

**Outline**

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**Immediately report** any suspected or confirmed cases of tularemia to:

**SFDPH Communicable Disease Control  
(24/7 Tel: 415-554-2830)**

- By law, health care providers must report suspected or confirmed cases of tularemia to their local health department immediately [within 1 hr].
- SFDPH Communicable Disease Control can facilitate specialized testing and will initiate the public health response as needed.

**Also notify your:**

- Infection Control Professional
- Clinical Laboratory

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## INTRODUCTION

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Tularemia is a zoonotic disease caused by *Francisella tularensis*, a non-sporulating, non-motile, aerobic, gram-negative coccobacillus. There are multiple subspecies of *F. tularensis*, with the biovars *tularensis* (type A) and *holarctica* (type B) occurring most commonly in the United States. The clinical syndromes caused by tularemia depend on the route of infection and subspecies of the infecting organism. Tularemia is highly infectious, requiring inhalation or inoculation of as few as 10 to 50 organisms to cause disease.<sup>1</sup> Although its virulence factors are not well characterized, type A is generally thought to be the more virulent subspecies.<sup>2-4</sup> However, the virulence of type A subspecies may vary between geographic regions within the US, with the mid-western and eastern states having more severe infections.<sup>5, 6</sup>

The Working Group for Civilian Biodefense considers tularemia to be a dangerous potential biological weapon because of its "extreme infectivity, ease of dissemination, and its capacity to cause illness and death." Of the potential ways that *F. tularensis* could be used as a biological weapon, an aerosol release is expected to have the most severe medical and public health outcomes.<sup>3</sup>

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## EPIDEMIOLOGY

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### Tularemia as a Biological Weapon

Weaponized *F. tularensis* was developed and stockpiled by the US military, though the supply was destroyed in the 1970's. The Soviet Union is reported to have developed antibiotic- and vaccine-resistant strains of weaponized *F. tularensis*.<sup>3</sup>

Experts believe that an aerosolized release is the most likely intentional use of *F. tularensis* organisms. Exposure to aerosolized *F. tularensis* would cause:<sup>2</sup>

- Via inhalation:
  - primary pneumonic tularemia (majority of patients)
  - typhoidal tularemia (nonspecific febrile illness of varying severity)
  - oropharyngeal tularemia
- Via contact with eyes: oculoglandular tularemia
- Via contact with broken skin: glandular or ulceroglandular disease

**An intentional release of tularemia would have the following characteristics:**

- Multiple similarly presenting cases clustering in time:
  - acute non-specific febrile illness with onset 3 to 5 days after the initial release (range 1-14 days)
  - community-acquired atypical pneumonia unresponsive to typical antimicrobials
- Atypical host characteristics: unexpected, unexplained cases of acute illness in previously healthy persons who rapidly develop pleuropneumonia and systemic infection, especially if patients develop pleural effusions and hilar lymphadenopathy
- Unusual geographic clustering: multiple cases in an urban area, where naturally occurring tularemia is not endemic
- Absence of risk factors: patients lack tularemia exposure risk factors (e.g. outdoor field work or recreational activity, contact with tissues of potentially infected animals)

Intentionally released *F. tularensis* strains may be altered to have enhanced virulence or antimicrobial resistance.<sup>3</sup>

## **Naturally Occurring Tularemia**

### **Reservoir**

The natural reservoirs for *F. tularensis* are small and medium-sized mammals. In the United States these are primarily lagomorphs (rabbits, hares) but may include beaver, squirrels, muskrats, field voles, and rats. Incidental hosts include some species of mammals (e.g. humans, cats, dogs, cattle), birds, fish, and amphibians. Organisms can survive for weeks in moist environments, including water, mud, and decaying animal tissue.<sup>2</sup> There is some evidence that the protozoa *Acanthamoeba castellanii*, may be an important reservoir for *F. tularensis*.<sup>7</sup>

