\text{OR}\]

- Meets presumptive laboratory evidence*

  \[\text{OR}\]

- Meets vital records criteria (death certificate lists COVID-19 disease or SARS-CoV-2 as an underlying cause of death or a significant condition contributing to death) with no confirmatory laboratory testing performed for SARS-CoV-2.

**Suspect:** A case that:

- Meets supportive laboratory evidence* with no prior history of being a confirmed or probable case.

**Laboratory Criteria for Reporting**

- Detection of SARS-CoV-2 RNA in a post-mortem obtained respiratory swab or clinical specimen using a diagnostic molecular amplification test performed by a CLIA-certified provider,

  \[\text{OR}\]

- Detection of SARS-CoV-2 genomic sequence,

  \[\text{OR}\]

- Detection of SARS-CoV-2 specific antigen in a post-mortem obtained respiratory swab or clinical specimen using a diagnostic test performed by a CLIA-certified provider

  \[\text{OR}\]

- Detection of SARS-CoV-2 nucleocapsid and spike protein receptor binding domain (RBD) specific antibodies in serum, plasma, or whole blood by a CLIA-certified provider.

**NOTE:** Testing performed by individuals at home using over-the-counter test kits is considered supportive laboratory evidence due to lack of CLIA oversight.
Clinical Criteria for Reporting:

In the absence of a more likely diagnosis, any medically-attended (including symptoms ascertained telephonically by public health staff, e.g., contact tracers) person with:

- Acute onset or worsening of at least two of the following symptoms or signs: fever (measured or subjective), chills, rigors, myalgia, headache, sore throat, nausea or vomiting, diarrhea, fatigue, congestion or runny nose;

OR

- Acute onset or worsening of any one of the following symptoms or signs: cough, shortness of breath, difficulty breathing, olfactory disorder, taste disorder, confusion or change in mental status, persistent pain or pressure in the chest, pale, gray, or blue-colored skin, lips, or nail beds, depending on skin tone, inability to wake or stay awake;

OR

- Severe respiratory illness with at least one of the following: Clinical or radiographic evidence of pneumonia, Acute respiratory distress syndrome (ARDS).

Epidemiologic Linkage Criteria for Reporting:

A person meeting the clinical reporting criteria with one or more of the following exposures in the 14 days before onset of symptoms:

- Close contact** with a confirmed or probable case of COVID-19 disease;

OR

- Member of an exposed risk cohort as defined by public health authorities during an outbreak or during high community transmission.

**Close contact is generally defined as being within 6 feet for at least 15 minutes (cumulative over a 24-hour period). However, it depends on the exposure level and setting: for example, in the setting of an aerosol generating procedure in healthcare settings without proper personal protective equipment (PPE), this may be defined as any duration.
### Vital Records Criteria for Reporting:

A person whose death certificate lists COVID-19 disease or SARS-CoV-2 or an equivalent term as an underlying cause of death or a significant condition contributing to death.

### Other Criteria for Reporting:

Autopsy findings consistent with pneumonia or acute respiratory distress syndrome without an identifiable cause.

### Criteria to distinguish a new case of this disease or condition from reports or notifications which should not be enumerated as a new case for surveillance:

The following should be enumerated as a new case:

- SARS-CoV-2 sequencing results from the new positive specimen and a positive specimen from the most recent previous case demonstrate a different lineage,

  **OR**

- Person was most recently enumerated as a confirmed or probable case with onset date (if available) or first positive specimen collection date for that classification >90 days prior‡,

  **OR**

- Person was previously reported but not enumerated as a confirmed or probable case (i.e., suspect)‡‡, but now meets the criteria for a confirmed or probable case.

‡Some individuals, e.g., severely immunocompromised persons, can shed SARS-CoV-2 detected by molecular amplification tests >90 days after infection. For severely immunocompromised individuals, clinical judgment should be used to determine if a repeat positive test is likely to result from long term shedding and therefore not be enumerated as a new case. CDC defines severe immunocompromise as certain conditions, such as being on chemotherapy for cancer, untreated HIV infection with CD4 T lymphocyte count 20mg/day for more than 14 days.

‡‡Repeat suspect cases should not be enumerated.
<table>
<thead>
<tr>
<th>Outbreaks, exotic diseases, and unusual expression of disease</th>
<th>In addition to specified reportable conditions, any outbreak, exotic disease, or unusual group expression of disease that may be of public health concern should be reported by the most expeditious means available.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Influenza, human isolates 11060</td>
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<tr>
<td>Norovirus 10996</td>
<td></td>
</tr>
<tr>
<td>Streptococcal toxic-shock syndrome 11700</td>
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</tbody>
</table>
| Paragonimiasis 80664                                        | Paragonimiasis (lung fluke trematode) is transmitted by eating inadequately cooked crustaceans (primarily crayfish in the US) that are infected with the parasite. Disease most frequently involves the lungs. Initial signs and symptoms may be diarrhea and abdominal pain followed several days later by fever, chest pain, and fatigue. The symptoms may also include a dry cough, which later becomes productive with rusty-colored or blood-tinged sputum on exertion, and pleuritic chest pain. X-ray findings may include diffuse and/or segmental infiltrates, nodules, cavities, ring cysts and/or pleural effusions. Extrapulmonary disease is not uncommon, with flukes found in such sites as the CNS, subcutaneous tissues, intestinal wall, peritoneal cavity, liver, lymph nodes and genitourinary tract. Infection usually lasts for years, and the infected person may be asymptomatic. Paragonimiasis may be mistaken for tuberculosis, clinically and on chest X-rays.  

**Confirmed:** A case that is laboratory confirmed  
**Probable:** A clinically compatible case with  
- Detection of *Paragonimus* antibodies by CF, EIA, or immunoblot, **OR**  
- Positive skin test for *Paragonimus*, **OR**  
- History of ingestion of inadequately cooked crustaceans and marked eosinophilia with total WBC count in the normal range or supportive x-ray findings  

- Microscopic identification of *Paragonimus* eggs in feces, sputum, pleural fluid, CSF, or pus, **OR**  
- Identification of worms or eggs in biopsies of pulmonary, cerebral, subcutaneous, or intra-abdominal nodules or cystic lesions |
<table>
<thead>
<tr>
<th>Pertussis</th>
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<td>Pertussis</td>
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<td>Pertussis</td>
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A cough illness lasting at least 14 days AND at least one of the following additional symptoms in the absence of a more likely diagnosis:

- Paroxysmal coughing, **OR**
- Inspiratory "whoop," **OR**
- Post-tussive vomiting, **OR**
- Apnea (with or without cyanosis)

**Confirmed:** A person with an acute cough illness of any duration who is laboratory confirmed 

**Probable:** In the absence of a more likely diagnosis, a person who is not laboratory confirmed (not tested, tests are negative, or tested by serology or DFA), and is either:
- A person with an acute cough illness of any duration, with
  - At least one of the following signs or symptoms:
    - Paroxysms of coughing, **OR**
    - Inspiratory whoop, **OR**
    - Post-tussive vomiting, **OR**
    - Apnea (with or without cyanosis)
  - AND epidemiological linkage to a laboratory confirmed case
- A person who meets the clinical case definition.

- Isolation (culture) of *Bordetella pertussis* from a clinical specimen,
  **OR**
- Positive polymerase chain reaction (PCR) assay for *Bordetella pertussis*

**Note:** Because *B. pertussis* can be difficult to culture, a negative culture result does not rule out pertussis. Negative PCR results do not require investigation unless reported as a suspected case by a healthcare provider. Direct fluorescent antibody (DFA) staining of a patient’s specimen and serological laboratory results (pertussis IgA, IgG or IgM) are **NOT** considered confirmatory for pertussis, but should be investigated as soon as possible.
**Plague**

| 10440 |

Plague, a bacterial infection caused by *Yersinia pestis*, is transmitted to humans via flea bites or by direct exposure to infected tissues or respiratory droplets. The disease is characterized by fever, chills, headache, malaise, prostration, and leukocytosis and can manifest in one or more specific clinical presentations which typically reflect the route of exposure to the pathogen.

*Clinical evidence:* Acute onset of fever as reported by the patient or healthcare provider with or without one or more of the following: regional lymphadenitis, septicemia, pneumonia, or pharyngitis with cervical lymphadenitis.

**Confirmed:** A clinically compatible case with confirmatory laboratory evidence, OR a clinically compatible case with presumptive laboratory evidence AND epidemiologic linkage (see below)

**Probable:** A clinically compatible case with a presumptive laboratory evidence* as listed below that lacks an alternative diagnosis and epidemiologic linkage (see below)

- Elevated serum antibody titer(s) to *Y. pestis* fraction 1 (F1) antigen (without documented four-fold or greater change) in a patient with no history of plague vaccination, OR
- Detection of *Y. pestis* specific DNA or antigens, including F1 antigen, in a clinical specimen by DFA, IHC, or PCR

**Suspect:** A clinically compatible case without laboratory evidence that has an epidemiologic linkage OR an individual with confirmed or presumptive laboratory evidence without any associated clinical information

Epidemiologic linkage is defined as one or more of the following:

- Person that is epidemiologically linked to a person or animals with confirmatory laboratory evidence within the prior two weeks; OR
- Close contact with a confirmed pneumonic plague case, including but not limited to presence within two meters of a person with active cough due to pneumonic plague; OR
- A person that lives in or has traveled within two weeks of illness onset to a geographically-localized area with confirmed plague epizootic activity in fleas or animals as determined by the relevant local authorities.