### **Mode of Transmission**

The primary vectors for infection in the United States are ticks (dog ticks, wood ticks) and flies, such as the deerfly. Humans become infected by a number of mechanisms:<sup>3</sup>

- bites by infected arthropods (majority of cases)
- contact with infectious animal tissues or fluids, during for example hunting or butchering
- ingestion of contaminated food, water, or soil
- inhalation of infectious aerosols, including aerosols generated during landscaping activities (e.g., lawn mowing, using a power blower, and brush cutting)

- exposure in the laboratory (accidental inhalation of aerosol, direct contact with an infectious specimen including accidental parenteral inoculation, or ingestion)

Tularemia is not spread from person to person.

### **Worldwide Occurrence**

Worldwide, human cases of tularemia occur throughout North America, Europe, and Asia. Infections with type A strain are generally only seen in North America. Within Europe and Asia, the greatest numbers of human cases are reported in Scandinavian countries and countries of the former Soviet Union.<sup>3</sup> Recent significant outbreaks of tularemia in humans include: Sweden (2000, 2003, 2006), Kosovo (2002), France (2004), Turkey (2004-2005), Bulgaria (1997-2005) and Spain (2007).<sup>2, 8-10</sup>

### **United States Occurrence**

Nationwide, incidence of tularemia has declined from approximately 2000 annually reported cases during the first half of the 20<sup>th</sup> century to an average of 124 cases per year during the 1990s. Most cases occur in rural or semirural environments, during the summer months, with the greatest number of cases occurring in Missouri, Oklahoma, South Dakota, Montana, and Martha's Vineyard, Massachusetts. From 1990 to 2000, incidence of tularemia in the United States was highest in children 5-9 years and adults 75 years and older. Regardless of age, males had a higher incidence of tularemia, potentially because of participation in activities more likely to cause exposures, such as hunting, trapping, butchering, and farming.<sup>11</sup>

Recent significant outbreaks include:

- In 1978 and 2000, outbreaks of the rare primary pneumonic tularemia occurred on Martha's Vineyard, Massachusetts. These represent the only outbreaks of primary pneumonic tularemia in the United States. Additional cases of tularemia have been reported each year in Martha's Vineyard (2000-2006). Exposure is most likely from breathing infectious aerosols generated during landscaping activities.<sup>12</sup> The reservoir in these outbreaks is still unclear, but may involve skunks and raccoons.<sup>13</sup>
- In 2002, tularemia was responsible for a die-off of several hundred prairie dogs caught in the wild in South Dakota and then commercially distributed widely throughout the USA. One human case occurred in an animal handler who cared for the infected animals.<sup>14</sup>
- In 2003, low levels of *F. tularensis* were identified in a biodetection air-monitoring system in Houston, Texas. No human cases occurred. An investigation supported contamination of the filters by naturally occurring *F. tularensis* organisms from an unidentified environmental reservoir.<sup>15</sup>

### **Occurrence in California and San Francisco**

From 2000 to 2007, 16 cases of plague were reported in California, and one of these occurred in San Francisco.<sup>16-19</sup>

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## **CLINICAL FEATURES**

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Human tularemia occurs in six recognized forms, determined primarily by route of infection. Tularemia infection can range from mild to severe clinical illness and can be life-threatening.

Overall case-fatality rates have declined from 5-15% in the pre-antibiotic era to approximately 2% currently. Mortality was historically much higher with pneumonic and typhoidal tularemia, with case-fatality as high as 30-60% if untreated.<sup>3</sup> Administration of appropriate antibiotic treatment typically leads to general symptom improvement within 24-48 hours. Recognition of tularemia as a potential etiologic agent is critical, because poor outcomes have been associated with delays in seeking care and/or instituting effective antimicrobial treatment.<sup>20</sup>

### **Pneumonic Tularemia**

Pneumonic tularemia causes the most severe disease, and presents as a non-specific febrile illness with progression to pleuropneumonitis and systemic infection.

| <b>PNEUMONIC TULAREMIA</b>   |   |
|--|---|
| <b>Incubation Period</b>   | 3-5 days (range 1-14 days)  |
| <b>Transmission</b>  | <ul style="list-style-type: none"> <li>• Inhalation of contaminated aerosols</li> <li>• Secondary hematogenous spread to the lung</li> </ul>  |
| <b>Signs and Symptoms</b>  | <ul style="list-style-type: none"> <li>• Initial presentation as atypical CAP unresponsive to routine antibiotic therapy, which can progress slowly OR rapidly to severe disease</li> <li>• Fever (abrupt onset), headache, cough, minimal or no sputum production, dyspnea, pleuritic chest pain, myalgias (often prominent in lower back), bronchiolitis and/or pharyngitis may be present</li> <li>• Generalized maculopapular rash with progression to pustules or erythema-nodosum type rash occurs in 20%</li> <li>• Nausea, vomiting, diarrhea is not uncommon</li> <li>• Hemoptysis (not common)</li> </ul> |
| <b>Progression and Complications</b>   | <ul style="list-style-type: none"> <li>• Respiratory failure, ARDS</li> <li>• Severe pneumonia</li> <li>• Lung abscess or cavitary lesions</li> <li>• Sepsis</li> </ul>   |
| <b>Laboratory Findings</b>   | <ul style="list-style-type: none"> <li>• Lobar, segmental, or subsegmental opacities on CXR, pleural effusion, pleural adhesions, Hilar adenopathy</li> <li>• Leukocytosis; differential may be normal</li> <li>• Liver enzymes and/or CK may be abnormal</li> <li>• Sputum gram stain usually nonspecific</li> </ul>   |
| ARDS, acute respiratory distress syndrome; CAP, community-acquired pneumonia; CK, creatine kinase; CXR, chest x-ray. |   |

### **Glandular and Ulceroglandular Tularemia**

Glandular and ulceroglandular tularemia account for the majority of naturally occurring cases of tularemia. In the *ulceroglandular* form, an ulcer is formed at the site of inoculation, with subsequent lymphadenopathy in the proximal draining lymph nodes. Occasionally, lymphadenopathy occurs without an ulcer, leading to the designation of *glandular* disease.

| <b>GLANDULAR AND ULCEROGLANDULAR TULAREMIA</b> |  |
|--|--|
| <b>Incubation Period</b>                       | 3-5 days (range 1-14 days)   |
| <b>Transmission</b>                            | <ul style="list-style-type: none"> <li>• Bite of an infective arthropod</li> <li>• Direct contact with infectious material (i.e., contaminated carcass, settled infectious aerosol)</li> </ul>   |
| <b>Signs and Symptoms</b>                      | <ul style="list-style-type: none"> <li>• Ulceroglandular form – local skin involvement at site of exposure that develops into a painful cutaneous papule with subsequent ulceration within several days. Papule becomes necrotic and scars.</li> <li>• Glandular form – no cutaneous lesion occurs</li> <li>• Enlarged and tender regional lymphadenopathy that can persist for months</li> <li>• Fever, chills, malaise, myalgias, arthralgias, headache, anorexia, GI symptoms are common</li> </ul> |
| <b>Progression and Complications</b>           | <ul style="list-style-type: none"> <li>• Lymph node suppuration</li> <li>• Secondary pneumonia</li> <li>• Hematogenous spread to other organs</li> <li>• Sepsis</li> </ul>   |
| <b>Laboratory Findings</b>                     | <ul style="list-style-type: none"> <li>• Leukocytosis; differential may be normal</li> <li>• Liver enzymes and/or CK may be abnormal</li> </ul>  |
| CK, creatine kinase; GI, gastrointestinal.     |  |