*Other laboratory tests, including rapid bedside tests, are in use in some low resourced international settings but are not recommended as laboratory evidence of plague infection in the United States.*

<p>| |</p>
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Isolation of *Y. pestis* from a clinical specimen with culture identification validated by a secondary assay (e.g. bacteriophage lysis assay, DFA assay) as performed by a CDC or LRN laboratory, OR
- Four-fold or greater change in paired serum antibody titer to *Y. pestis* F1 antigen

For isolates of other species of *Yersinia*, see **Yersiniosis**

Note: As required by [TAC](#), all *Y. pestis* isolates must be submitted to an LRN laboratory.
<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
<th>Confirmation Criteria</th>
</tr>
</thead>
</table>
| Poliomyelitis, paralytic                       | Acute onset of a flaccid paralysis of one or more limbs with decreased or absent tendon reflexes in the affected limbs, without other apparent cause, and without sensory or cognitive loss | *Confirmed*: A case that meets the clinical case definition in which the patient has a neurological deficit 60 days after onset of initial symptoms, has died, or has unknown follow-up status  
*Probable*: A case that meets the clinical case definition  
*Note: All suspected cases of paralytic poliomyelitis are reviewed by a panel of expert consultants at the Centers for Disease Control and Prevention (CDC) before final case classification occurs. |
| Poliovirus infection, nonparalytic             | Most poliovirus infections are asymptomatic or cause mild febrile disease.                       | *Confirmed*: Laboratory confirmed poliovirus infection in a person without symptoms of paralytic poliomyelitis                                                                                                   |
| Primary amebic meningoencephalitis (PAM)       | See [Amebic meningoencephalitis (PAM)](#)                                                         | • Poliovirus isolate identified in an appropriate clinical specimen, with confirmatory typing and sequencing performed by the CDC Poliovirus Laboratory          |
**Prion diseases such as Creutzfeldt-Jakob disease (CJD)**

Creutzfeldt-Jakob disease (CJD) is a human prion disease described as rapidly progressive, neurodegenerative, and invariably fatal. Human prion diseases include sporadic forms of disease (sporadic CJD (sCJD), sporadic fatal insomnia (sFI), and variably protease-sensitive prionopathy (VPSPr)), familial/genetic forms of disease (familial or genetic CJD (fCJD or gCJD), fatal familial insomnia (FFI), and Gerstmann-Sträussler-Scheinker syndrome (GSS)) and acquired forms of disease (iatrogenic CJD (iCJD), Kuru (described only in the Fore population of Papua New Guinea), and variant CJD (vCJD)).

Classical sporadic CJD presentation consists of rapidly progressive dementia, visual abnormalities, myoclonus, or cerebellar dysfunction (where both balance abnormalities and muscle incoordination are seen which commonly present as gait, speech, and swallowing disorders). Most patients eventually develop pyramidal and extrapyramidal dysfunction, such as abnormal reflexes (hyperreflexia), spasticity, tremors, and rigidity. Akinetic mutism appears late in the disease. Median duration of illness is 4-5 months; the duration of illness rarely exceeds 12 months.

For purposes of surveillance and notification: prion diseases such as CJD also includes SFI, VPSPr, FFI, GSS syndrome, Kuru, and any novel prion disease affecting humans.

**Sporadic CJD (sCJD)**

*Confirmed:* Satisfactory confirmatory test findings on autopsy or biopsy of brain tissue

*Probable:*
- Neuropsychiatric disorder AND positive RT-QuIC in CSF or other tissues OR
- Rapidly progressive dementia AND at least two of the following four clinical features:
  a) Myoclonus
  b) Visual or cerebellar signs
  c) Pyramidal/extrapyramidal signs
  d) Akinetic mutism
- AND satisfying at least 1 of the supportive laboratory criteria,
- AND absence of routine investigations indicating an alternative diagnosis

*Possible:*
- Progressive dementia AND at least two of the following four clinical features:
  a) Myoclonus
  b) Visual or cerebellar signs
  c) Pyramidal/extrapyramidal signs
  d) Akinetic mutism
- AND absence of any supportive laboratory criteria,
- AND duration of illness < 2 years,
- AND absence of routine investigations indicating an alternative diagnosis

*sCJD includes sporadic fatal insomnia (sFI) and variably protease-sensitive prionopathy (VPSPr) which are typically neuropathologic diagnoses

**Confirmatory Laboratory Criteria - sporadic, familial/genetic & iatrogenic CJD**

- Diagnosis by standard neuropathological techniques
  AND/OR
- Immunohistochemistry
  AND/OR
- Western blot confirmed protease-resistant PrP
  AND/OR
- Presence of scrapie-associated fibrils

**Supportive Laboratory Criteria - sporadic, familial/genetic & iatrogenic CJD**

- **CSF 14-3-3 protein:** Reported as elevated, above normal limits, or positive. If 14-3-3 protein is the only supportive test used in determining classification, then duration of illness must be < 2 years.
- **RT-QuIC:** Positive
- **EEG:** Reported as “typical of” or “consistent with” sporadic CJD or the report indicates the presence of generalized bi- or triphasic “periodic sharp wave complexes” (PSWC) at a frequency of 1-2 per second. No limitation on duration of illness.
- **Brain MRI:** High signal abnormalities in the caudate nucleus and/or putamen OR in at least two cortical regions (temporal, parietal, occipital) on diffusion-weighted imaging (DWI) or fluid attenuated inversion recovery (FLAIR). No limitation on duration of illness.

(Continued on next page – see Exclusion Criteria.)
| Prion diseases such as Creutzfeldt-Jakob disease (CJD) (continued on next page) | Familial/Genetic CJD (fCJD)** | Exclusion Criterion: On neurohistopathological analysis of whole brain autopsy tissue, the absence of findings consistent with prion disease (negative results) is sufficient to “rule out” possible and probable cases and reclassify as “Not a Case”. |
| --- | --- | Note: Whole brain autopsy and neuropathology is the only way to confirm or rule-out prion disease. Biopsy tissue can only confirm presence of prion disease but is not sufficient to rule-out prion disease. Autopsy or postmortem biopsy (when autopsy is not possible) is strongly encouraged, while biopsy on living patients should be reserved for diagnosing treatable diseases. The National Prion Disease Pathology Surveillance Center (NPDPSC) performs analysis on CSF, blood, and brain tissue. They provide free transport, shipping, and autopsy services for suspected cases of CJD (the family must initiate contact). Physicians are strongly encouraged to confirm the diagnosis of CJD by discussing and arranging autopsy with the NPDPSC and family members. Autopsy is “highly suggested” for all cases with onset age less than 55 years or physician diagnosed CJD that does not meet the epidemiologic case criteria. |
| Familial/Genetic CJD (fCJD)** | A classification of Confirmed or Probable requires:  
- Confirmed or probable CJD AND confirmed or probable CJD classification in a first degree relative  
- AND/OR  
- Neuropsychiatric disorder AND fCJD-specific PRNP gene mutation | |
| **Familial/Genetic CJD (fCJD)** | **Fatal familial insomnia (FFI) and Gerstmann-Sträussler-Scheinker syndrome (GSS) are specific familial/genetic CJD diseases, and classification will be based on pathology results and/or a specific PRNP gene mutation for the disease and family history.** | |
| Acquired CJD | Iatrogenic CJD (iCJD):  
- Progressive cerebellar syndrome in a recipient of human cadaveric-derived pituitary hormone  
OR  
- Meets sCJD criteria AND a recognized exposure risk (e.g., antecedent neurosurgery with dura mater graft) | |
| Exclusion Criterion: On neurohistopathological analysis of whole brain autopsy tissue, the absence of findings consistent with prion disease (negative results) is sufficient to “rule out” possible and probable cases and reclassify as “Not a Case”. | Note: Whole brain autopsy and neuropathology is the only way to confirm or rule-out prion disease. Biopsy tissue can only confirm presence of prion disease but is not sufficient to rule-out prion disease. Autopsy or postmortem biopsy (when autopsy is not possible) is strongly encouraged, while biopsy on living patients should be reserved for diagnosing treatable diseases. The National Prion Disease Pathology Surveillance Center (NPDPSC) performs analysis on CSF, blood, and brain tissue. They provide free transport, shipping, and autopsy services for suspected cases of CJD (the family must initiate contact). Physicians are strongly encouraged to confirm the diagnosis of CJD by discussing and arranging autopsy with the NPDPSC and family members. Autopsy is “highly suggested” for all cases with onset age less than 55 years or physician diagnosed CJD that does not meet the epidemiologic case criteria. |
Prion diseases such as Creutzfeldt-Jakob disease (CJD)

<table>
<thead>
<tr>
<th>Variants of CJD (vCJD) is characterized by exposure to the causative agent of bovine spongiform encephalopathy (BSE) through consumption of contaminated meat, a prolonged incubation period of ~10 years (possibly decades), and presence of a neuropsychiatric disease that is progressive and invariably fatal. Median age at death is 28 years. Clinical presentation: early psychiatric symptoms (anxiety/depression/withdrawal), or sensory symptoms, and delayed development of neurologic signs (≥ 4 months), and duration of illness lasting over 6 months with a median duration of illness of 13-14 months.</th>
</tr>
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<tbody>
<tr>
<td><strong>Confirmed:</strong> Confirmatory laboratory criteria are met</td>
</tr>
<tr>
<td><strong>Suspect</strong>*: The following criteria are met:</td>
</tr>
<tr>
<td>a) Current age or age at death &lt;55 years (a brain autopsy is recommended, however, for all physician-diagnosed CJD cases)</td>
</tr>
<tr>
<td>b) Psychiatric symptoms at illness onset AND/OR persistent painful sensory symptoms (frank pain and/or dysesthesia)</td>
</tr>
<tr>
<td>c) Dementia AND development ≥4 months after illness onset of at least two of the following five neurologic signs: poor coordination, myoclonus, chorea, hyperreflexia, or visual signs. (If persistent painful sensory symptoms exist, ≥4 months delay in the development of the neurologic signs is not required.)</td>
</tr>
<tr>
<td>d) A normal or an abnormal EEG, BUT NOT the diagnostic EEG changes often seen in classic CJD</td>
</tr>
<tr>
<td>e) Duration of illness of over 6 months</td>
</tr>
<tr>
<td>f) Routine investigations of the patient do not suggest an alternative, non-CJD diagnosis</td>
</tr>
<tr>
<td>g) No history of receipt of cadaveric human pituitary growth hormone or a dura mater graft</td>
</tr>
<tr>
<td>h) No history of CJD in a first degree relative or PRNP gene mutation in the patient OR</td>
</tr>
<tr>
<td>▪ Presence of “bilateral pulvinar high signal” or “pulvinar sign” or “symmetrical, bilateral high signal in the posterior thalamic nuclei” on MRI,</td>
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<tr>
<td>▪ AND</td>
</tr>
<tr>
<td>▪ Presence of all of the following: a progressive neuropsychiatric disorder, d, e, f, &amp; g of the above criteria AND four of the following five criteria:</td>
</tr>
<tr>
<td>▪ Early psychiatric symptoms (anxiety, apathy, delusions, depression, withdrawal)</td>
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<tr>
<td>▪ Persistent painful sensory symptoms (frank pain and/or dysesthesia)</td>
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<tr>
<td>▪ Ataxia</td>
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<tr>
<td>▪ Myoclonus or chorea or dystonia</td>
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<tr>
<td>▪ Dementia</td>
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</tbody>
</table>

***A history of possible exposure to bovine spongiform encephalopathy (BSE) such as residence or travel to a BSE-affected country after 1980 increases the index of suspicion for a variant CJD diagnosis. |

Confirmatory Laboratory Criteria – variant CJD
- Numerous widespread kuru-type amyloid plaques surrounded by vacuoles in both the cerebellum and cerebrum (i.e., florid plaques) AND
- Spongiform change and extensive prion protein deposition shown by immunohistochemistry throughout the cerebellum and cerebrum

Supportive Laboratory Criteria – variant CJD
- EEG with normal or abnormal findings BUT WITHOUT findings consistent with sporadic CJD (absence of “periodic sharp wave complexes” - PSWC), OR EEG not reported or performed
- Presence of “bilateral pulvinar high signal” OR “pulvinar sign” OR “symmetrical, bilateral high signal in the posterior thalamic nuclei” on MRI (relative to other deep gray-matter nuclei)

Note: Whole brain autopsy and neuropathology is the only way to confirm or rule-out prion disease. Biopsy tissue can only confirm presence of prion disease but is not sufficient to rule-out prion disease. Autopsy or postmortem biopsy (when autopsy is not possible) is strongly encouraged, while biopsy on living patients should be reserved for diagnosing treatable diseases. The National Prion Disease Pathology Surveillance Center (NPDPSC) performs analysis on CSF, blood, and brain tissue. They provide free transport, shipping, and autopsy services for suspected cases of CJD (the family must initiate contact). Physicians are strongly encouraged to confirm the diagnosis of CJD by discussing and arranging autopsy with the NPDPSC and family members. Autopsy is “highly suggested” for all cases with onset age less than 55 years or physician diagnosed CJD that does not meet the epidemiologic case criteria.
| Q Fever, acute | Q fever is a zoonotic disease caused by *Coxiella burnetii*. Asymptomatic infection occurs in approximately half of those infected. Exposure to Q fever is usually via aerosol, and the source can be unknown (especially for chronic infection). Exposure can be associated with goats, sheep, or other livestock, but direct contact with animals is not required, and variable incubation periods can be dose dependent. Acute infection, if symptomatic, is characterized by acute onset of fever accompanied by rigors, myalgia, malaise, and severe retrobulbar headache, and can include fatigue, night sweats, dyspnea, confusion, nausea, diarrhea, abdominal pain, vomiting, non-productive cough, or chest pain. Acute hepatitis, atypical pneumonia, and meningoencephalitis may be present with severe disease. Pregnant women are at risk for fetal death and abortion. Clinical laboratory findings can include elevated liver enzyme levels, leukocytosis, and thrombocytopenia.

**Clinical evidence:** Acute fever and one or more of the following: rigors, severe retrobulbar headache, acute hepatitis, pneumonia, or elevated liver enzyme levels.

**Confirmed:** A clinically compatible case that is laboratory confirmed

**Probable:** A clinically compatible case with a single supportive IgG-specific antibody titer to *C. burnetii* Phase II antigen of ≥1:128 by IFA, and the absence of a more likely clinical explanation

| Q Fever, chronic | Chronic Q fever is characterized by a *Coxiella burnetii* infection that persists for more than 6 months. Potentially fatal endocarditis can evolve months to years after acute infection, particularly in persons with underlying valvular disease. Infections of aneurysms and vascular prostheses have been reported. Immunocompromised individuals are particularly susceptible. Rare cases of chronic hepatitis without endocarditis, osteomyelitis, osteoarthritis, and pneumonitis have been described.

**Clinical evidence:** Chronic hepatitis, osteomyelitis, osteoarthritis, or pneumonitis (in the absence of other known etiology); suspected infection of a vascular aneurysm or vascular prosthesis; or newly recognized, culture-negative endocarditis (particularly in a patient with previous valvulopathy or a compromised immune system).

**Confirmed:** A clinically compatible (meets clinical evidence criteria) case of chronic illness that is laboratory confirmed

**Probable:** A clinically compatible case of chronic illness with an antibody titer to *C. burnetii* Phase I IgG antigen that is ≥1:128 and <1:800 by IFA

| • Serological evidence of a four-fold change in IgG-specific antibody titer to *C. burnetii* Phase II antigen by IFA between paired serum samples (preferably one taken during the first week of illness and a second 3-6 weeks later; phase I titer may be elevated as well),
| • Detection of *C. burnetii* DNA in a clinical specimen by PCR,
| • Demonstration of *C. burnetii* antigen in a clinical specimen by IHC,
| • Isolation of *C. burnetii* from a clinical specimen in cell culture

| • Serological evidence of IgG antibody to *C. burnetii* Phase I antigen of ≥1:800 by IFA (phase II will likely be elevated as well but will generally be lower than phase I)
| • Detection of *C. burnetii* DNA in a clinical specimen by PCR,
| • Demonstration of *C. burnetii* antigen in a clinical specimen by IHC,
| • Isolation of *C. burnetii* from a clinical specimen in cell culture

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<table>
<thead>
<tr>
<th>Rabies, animal</th>
<th>All warm-blooded animals, including humans, are susceptible to rabies. In Texas, skunks, bats, coyotes, and foxes are the most commonly infected animals. Domestic dogs, cats, and livestock usually acquire rabies infections from wild animals. Medical authorities distinguish between &quot;furious&quot; and &quot;dumb&quot; rabies on the basis of clinical signs. In the furious variety, the &quot;mad dog&quot; symptoms are pronounced. The animal is irritable and will snap and bite at real or imaginary objects. It can run for miles and attack anything in its path. The animal is extremely vicious and violent. Paralysis sets in shortly, usually affecting the hind legs first. Death follows four to seven days after the onset of clinical signs. In dumb rabies, the prominent symptoms are drowsiness and paralysis of the lower jaw. The animal can appear to have a bone lodged in its throat, sometimes causing owners to force open an animal's mouth to investigate and become unwittingly exposed to rabies. Animals with dumb rabies have no tendency to roam but will snap at movement. They are completely insensitive to pain, and usually become comatose and die from three to ten days after first symptoms appear. <strong>Confirmed:</strong> A case that is laboratory confirmed</th>
</tr>
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<tbody>
<tr>
<td>Rabies, human</td>
<td>Rabies is an acute encephalomyelitis that almost always progresses to coma or death within 10 days after the first symptom. <strong>Confirmed:</strong> A clinically compatible case that is laboratory confirmed by testing at a state or federal public health laboratory Note: Laboratory confirmation by all of the methods listed under “Lab Confirmation Tests” is strongly recommended.</td>
</tr>
<tr>
<td></td>
<td><strong>Confirmed:</strong> A case that is laboratory confirmed by:</td>
</tr>
<tr>
<td></td>
<td>- A positive DFA test (preferably performed on central nervous system tissue), <strong>OR</strong></td>
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<tr>
<td></td>
<td>- Isolation of rabies virus (in cell culture or in a laboratory animal)</td>
</tr>
<tr>
<td></td>
<td><strong>Detected:</strong> A case that is laboratory confirmed by:</td>
</tr>
<tr>
<td></td>
<td>- Detection of Lyssavirus antigens in a clinical specimen (preferably the brain or the nerves surrounding hair follicles in the nape of the neck) by DFA, <strong>OR</strong></td>
</tr>
<tr>
<td></td>
<td>- Isolation (in cell culture or in a laboratory animal) of Lyssavirus from saliva, or central nervous system tissue, <strong>OR</strong></td>
</tr>
<tr>
<td></td>
<td>- Identification of Lyssavirus specific antibody (i.e., by IFA or complete rabies virus neutralization at 1:5 dilution) in the CSF, <strong>OR</strong></td>
</tr>
<tr>
<td></td>
<td>- Identification of Lyssavirus specific antibody (i.e., by IFA or complete rabies virus neutralization at 1:5 dilution) in the serum of an unvaccinated person, <strong>OR</strong></td>
</tr>
<tr>
<td></td>
<td>- Detection of Lyssavirus viral RNA using RT-PCR in saliva, CSF, or tissue</td>
</tr>
</tbody>
</table>
**Tick-borne Relapsing Fever (TBRF)**

Tick-borne Relapsing Fever (TBRF) is an illness caused by infection with some members of the genus *Borrelia*, including *B. hermsii*, *B. parkeri*, and *B. turicatae*. *Borrelia* spirochetes that cause TBRF are transmitted to humans through the bite of infected “soft ticks” of the genus *Ornithodoros*. Each relapsing fever group *Borrelia* species is usually associated with a specific tick species: *B. hermsii* is transmitted by *O. hermsi*; *B. parkeri* by *O. parkeri*; and *B. turicatae* by *O. turicata* ticks. Disease incubation averages one week following a tick bite. Illness is characterized by periods of fever, often exceeding 103°F, lasting 2-7 days, alternating with afebrile periods of 4-14 days. Febrile periods are often accompanied by shaking chills, sweats, headache, muscle and joint pain, and nausea/vomiting. TBRF may be fatal in 5-10% of untreated cases. TBRF contracted during pregnancy can cause spontaneous abortion, premature birth, and neonatal death.

**Clinical evidence:** Measured fever ≥38.8°C (102°F) alone OR one or more episodes of subjective or measured fever <101°F AND two or more of the following: headache, myalgia, nausea/vomiting, or arthralgia.

**Epidemiologic linkage criteria:** Onset of clinically compatible illness 2-18 days after sharing the same exposure site and time as a confirmed case.

**Exposure criteria:** Exposure is defined as time spent in a county in which *Ornithodoros* soft ticks are present or where a confirmed autochthonous case of TBRF has been previously reported. Time spent in cabins, caves, around firewood, or other possible soft tick habitat within 2-18 days of symptom onset is considered highest risk.

**Confirmed:** A clinically compatible illness that is laboratory confirmed, OR a clinically compatible illness with presumptive laboratory evidence* that meets the exposure and/or epidemiologic linkage criteria.

**Probable:** A clinically compatible illness with presumptive laboratory evidence*, defined as:

- Identification of *Borrelia* spirochetes in peripheral blood, bone marrow, or cerebral spinal fluid (CSF), OR
- Serologic evidence of *Borrelia hermsii*, *B. parkeri*, or *B. turicatae* infection by equivocal or positive EIA and positive Western blot, OR
- Relapsing fever *Borrelia* detection through nucleic acid testing, such as PCR, which does not differentiate soft-tick relapsing fever *Borrelia* spp. from other relapsing fever *Borrelia* spp.

Note: Antibodies stimulated by other spirochetal infections (e.g. Lyme disease and syphilis) may cross react on TBRF serologic assays. Epidemiological information including exposure history is crucial to differentiate positive serology results.
<table>
<thead>
<tr>
<th>Disease</th>
<th>Description</th>
<th>Probable Case Definition</th>
<th>Notes</th>
</tr>
</thead>
</table>
| Rickettsiosis, unspecified | Flea-borne typhus and spotted fever rickettsioses (SFR) are vector-borne infections caused by some members of the genus *Rickettsia*. These infections can be difficult to differentiate clinically and serologically due to antibody cross-reactivity.  
*Clinical evidence:* Acute illness lasting less than 30 days with fever and two or more of the following: rash, headache, nausea/vomiting, myalgia, anemia, thrombocytopenia, or elevated liver enzymes. | A case that meets clinical criteria with similar elevations* in IgG serologic titers (≥1:128 to spotted fever and/or typhus group antigens) in a sample taken within 60 days of illness onset that cannot be definitively classified as spotted fever rickettsiosis or flea-borne typhus and does not have a more likely clinical explanation.  
*Serologic IgG titers that are equal or within one dilution of each other* | Not applicable – see note |
| Rubella | An illness that has all the following characteristics: Acute onset of generalized maculopapular rash; temperature ≥99°F (37.2°C), if measured; and arthralgia/arthritis, lymphadenopathy, or conjunctivitis.  
*Confirmed:* A case that is clinically compatible and is laboratory confirmed or epidemiologically linked to a laboratory-confirmed case. | A case that is clinically compatible and is laboratory confirmed or epidemiologically linked to a laboratory-confirmed case.  
Note: Serum rubella IgM test results that are false positives have been reported in persons with other viral infections (e.g., acute infection with Epstein-Barr virus [infectious mononucleosis], recent cytomegalovirus infection, and parvovirus infection) or in the presence of rheumatoid factor. Patients who have laboratory evidence of recent measles infection are excluded. | Isolation of rubella virus,  
**Significant rise between acute- and convalescent-phase titers in serum rubella immunoglobulin G (IgG) antibody level* by any standard serologic assay,**  
**Positive serologic test for rubella-specific immunoglobulin M (IgM) antibody* not otherwise ruled out by more specific testing in a public health laboratory,**  
**Detection of rubella-virus-specific nucleic acid by PCR**  
*Not explained by MMR vaccination during the previous 6-45 days.* |
| **Rubella, congenital syndrome** 10370 | An illness of newborns resulting from rubella infection *in utero* and characterized by signs or symptoms from the following categories:  
  a) Cataracts/congenital glaucoma, congenital heart disease (most commonly patent ductus arteriosus or peripheral pulmonary artery stenosis), hearing loss, or pigmentary retinopathy  
  b) Purpura, hepatosplenomegaly, jaundice, microcephaly, developmental delay, meningoencephalitis, or radiolucent bone disease  
  **Confirmed:** A clinically consistent case that is laboratory confirmed  
  **Probable:** A case that is not laboratory confirmed, that has any two complications listed in (a) of the clinical case definition or one complication from (a) and one from (b), and lacks evidence of any other etiology  
  • Isolation of rubella virus,  
  OR  
  • Demonstration of rubella-specific immunoglobulin M (IgM) antibody,  
  OR  
  • Infant rubella antibody level that persists at a higher level and for a longer period than expected from passive transfer of maternal antibody (i.e., rubella titer that does not drop at the expected rate of a twofold dilution per month),  
  OR  
  • Detection of rubella-virus-specific nucleic acid by PCR |
| **Salmonella Paratyphi** 50266 | An illness caused by *Salmonella* Paratyphi serotypes A, B (tartrate negative), and C that is often characterized by insidious onset of sustained fever, headache, malaise, anorexia, relative bradycardia, constipation or diarrhea, and non-productive cough. However, mild and atypical infections may occur. Carriage of *S. Paratyphi* A, B (tartrate negative), and C may be prolonged.  
  **Confirmed:** A case that is laboratory confirmed  
  **Probable:**  
  • A clinically compatible case with *S. Paratyphi* A, B (tartrate negative), or C detected by use of culture independent laboratory methods (non-culture based),  
  OR  
  • A clinically compatible case that is epidemiologically linked to a case that meets the probable or confirmed laboratory criteria for diagnosis  
  **Notes:**  
  • Both asymptomatic infections and infections at sites other than the gastrointestinal tract, if laboratory confirmed, are considered confirmed cases that should be reported.  
  • Carriage of *S. Paratyphi* A, B (tartrate negative), and C can be prolonged. A case should not be counted as a new case if laboratory results were reported within 365 days of a previously reported infection in the same individual, unless additional information is available indicating a separate infection, e.g., different serotype.  
  • Isolation of *S. Paratyphi* A, B (tartrate negative), or C from a clinical specimen  
  Note: As required by *TAC* all *Salmonella* spp. isolates must be submitted to the DSHS Laboratory. |
| **Salmonella Typhi** 50267 | An illness caused by *Salmonella Typhi* that is often characterized by insidious onset of sustained fever, headache, malaise, anorexia, relative bradycardia, constipation or diarrhea, and non-productive cough. However, mild and atypical infections may occur. Carriage of *S. Typhi* may be prolonged.  
**Confirmed:** A case that is laboratory confirmed  
**Probable:**  
- A clinically compatible case with *S. Typhi* detected by use of culture independent laboratory methods (non-culture based),  
  **OR**  
- A clinically compatible case that is epidemiologically linked to a case that meets the probable or confirmed laboratory criteria for diagnosis  
**Notes:**  
- Both asymptomatic infections and infections at sites other than the gastrointestinal tract, if laboratory confirmed, are considered confirmed cases that should be reported.  
- Carriage of *S. Typhi* can be prolonged. A case should not be counted as a new case if laboratory results were reported within 365 days of a previously reported infection in the same individual, unless additional information is available indicating a separate infection, e.g., different serotype.  
  • Isolation of *S. Typhi* from blood, stool, or other clinical specimen  
Note: As required by TAC all *Salmonella* spp. isolates must be submitted to the DSHS Laboratory. |
| **Salmonellosis, non-Paratyphi/non-Typhi 50265** | An illness of variable severity commonly manifested by diarrhea, fever, abdominal pain, nausea, and sometimes vomiting. Asymptomatic infections can occur, and the organism can cause extraintestinal infections.  
**Confirmed:** A case that is laboratory confirmed. When available, *Salmonella* serotype characterization should be reported  
**Probable:**  
- A case with *Salmonella sp.* (excluding *S. Typhi* and *S. Paratyphi* [A, B (tartrate negative), and C]) detected by use of culture independent laboratory methods (non-culture based),  
  **OR**  
- A clinically compatible case that is epidemiologically linked to a case that meets the probable or confirmed laboratory criteria for diagnosis  
**Notes:**  
- A case with isolation of *S. Paratyphi* B (tartrate positive) from a clinical specimen should be reported as a salmonellosis, non-Paratyphi/non-Typhi case.  
- Both asymptomatic infections and infections at sites other than the gastrointestinal tract, if laboratory confirmed, are considered confirmed cases that should be reported.  
- A case should not be counted as a new case if laboratory results were reported within 365 days of a previously reported infection in the same individual, unless additional information is available indicating a separate infection, e.g., different serotype.  
  • Isolation of *Salmonella* (excluding *S. Typhi* and *S. Paratyphi* [A, B (tartrate negative), and C])* from a clinical specimen  
Notes:  
* *S. Typhi* is reportable as *Salmonella Typhi*.  
* *S. Paratyphi* is reportable as *Salmonella Paratyphi*.  
Note: As required by TAC all *Salmonella* spp. isolates must be submitted to the DSHS Laboratory. |
Shiga toxin-producing *Escherichia coli* (STEC) 11563

An infection of variable severity characterized by diarrhea (often bloody) and abdominal cramps. Illness can be complicated by hemolytic uremic syndrome (HUS) or thrombotic thrombocytopenic purpura (TTP); asymptomatic infections also can occur and the organism can cause extraintestinal infections.

**Confirmed:** A case that meets the laboratory criteria for diagnosis; when available, O and H antigen serotype characterization should be reported

**Probable:**
- A case with isolation of *E. coli* O157 from a clinical specimen, without confirmation of H antigen, with detection of Shiga toxin or detection of Shiga toxin genes, OR
- A clinically compatible case that is epidemiologically linked to a confirmed or probable case with laboratory evidence, OR
- A clinically compatible illness in a person with identification of an elevated antibody titer to a known Shiga toxin-producing *E. coli* serotype, OR
- A clinically compatible illness in a person with detection of Shiga toxin or Shiga toxin genes in a clinical specimen using a CIDT and no known isolation of *Shigella* from a clinical specimen, OR
- A clinically compatible illness in a person with detection of *E. coli* O157 or Shiga toxin-producing *E. coli* in a clinical specimen using a CIDT, OR
- A clinically compatible illness in a person that is a member of a risk group as defined by public health authorities during an outbreak

**Suspect:**
- Identification of an elevated antibody titer against a known Shiga toxin-producing serogroup of *E. coli* in a person with no known clinical compatibility, OR
- Detection of Shiga toxin or Shiga toxin genes in a clinical specimen using a CIDT and no known isolation of *Shigella* from a clinical specimen in a person with no known clinical compatibility, OR
- Detection of *E. coli* O157 or Shiga toxin-producing *E. coli* in a clinical specimen using a CIDT with no known clinical compatibility, OR
- A person with a diagnosis of post-diarrheal HUS/TTP

**Notes:**
- EIA and/or PCR positive results for Shiga toxin-production, in the absence of isolation of *E.coli*, can only qualify a case as “probable.”
- Cases meeting confirmed or probable criteria for both STEC and HUS should be reported separately under each condition.
- A case should not be counted as a new case if a positive laboratory result is reported within 180 days of a previously reported positive laboratory result in the same individual, OR
- When two or more different serogroups are identified in one or more specimens from the same individual, each serogroup/serotype should be reported as a separate case.

**Isolation of Escherichia coli from a clinical specimen with detection of Shiga toxin or Shiga toxin genes,**
**OR**
**Isolation of Escherichia coli O157:H7 from a clinical specimen,**

**Notes:**
*Escherichia coli* non-O157:H7 isolates must also have Shiga toxin-production verified to qualify for the “confirmed” case status. Shiga toxin can be demonstrated by EIA or PCR testing.

As required by TAC, for all cases of Shiga toxin-producing *E.coli* infections, including *E.coli* O157:H7 and cases where Shiga-toxin activity is demonstrated, available isolates or specimens must be submitted to the DSHS Laboratory.
| **Shigellosis** 11010 | An illness of variable severity characterized by diarrhea, fever, nausea, cramps, and tenesmus. Asymptomatic infections can occur.  
**Confirmed:** A case that is laboratory confirmed. When available, *Shigella* serogroup or species and serotype characterization should be reported.  
**Probable:**  
- A case with *Shigella* spp. or *Shigella* detected, in a clinical specimen, by use of culture independent laboratory methods (non-culture based), **OR**  
- A clinically compatible case that is epidemiologically linked to a case that meets the probable or confirmed laboratory criteria for diagnosis  
**Notes:**  
- Both asymptomatic infections and infections at sites other than the gastrointestinal tract, if laboratory confirmed, are considered confirmed cases that should be reported.  
- A case should not be counted as a new case if laboratory results were reported within 90 days of a previously reported infection in the same individual, unless additional information is available indicating a separate infection, e.g., different serotype.  
**Isolation of Shigella from a clinical specimen** |  
**Smallpox** 11800 | An illness with acute onset of fever $\geq 101^\circ$ F ($\geq 38.3$ $^\circ$ C) followed by a rash characterized by firm, deep seated vesicles or pustules in the same stage of development without other apparent cause.  
**Confirmed:** A case of smallpox that is laboratory confirmed, or a case that meets the clinical case definition and is epidemiologically linked to a laboratory confirmed case  
**Probable:** A case that meets the clinical case definition without laboratory confirmation or epidemiological link to a confirmed case, **OR** a case with an atypical presentation of smallpox (e.g., hemorrhagic type, flat type, and variola sine eruptione) that has an epidemiological link to a confirmed case of smallpox.  
(Detailed clinical description is available on the CDC web site, see [https://www.cdc.gov/smallpox/clinicians/clinical-disease.html](https://www.cdc.gov/smallpox/clinicians/clinical-disease.html).)  
**Suspect:** A case with a generalized, acute vesicular or pustular rash illness with fever preceding development of rash by 1-4 days  
**Exclusion Criteria:** A case can be excluded as a suspect or probable smallpox case if an alternative diagnosis fully explains the illness or appropriate clinical specimens are negative for laboratory criteria for smallpox.  
**Note:** The smallpox case definition above is to be used only during post-event surveillance. Pre-event surveillance relies on a highly specific clinical case definition focused on identifying a classic case (ordinary type) of smallpox. In the absence of known smallpox disease, the predictive value of a positive smallpox diagnostic test is extremely low, therefore, testing to rule out smallpox should be limited to cases that fit the clinical case definition in order to lower the risk of obtaining a false positive test result.  
- **Polymerase chain reaction (PCR) identification of variola DNA in a clinical specimen,**  
- **Isolation of smallpox (variola) virus from a clinical specimen (National LRN laboratory only; confirmed by variola PCR)**  
**Note:** Laboratory diagnostic testing for variola virus should be conducted in a CDC Laboratory Response Network (LRN) laboratory utilizing LRN-approved PCR tests and protocols for variola virus. Initial confirmation of a smallpox outbreak requires additional testing at CDC. Generic orthopox PCR and negative stain electron microscopy (EM) identification of a pox virus in a clinical specimen are suggestive of an orthopox virus infection but not diagnostic for smallpox. |
| Spotted fever rickettsiosis | Spotted fever rickettsioses (SFR) are tick-borne infections caused by some members of the genus *Rickettsia*. The most well-known SFR is Rocky Mountain spotted fever (RMSF), an illness caused by *Rickettsia rickettsii*. Disease onset for RMSF averages one week following a tick bite. Illness is characterized by acute onset of fever and can be accompanied by headache, malaise, myalgia, nausea/vomiting, or neurologic signs; a macular or maculopapular rash may appear 4-7 days following onset in many (~80%) patients, often present on the palms and soles. RMSF can be fatal in as many as 20% of untreated cases, and severe fulminant disease can occur. In addition to RMSF, human illness associated with other spotted fever group *Rickettsia* (SFGR) species, including infection with *R. parkeri*, has also been reported. In these patients, clinical presentation appears similar to, but can be milder than, RMSF; the presence of an eschar at the site of tick attachment has been reported for some other SFR.  

**Clinical evidence:** Acute illness lasting less than 30 days with fever and one or more of the following: rash, eschar, headache, myalgia, anemia, thrombocytopenia, or any hepatic transaminase elevation.

**Confirmed:** Clinically compatible case (meets clinical evidence criteria) that is laboratory confirmed

**Probable:** Clinically compatible case with serological evidence of elevated IgG antibody reactive with SFGR antigen* by IFA (serologic titer of ≥1:128; specimen collected within 60 days of onset) and the absence of a more likely clinical explanation

Notes:
- Because antibodies for rickettsial diseases can be cross-reactive, specimens should be tested against a panel* of *Rickettsia* antigens, including, at a minimum, *R. rickettsii* and *R. typhi*, to differentiate between SFGR and non-SFGR species.
- A case should not be counted as new if the case has ever previously been reported for the same condition.

*Specimens can be forwarded to the DSHS Serology lab for rickettsial panel testing.*

See *Rickettsia* Classification

| **Clinical evidence:** | Serological evidence of a four-fold increase in IgG-specific antibody titer reactive with SFGR** antigen by IFA between paired acute (taken in the first two weeks after illness onset) and convalescent (taken two to ten weeks after acute specimen collection) serum specimens, OR

- Detection of SFGR** nucleic acid in a clinical specimen via amplification of a species-specific target by PCR assay, OR

- Demonstration of SFGR** antigen in a biopsy or autopsy specimen by IHC, OR

- Isolation of SFGR** from a clinical specimen in cell culture and molecular confirmation (e.g., PCR or sequence).

**The spotted fever group *Rickettsia* (SFGR) are *R. aeschlimannii, R. africae, R. australis, R. conorii, R. heilongjiangensis, R. helvetica, R. honei, R. japonica, R. mansonii, R. massiliae, R. parkeri, R. rickettsii, R. sibirica, R. sibirica mongolotimonae, and R. slovaca.* *Rickettsia* spp. excluded from this group are *R. felis* and *R. akari.*

Note: DNA-detection methods outside of PCR (e.g. cell-free DNA) does not meet lab criteria. Samples can be forwarded for additional testing at the DSHS lab or CDC. |
Streptococcal toxic shock syndrome - [outbreaks only] 11700

Streptococcal toxic-shock syndrome (STSS) is a severe illness associated with invasive or noninvasive group A streptococcal (*Streptococcus pyogenes*) infection. STSS may occur with infection at any site but most often occurs in association with infection of a cutaneous lesion. Signs of toxicity and a rapidly progressive clinical course are characteristic, and the case fatality rate may exceed 50%.

An illness with the following clinical manifestations:

1) Hypotension defined by a systolic blood pressure less than or equal to 90 mm Hg for adults or less than the fifth percentile by age for children aged less than 16 years, 

AND

2) Multi-organ involvement characterized by two or more of the following:

   - **Renal Impairment**: Creatinine greater than or equal to 2 mg/dL (greater than or equal to 177 µmol/L) for adults or greater than or equal to twice the upper limit of normal for age. In patients with preexisting renal disease, a greater than twofold elevation over the baseline level.
   - **Coagulopathy**: Platelets less than or equal to 100,000/mm³ (less than or equal to 100 x 10⁹/L) or disseminated intravascular coagulation, defined by prolonged clotting times, low fibrinogen level, and the presence of fibrin degradation products
   - **Liver Involvement**: Alanine aminotransferase, aspartate aminotransferase, or total bilirubin levels greater than or equal to twice the upper limit of normal for the patient's age. In patients with preexisting liver disease, a greater than twofold increase over the baseline level.
   - **Acute Respiratory Distress Syndrome**: Defined by acute onset of diffuse pulmonary infiltrates and hypoxemia in the absence of cardiac failure or by evidence of diffuse capillary leak manifested by acute onset of generalized edema, or pleural or peritoneal effusions with hypoalbuminemia
   - **A generalized erythematous macular rash that may desquamate**
   - **Soft-tissue necrosis, including necrotizing fasciitis or myositis, or gangrene**

Confirmed: A case that meets the clinical case definition and is laboratory confirmed with isolation of group A *Streptococcus* from a normally sterile site (e.g., blood or cerebrospinal fluid or, less commonly, joint, pleural, or pericardial fluid)

Probable: A case that meets the clinical case definition in the absence of another identified etiology for the illness and with isolation of group A *Streptococcus* from a non-sterile site

Note: Enter all confirmed and probable STSS cases as confirmed group A *Streptococcus*, invasive disease, code 11710.

- Isolation of group A *Streptococcus* (*S. pyogenes*) (GAS)
<table>
<thead>
<tr>
<th>Disease Description</th>
<th>Disease Code</th>
<th>Description</th>
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<tbody>
<tr>
<td><strong>Streptococcus pneumoniae, invasive disease (IPD)</strong> 11723*</td>
<td>Streptococcus pneumoniae bacteria cause many clinical syndromes, depending on the site of infection (e.g., acute otitis media, pneumonia, bacteremia, or meningitis). Only invasive Streptococcus pneumoniae is reportable.</td>
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<tr>
<td><strong>Confirmed:</strong></td>
<td>A case that is laboratory confirmed</td>
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<tr>
<td><strong>Probable:</strong></td>
<td>A case with detection of S. pneumoniae from a normally sterile site using a culture independent diagnostic test (CIDT) (e.g., PCR, antigen-based tests) without isolation of the bacteria</td>
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<tr>
<td>Note: Positive lab results from a specimen collected more than 30 days after the collection date of a prior case should be counted as a new case. If specimen collection occurred within 30 days of the collection date of a prior case, it should not be counted as a new case.</td>
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<td><strong>Note:</strong> Code 11717 was used prior to 2010 and for 2010 there are cases under both codes.</td>
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<tr>
<td><strong>Taenia solium and undifferentiated Taenia infection</strong> 80680</td>
<td>Taeniasis is an intestinal infection with the adult stage of the pork (T. solium) or beef (T. saginata) tapeworm. Clinical manifestations of infection with the adult worm, if present, are variable and can include nervousness, insomnia, anorexia, weight loss, abdominal pain, and digestive disturbances; many infections are asymptomatic. Taeniasis is usually a nonfatal infection, but the larval stage of T. solium can cause fatal cysticercosis.</td>
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<tr>
<td><strong>Confirmed:</strong></td>
<td>Laboratory identification of the presence of T. solium proglottids, eggs, or antigens in a clinical specimen</td>
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<tr>
<td><strong>Probable:</strong></td>
<td>Laboratory identification of the presence of undifferentiated Taenia spp. tapeworm proglottids or eggs in a clinical specimen</td>
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<tr>
<td><strong>Note:</strong> Serotyping of isolates can be performed at the DSHS laboratory. Serotyping is required by TAC for invasive Streptococcus pneumoniae cases on all isolates from children under 5 years old.</td>
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<tr>
<td><strong>Tetanus</strong> 10210</td>
<td>Acute onset of hypertonia and/or painful muscular contractions (usually of the muscles of the jaw and neck) and generalized muscle spasms without other apparent medical cause.</td>
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</tr>
<tr>
<td><strong>Probable:</strong></td>
<td>A clinically compatible case, as reported by a health-care professional</td>
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<tr>
<td><strong>Note:</strong> Eggs of T. solium and T. saginata cannot be differentiated morphologically. Specific diagnosis is based on the morphology of the scolex (head) and/or gravid proglottids.</td>
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<tr>
<td><strong>Infection with an adult tapeworm is diagnosed by identification of proglottids (segments), eggs, or antigens of the worm in the feces or on anal swabs</strong></td>
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</table>

"Isolation of S. pneumoniae from a normally sterile site (e.g., blood or cerebrospinal fluid, or, less commonly, joint, pleural, or pericardial fluid)"
| Trichinellosis (Trichinosis) | A disease caused by ingestion of *Trichinella* larvae. The disease has variable clinical manifestations. Common signs and symptoms include eosinophilia, fever, myalgia, and periorbital edema.  
**Confirmed:** A clinically compatible case that is laboratory confirmed in the patient  
**Probable:** A clinically compatible illness in a person who shared an epidemiologically implicated meal or ate an epidemiologically implicated meat product, **OR**  
a clinically compatible illness in a person who consumed a meat product in which the parasite was demonstrated  
**Suspect:** A person without clinically compatible illness who shared an implicated meal or ate an implicated meat product, has no known prior history of *Trichinella* infection, and has a positive serologic test for trichinellosis  
**Notes:**  
- Epidemiologically implicated meals or meat products are defined as a meal/meat product that was consumed by a person who subsequently developed a clinically compatible illness that was laboratory confirmed.  
- Subsequent cases of trichinellosis experienced by one individual should only be counted if there is a clinically-compatible illness **AND** a compatible exposure.  
**Demonstration of *Trichinella* spp. larvae in tissue obtained by muscle biopsy, **OR**  
**Positive serologic test for *Trichinella* spp.** |
| Trichuriasis | A parasitic infection caused by the soil-transmitted helminth *Trichuris trichiura* (whipworm). People with light infections are usually asymptomatic. Cases with heavy infections may experience frequent, painful passage of stool that contains a mixture of mucus, water, and blood. Rectal prolapse can also occur. Heavy infections in children can lead to severe anemia, delayed physical growth and impaired cognitive development.  
**Confirmed:** A case that is laboratory confirmed  
**Notes:**  
- Microscopic identification of *Trichuris* eggs or worms in feces, **OR**  
- Observation during sigmoidoscopy, proctoscopy, or colonoscopy of *Trichuris* worms characterized by a threadlike form with an attenuated, whip-like end, **OR**  
- Identification of *Trichuris* worms on prolapsed rectal mucosa |
The signs and symptoms of tularemia vary depending on how the bacteria enter the body. Illness ranges from mild to life-threatening. All forms are accompanied by fever, which can be as high as 104°F. Clinical diagnosis is supported by evidence or history of a tick or deerfly bite, exposure to tissues of a mammalian host of *Francisella tularensis*, or exposure to potentially contaminated water. Illness is characterized by several distinct forms, including the following:

- Ulceroglandular: cutaneous ulcer with regional lymphadenopathy
- Glandular: regional lymphadenopathy with no ulcer
- Oculoglandular: conjunctivitis with preauricular lymphadenopathy
- Oropharyngeal: stomatitis or pharyngitis or tonsillitis and cervical lymphadenopathy
- Pneumonic: primary pleuropulmonary disease
- Typhoidal: febrile illness without early localizing signs and symptoms

**Confirmed:** A clinically compatible case with confirmatory laboratory results

**Probable:** A clinically compatible case with laboratory results indicative of presumptive infection and the absence of a more likely clinical explanation:

- Elevated serum antibody titer(s)* to *F. tularensis* antigen (without documented fourfold or greater change) in a patient with no history of tularemia vaccination, **OR**
- Detection of *F. tularensis* in a clinical or autopsy specimen by fluorescent assay **OR**
- Detection of *F. tularensis* in a clinical or autopsy specimen by PCR

*Most ELISAs are qualitative tests and do not provide a titer. Some commercial labs perform reflex titer testing for ELISA-positive specimens; contact the commercial lab for these results. Samples that are ELISA-positive with no reflex testing should be forwarded to DSHS for tularemia serologic testing to validate results.

**Isolation of *F. tularensis* in a clinical or autopsy specimen, **OR**

- Four-fold or greater rise in serum antibody titer* to *F. tularensis* antigen between acute and convalescent specimens.

Note: As required by **TAC**, all *F. tularensis* isolates must be submitted to the DSHS Laboratory.
Flea-borne typhus is a rickettsial disease whose course resembles that of louse-borne typhus, but is generally milder. The onset is variable, often sudden and marked by headache, chills, fatigue, fever, and general body aches. A macular rash may appear on the 5th or 6th day, initially on the upper trunk, followed by spread to the entire body, but usually not to the face, palms or soles. Absence of louse infestation, geographic and seasonal distribution, and sporadic occurrence of the disease help to differentiate it from louse-borne typhus.

**Clinical evidence:** Acute illness lasting less than 30 days with fever and two or more of the following: headache, myalgia, rash, nausea/vomiting, thrombocytopenia, or any elevated liver enzyme **Confirmed:** Clinically compatible case that is laboratory confirmed

**Probable:** Clinically compatible case with evidence of epidemiologic linkage*, the absence of a more likely clinical explanation, and supportive lab evidence:

- Serologic evidence of elevated IgG at a titer of ≥1:128 reactive with *R. typhi* antigen by IFA in a sample taken within 60 days of illness onset, **OR**
- Serologic evidence of elevated IgM at a titer of ≥1:256 reactive with *R. typhi* antigen by IFA in a sample taken within 60 days of illness onset.

*Epidemiologic linkage criteria:* Was in same household or had same defined exposure as a confirmed case within the past 14 days before onset of symptoms, **OR** likely vector exposure in an area with suitable seasonal and ecological conditions for potential local vector-borne transmission

**Notes:**
- Because antibodies for rickettsial diseases can be cross-reactive, specimens should be tested against a panel** of *Rickettsia* antigens, including, at a minimum, *R. rickettsii* and *R. typhi*, to differentiate between SFG and non-SFG *Rickettsia* spp.
- According to CDC, rickettsial IgM tests lack specificity (resulting in false positives); thus, IgG titers are much more reliable.
- A case should not be counted as new if the case has ever previously been reported for the same condition.

**Specimens can be forwarded to the DSHS Serology Laboratory for rickettsial panel testing.**

See *Rickettsia Classification*
| Typhus fever (epidemic, louse-borne) | A rickettsial disease caused by *Rickettsia prowazekii* and transmitted by the human body louse. The illness may have a variable onset which is often sudden and marked by headache, chills, prostration, fever, and general body aches. A macular rash may appear on the 5th or 6th day, initially on the upper trunk, followed by spread to the entire body, but usually not to the face, palms or soles. The rash is often difficult to observe on dark skin. Toxemia is usually pronounced, and the disease terminates by rapid defervescence after about 2 weeks of fever.  
**Confirmed:** Clinically compatible case that is laboratory confirmed  
**Probable:** Clinically compatible case with supportive laboratory results and the absence of a more likely clinical explanation:  
- IFA serologic titer of $\geq 1:128$  
Note: The IFA test is most commonly used for laboratory confirmation, but it does not discriminate between louse-borne and flea-borne typhus unless the sera are differentially absorbed with the respective rickettsial antigen prior to testing.  
See *Rickettsia Classification* |
| Vancomycin-intermediate *Staphylococcus aureus* (VISA) | *Staphylococcus aureus* can produce a variety of syndromes with clinical manifestations including skin and soft tissue lesions, empyema, pyarthrosis, bloodstream infection, pneumonia, osteomyelitis, septic arthritis, endocarditis, sepsis, and meningitis.  
**Confirmed:** A vancomycin-intermediate *Staphylococcus aureus* from any body site that is laboratory confirmed. (MIC: 4-8 µg/ml)  
Note: The DSHS Laboratory uses the Etest for confirmation of resistance. Etest generates MIC values from a continuous scale and can give results in-between conventional two-fold dilutions. According to manufacturer’s protocol, a value which falls between standard two-fold dilutions is rounded up to the next upper two-fold value before categorization so that a MIC of 3µg/ml is reported as intermediate resistance.  
Additional information on VISA can be found at: https://www.cdc.gov/hai/organisms/visa_vrsa/visa_vrsa.html |
|   | Four-fold or greater rise in IgG-specific antibody titer to *R. prowazekii* antigen by IFA test in acute and convalescent specimens ideally taken at least 2 weeks apart,  
**OR**  
Positive PCR assay to *R. prowazekii*,  
**OR**  
Demonstration of positive *R. prowazekii* IHC of skin lesion (biopsy) or organ tissue (autopsy) |
|   | Isolation of *Staphylococcus aureus* from any body site,  
**AND**  
Intermediate-level resistance (MIC: 4-8 µg/ml) of the *Staphylococcus aureus* isolate to vancomycin, detected and defined according to CLSI approved standards and recommendations.  
**AND**  
Confirmed by the DSHS Laboratory  
Note: As required by TAC, all *Staphylococcus aureus* isolates with a vancomycin MIC greater than 2 µg/mL must be submitted to the DSHS Laboratory. Please contact a DSHS HAI Epidemiologist or the DSHS Laboratory for additional information on available laboratory support.  
**http://www.cdc.gov/HAI/settings/lab/visa_vrsa_lab_detection.html** |
| **Vancomycin-resistant Staphylococcus aureus (VRSA)**<br>11665 | *Staphylococcus aureus* can produce a variety of syndromes with clinical manifestations including skin and soft tissue lesions, empyema, pyarthrosis, bloodstream infection, pneumonia, osteomyelitis, septic arthritis, endocarditis, sepsis, and meningitis.  
**Confirmed:** A vancomycin-resistant *Staphylococcus aureus* from any body site that is laboratory confirmed. (MIC: ≥ 16 µg/ml)  
Additional information on VRSA can be found at: [https://www.cdc.gov/hai/organisms/visa_vrsa/visa_vrsa.html](https://www.cdc.gov/hai/organisms/visa_vrsa/visa_vrsa.html) | • Isolation of *Staphylococcus aureus* from any body site,  
**AND**  
• High-level resistance of the *Staphylococcus aureus* isolate to vancomycin (MIC: ≥16 µg/ml), detected and defined according to CLSI approved standards and recommendations.  
**AND**  
• Confirmed by the DSHS Laboratory  
Note: As required by TAC, all *Staphylococcus aureus* isolates with a vancomycin MIC greater than 2 µg/mL must be submitted to the DSHS Laboratory. Please contact a DSHS HAI Epidemiologist or the DSHS Laboratory for additional information on available laboratory support. [http://www.cdc.gov/HAI/settings/lab/visa_vrsa_lab_detection.html](http://www.cdc.gov/HAI/settings/lab/visa_vrsa_lab_detection.html) |
| **Varicella (chickenpox)**<br>10030 | An illness with acute onset of diffuse (generalized) maculopapulovesicular rash without other apparent cause. In vaccinated persons who develop varicella more than 42 days after vaccination (breakthrough disease), the disease is almost always mild with fewer than 50 skin lesions and shorter duration of illness. The rash can also be atypical in appearance (maculopapular with few or no vesicles).  
**Confirmed:** A case that meets the clinical case definition **AND** is either laboratory confirmed,  
**OR** epidemiologically linked to another probable or confirmed case  
**Probable:** A case that meets the clinical case definition **without** epidemiologic linkage or laboratory confirmation  
Note: Two or more patients that meet clinical case definition and are epidemiologically linked to one another meet the confirmed case definition. | • Isolation of varicella-zoster virus (VZV) from a clinical specimen,  
**OR**  
• Varicella antigen detected by direct fluorescent antibody (DFA),  
**OR**  
• Varicella-specific nucleic acid detected by polymerase chain reaction (PCR),  
**OR**  
• Significant rise in serum varicella immunoglobulin G (IgG) antibody level by any standard serologic assay |
<table>
<thead>
<tr>
<th><strong>Vibrio parahaemolyticus</strong></th>
<th>An intestinal disorder commonly involving watery diarrhea and abdominal cramps, and occasionally with nausea, vomiting, fever and headache. A quarter of cases develop a dysentery-like illness associated with bloody or mucoid stools, high fever and high WBC count. Wound infections can also occur. Typically, it is a disease of moderate severity lasting 1-7 days; systemic infection and death rarely occur. <strong>Confirmed:</strong> A case that is laboratory confirmed <strong>Probable:</strong> ▪ A case with <em>Vibrio parahaemolyticus</em> detected, in a clinical specimen, by use of culture independent laboratory methods (non-culture based), OR ▪ A clinically compatible case that is epidemiologically linked to a case that meets the probable or confirmed laboratory criteria for diagnosis Note: A case should not be counted as a new case if laboratory results were reported within 30 days of a previously reported infection in the same individual, unless additional information is available indicating a separate infection, e.g., different species</th>
<th>▪ Isolation of <em>Vibrio parahaemolyticus</em> from a clinical specimen Note: As required by TAC all <em>Vibrio</em> species isolates must be submitted to the DSHS Laboratory.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Vibrio vulnificus</strong></td>
<td>Infection with <em>Vibrio vulnificus</em> produces septicemia in persons with chronic liver disease, chronic alcoholism or hemochromatosis, or those who are immunosuppressed. The disease appears 12 hours to 3 days after eating raw or undercooked seafood, especially oysters. One third of patients are in shock when they present for care or develop hypotension within 12 hours after hospital admission. Three quarters of patients have distinctive bullous skin lesions; thrombocytopenia is common and there is often evidence of disseminated intravascular coagulation. <em>V. vulnificus</em> can also infect wounds sustained in coastal or estuarine waters; wounds range from mild, self-limited lesions to rapidly progressive cellulitis and myositis that can mimic clostridial myonecrosis in the rapidity of spread and destructiveness. <strong>Confirmed:</strong> A case that is laboratory confirmed <strong>Probable:</strong> ▪ A case with <em>Vibrio vulnificus</em> detected, in a clinical specimen, by use of culture independent laboratory methods (non-culture based), OR ▪ A clinically compatible case that is epidemiologically linked to a case that meets the probable or confirmed laboratory criteria for diagnosis Note: A case should not be counted as a new case if laboratory results were reported within 30 days of a previously reported infection in the same individual, unless additional information is available indicating a separate infection, e.g., different species</td>
<td>▪ Isolation of <em>Vibrio vulnificus</em> from a clinical specimen Note: As required by TAC all <em>Vibrio</em> species isolates must be submitted to the DSHS Laboratory.</td>
</tr>
</tbody>
</table>
### Vibriosis, other or unspecified

<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>11540</td>
<td>An infection of variable severity characterized by diarrhea and vomiting, primary septicemia, or wound infections. Asymptomatic infections can occur, and the organism can cause extraintestinal infections. <strong>Confirmed:</strong> A case that is laboratory confirmed. <strong>Probable:</strong> A case with a species of the family <em>Vibrionaceae</em> (other than <em>Vibrio parahaemolyticus</em>, <em>Vibrio vulnificus</em>, and toxigenic <em>Vibrio cholerae</em> O1 or O139) detected, in a clinical specimen, by use of culture independent laboratory methods (non-culture based), OR A clinically compatible case that is epidemiologically linked to a case that meets the probable or confirmed laboratory criteria for diagnosis. Note: A case should not be counted as a new case if laboratory results were reported within 30 days of a previously reported infection in the same individual, unless additional information is available indicating a separate infection, e.g., different species.</td>
</tr>
</tbody>
</table>

### Viral Hemorrhagic Fever (VHF) non-Ebola *

<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>11640</td>
<td>Crimean-Congo HF</td>
</tr>
<tr>
<td>11648</td>
<td>Guaranito HF</td>
</tr>
<tr>
<td>11638</td>
<td>Junin (Argentine) HF</td>
</tr>
<tr>
<td>11632</td>
<td>Lassa fever</td>
</tr>
<tr>
<td>11644</td>
<td>Lujo HF</td>
</tr>
<tr>
<td>11637</td>
<td>Machupo (Bolivian) HF</td>
</tr>
<tr>
<td>11631</td>
<td>Marburg fever</td>
</tr>
<tr>
<td>11639</td>
<td>Sabia (Brazilian) HF</td>
</tr>
</tbody>
</table>

*Viral Hemorrhagic Fevers include Ebola - please see Ebola case definition for Ebola specific information*
| **Yellow fever**<br>10660 | Yellow fever virus is a mosquito-borne flavivirus that is closely related to dengue, Japanese encephalitis, West Nile, and Zika viruses. Yellow fever is preventable by a safe and effective vaccine.  
Most yellow fever virus infections are asymptomatic. Following an incubation period of 3–9 days, approximately one-third of infected people develop symptomatic illness characterized by fever and headache. Other clinical findings include chills, vomiting, myalgia, lumbosacral pain, and bradycardia relative to elevated body temperature. An estimated 5%–25% of patients progress to more severe disease, including jaundice, renal insufficiency, cardiovascular instability, or hemorrhage (e.g., epistaxis, hematemesis, melena, hematuria, petechiae, or ecchymoses). The case-fatality rate for severe yellow fever is 30%–60%.  
**Clinical criteria**: An acute illness with at least one of the following: fever, jaundice, or elevated total bilirubin ≥3 mg/dl, and the absence of a more likely clinical explanation.  
**Confirmed**: A clinically compatible case that is laboratory confirmed  
**Probable**: A clinically compatible case with supportive serology:  
- Yellow fever virus-specific IgM antibodies in CSF or serum, AND negative IgM results for other arboviruses endemic to the region where exposure occurred, AND no history of yellow fever vaccination,  
- Epidemiologic linkage to a confirmed yellow fever case or having visited or resided in an area with a risk of yellow fever in the 2 weeks before onset of illness.  
- Isolation of yellow fever virus from, or demonstration of yellow fever viral antigen or nucleic acid in, tissue, blood, CSF, or other body fluid, AND no history of yellow fever vaccination within 30 days before onset of illness unless there is molecular evidence of infection with wild-type yellow fever virus, OR  
- Four-fold or greater rise or fall in yellow fever virus-specific neutralizing antibody titers in paired sera, AND no history of yellow fever vaccination within 30 days before onset of illness, OR  
- Yellow fever virus-specific IgM antibodies in CSF or serum with confirmatory virus-specific neutralizing antibodies in the same or a later specimen, AND no history of yellow fever vaccination. |
| **Yersiniosis**<br>11565 | An illness characterized by acute diarrhea (may be bloody) with abdominal pain. Other symptoms include acute mesenteric lymphadenitis mimicking appendicitis, exudative pharyngitis, and systemic infection.  
Note: Extra-intestinal manifestations may also be present, such as abscess, which could be a source for testing, and reactive arthritis and erythema nodosum, which are often immunologic phenomena not directly caused by the infection. These manifestations are not required as part of the clinical criteria.  
**Confirmed**: A case that is laboratory confirmed  
**Probable**: A clinically compatible case that is epidemiologically linked to a confirmed case, or a clinically compatible case identified through use of a culture independent diagnostic test (CIDT) such as PCR.  
- Note: A case should not be counted as a new case if laboratory results were reported within 365 days of a previously reported infection in the same individual  
- Isolation* of *Yersinia* (except *Y. pestis**) in a clinical specimen  
*As required by TAC all *Yersinia pestis* isolates must be submitted to the DSHS Laboratory.  
**For *Yersinia pestis* isolates, see Plague
Zika disease, congenital
Clinical evidence: A neonate with one or more of the following not explained by another etiology:
- congenital microcephaly
- congenital intracranial calcification
- other structural brain or eye abnormalities
- other congenital central nervous system-related abnormalities including defects such as clubfoot or multiple joint contractures

Confirmed: A clinically compatible neonate with laboratory confirmation.

Probable: A clinically compatible neonate whose mother has an epidemiologic link* OR meets laboratory criteria for recent ZIKV or flavivirus infection; AND the neonate has laboratory evidence of recent ZIKV or flavivirus infection by:
- Positive ZIKV IgM antibody test of serum or CSF within 2 days of birth**; AND
  - positive neutralizing antibody titers against ZIKV and dengue or other flaviviruses endemic to the region where exposure occurred; OR
  - negative dengue virus IgM antibody test and no neutralizing antibody test performed

*Epidemiologic link defined as one or more of the following:
- Resides in or recent travel to an area with known ZIKV transmission, OR
- Sexual contact with a confirmed or probable case of ZIKV infection or person with recent travel to an area with known ZIKV transmission; OR
- Receipt of blood or blood products within 30 days of symptom onset; OR
- Organ or tissue transplant recipient within 30 days of symptom onset; OR
- Association in time or place with a confirmed or probable case; OR
- Likely vector exposure in an area with suitable seasonal and ecological conditions for potential local vector-borne transmission

**The requirement that samples be collected within 2 days only applies to areas with ongoing local Zika transmission.
**Zika disease, non-congenital**

A mosquito-borne viral illness transmitted by *Aedes* mosquitoes, including *Ae. aegypti* and *Ae. albopictus*. Infection is asymptomatic in up to 80% of cases and clinical illness, when it occurs, is typically mild and lasts for several days to a week. Transmission of Zika virus (ZIKV) *in utero* has been associated with severe birth outcomes, including microcephaly and fetal loss.

**Clinical evidence:** An individual with one or more of the following not explained by another etiology:
- Clinically compatible illness that includes:
  - acute onset of fever (measured or reported), or
  - rash, or
  - arthralgia, or
  - conjunctivitis
- Complication of pregnancy
  - fetal loss, or
  - fetus or neonate with congenital microcephaly, congenital intracranial calcification, other structural brain or eye abnormalities, or other congenital central nervous system-related abnormalities including defects such as clubfoot or multiple joint contractures (note: if detected prior to infant’s birth, the relevant birth defects must be documented in at least two separate ultrasounds and/or verified at birth)
- Guillain-Barré syndrome or other neurologic manifestations

**Confirmed:** A clinically compatible individual with laboratory confirmation.

**Probable:** A clinically compatible individual with an epidemiologic link* AND laboratory evidence of recent ZIKV or flavivirus infection by:
- Positive ZIKV IgM antibody test of serum or CSF with:
  - positive neutralizing antibody titers against ZIKV and dengue or other flaviviruses endemic to the region where exposure occurred; OR
  - negative dengue virus IgM antibody test and no neutralizing antibody test performed

*Epidemiologic link defined as one or more of the following:
- Resides in or recent travel to an area with known ZIKV transmission, OR
- Sexual contact with a confirmed or probable case of ZIKV infection or person with recent travel to an area with known ZIKV transmission; OR
- Receipt of blood or blood products within 30 days of symptom onset; OR
- Organ or tissue transplant recipient within 30 days of symptom onset; OR
- Association in time or place with a confirmed or probable case; OR
- Likely vector exposure in an area with suitable seasonal and ecological conditions for potential local vector-borne transmission

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- Detection of ZIKV by culture, viral antigen or viral RNA in serum, CSF, tissue, or other specimen (i.e. amniotic fluid, urine, semen, saliva) with a validated diagnostic test OR
- Positive ZIKV IgM antibody test in serum or CSF with positive ZIKV neutralizing antibody titers and negative neutralizing antibody titers against dengue or other flaviviruses endemic to the region where exposure occurred
<table>
<thead>
<tr>
<th>Zika infection, congenital</th>
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</thead>
<tbody>
<tr>
<td><strong>Confirmed:</strong> A neonate who does not meet clinical criteria for congenital Zika disease, BUT who meets confirmatory laboratory criteria.</td>
</tr>
<tr>
<td><strong>Probable:</strong> A neonate who does not meet clinical criteria for congenital Zika disease whose mother has an epidemiologic link* OR meets laboratory criteria for recent ZIKV or flavivirus infection; <strong>AND</strong> the neonate has laboratory evidence of recent ZIKV or flavivirus infection by:</td>
</tr>
<tr>
<td>▪ Positive ZIKV IgM antibody test of serum or CSF within 2 days of birth**; <strong>AND</strong></td>
</tr>
<tr>
<td>▪ positive neutralizing antibody titers against ZIKV and dengue or other flaviviruses endemic to the region where exposure occurred; <strong>OR</strong></td>
</tr>
<tr>
<td>▪ negative dengue virus IgM antibody test and no neutralizing antibody test performed</td>
</tr>
<tr>
<td>*Epidemiologic link defined as one or more of the following:</td>
</tr>
<tr>
<td>▪ Resides in or recent travel to an area with known ZIKV transmission, <strong>OR</strong></td>
</tr>
<tr>
<td>▪ Sexual contact with a confirmed or probable case of ZIKV infection or person with recent travel to an area with known ZIKV transmission; <strong>OR</strong></td>
</tr>
<tr>
<td>▪ Receipt of blood or blood products within 30 days of symptom onset; <strong>OR</strong></td>
</tr>
<tr>
<td>▪ Organ or tissue transplant recipient within 30 days of symptom onset; <strong>OR</strong></td>
</tr>
<tr>
<td>▪ Association in time or place with a confirmed or probable case; <strong>OR</strong></td>
</tr>
<tr>
<td>▪ Likely vector exposure in an area with suitable seasonal and ecological conditions for potential local vector-borne transmission</td>
</tr>
<tr>
<td><strong>Note:</strong> Zika IgM may be detectable between 1-12 weeks after infection but may persist for months to years and interpretation is complicated by cross-reactivity with other flaviviruses. In the absence of clinical illness, interpretation of Zika IgM and/or PRNT results is based on epidemiological context.</td>
</tr>
</tbody>
</table>

- Detection of ZIKV by culture, viral antigen or viral RNA in fetal tissue, umbilical cord blood, or amniotic fluid with a validated diagnostic test **OR**
- Detection of ZIKV by culture, viral antigen or viral RNA in neonatal serum, CSF, or urine collected within 2 days of birth** **OR**
- Positive ZIKV IgM antibody test in umbilical cord blood, neonatal serum or CSF collected within 2 days of birth** with positive ZIKV neutralizing antibody titers and negative neutralizing antibody titers against dengue or other flaviviruses endemic to the region where exposure occurred
**Zika infection, non-congenital**

| Confirmed: | An individual who does not meet clinical criteria for non-congenital Zika disease, BUT who meets confirmatory laboratory criteria. |
| Probable: | An individual who does not meet clinical criteria for non-congenital Zika disease, BUT who has an epidemiologic link* AND laboratory evidence of recent ZIKV or flavivirus infection by: |
| | - Positive ZIKV IgM antibody test of serum or CSF with: |
| | - positive neutralizing antibody titers against ZIKV and dengue or other flaviviruses endemic to the region where exposure occurred; OR |
| | - negative dengue virus IgM antibody test and no neutralizing antibody test performed |

*Epidemiologic link defined as one or more of the following:
- Resides in or recent travel to an area with known ZIKV transmission, OR
- Sexual contact with a confirmed or probable case of ZIKV infection or person with recent travel to an area with known ZIKV transmission; OR
- Receipt of blood or blood products within 120 days of diagnosis; OR
- Organ or tissue transplant recipient within 120 days of diagnosis; OR
- Association in time or place with a confirmed or probable case; OR

Likely vector exposure in an area with suitable seasonal and ecological conditions for potential local vector-borne transmission

**Note:** Zika IgM may be detectable between 1-12 weeks after infection but may persist for months to years and interpretation is complicated by cross-reactivity with other flaviviruses. In the absence of clinical illness, interpretation of Zika IgM and/or PRNT results is based on epidemiological context.

| Detection of ZIKV by culture, viral antigen or viral RNA in serum, CSF, tissue, or other specimen (e.g., amniotic fluid, urine, semen, saliva) with a validated diagnostic test, OR |
| Positive ZIKV IgM antibody test in serum or CSF with positive ZIKV neutralizing antibody titers and negative neutralizing antibody titers against dengue or other flaviviruses endemic to the region where exposure occurred. |