### **Oculoglandular Tularemia**

Oculoglandular tularemia results either from ocular inoculation from the hands after contact with contaminated material or from splashes or aerosols generated during handling of infective material (e.g., animal carcasses). This form of tularemia could occur in a bioterrorism setting as a result of an aerosol exposure. Organisms spread from the conjunctiva to regional nodes, where they cause focal necrosis and lesions.<sup>2-4</sup>

After an incubation period of 3-5 (range 1-14) days, oculoglandular tularemia presents as a painful “red eye” with purulent exudation, chemosis, vasculitis, and painful regional lymphadenopathy. Additional signs and symptoms may include photophobia, lacrimation, itching, local edema, and changes in visual acuity. There is a potential for lymph node suppuration, hematogenous dissemination, and development of sepsis.<sup>2-4</sup>

Laboratory values are generally nonspecific, and Gram stain of conjunctival scrapings may or may not demonstrate organisms.<sup>2</sup>

### **Oropharyngeal Tularemia**

Oropharyngeal or gastrointestinal tularemia occurs via ingestion of contaminated food including undercooked meat, contaminated water or droplets, and oral inoculation from the hands after contact with contaminated material.<sup>3, 4</sup>

After an incubation period of 3-5 (range 1-14) days, oropharyngeal tularemia presents either as acute pharyngitis with cervical lymphadenopathy or as ulcerative gastrointestinal lesions with fever, abdominal pain, diarrhea, nausea, vomiting, mesenteric lymphadenopathy, and gastrointestinal bleeding. Severity can range from mild diarrhea to overwhelming ulceration with frank gastrointestinal bleeding and sepsis. A large inoculum (approximately  $10^8$  organisms) is required to transmit disease orally. There is a potential for lymph node suppuration, hematogenous dissemination, and development of sepsis.<sup>2-4</sup> Routine tests are generally nonspecific. Leukocytosis may or may not be present.<sup>2</sup>

### **Typhoidal Tularemia**

Typhoidal (septicemic) tularemia is an acute, nonspecific febrile illness associated with *F. tularensis* without prominent lymphadenopathy.

| <b>TYPHOIDAL TULAREMIA</b>                 |   |
|--|---|
| <b>Incubation Period</b>                   | 3-5 days (range 1-14 days)  |
| <b>Transmission</b>                        | <ul style="list-style-type: none"> <li>• Site of primary infection usually unknown</li> </ul>   |
| <b>Signs and Symptoms</b>                  | <ul style="list-style-type: none"> <li>• Fever, chills, headache, malaise, weakness, myalgias, arthralgias, cough</li> <li>• Prostration, dehydration, hypotension, pharyngitis</li> <li>• Watery diarrhea, anorexia, nausea, vomiting, abdominal pain (children may have more severe GI involvement)</li> <li>• Generalized maculopapular rash with progression to pustules or erythema-nodosum type rash may occur</li> <li>• Splenomegaly and hepatomegaly (not common)</li> </ul> |
| <b>Progression and Complications</b>       | <ul style="list-style-type: none"> <li>• Secondary pneumonia</li> <li>• Hematogenous spread to other organs – osteomyelitis, pericarditis, peritonitis, endocarditis, meningitis</li> <li>• Sepsis</li> <li>• Rhabdomyolysis</li> <li>• Cholestasis with jaundice</li> <li>• Renal failure</li> <li>• Debilitating illness lasting several months</li> </ul>  |
| <b>Laboratory Findings</b>                 | <ul style="list-style-type: none"> <li>• Pleural effusions</li> <li>• Leukocytosis; differential may be normal</li> <li>• Liver enzymes and/or CK may be abnormal</li> <li>• Sterile pyuria may occur</li> </ul>  |
| CK, creatine kinase; GI, gastrointestinal. |   |

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## **DIFFERENTIAL DIAGNOSIS**

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A high index of suspicion is required to diagnose tularemia because there are no readily available rapid and specific confirmatory tests. In addition, the various forms of tularemia can have a nonspecific appearance and/or resemble a wide range of much more common illnesses.

### **Differential: Pneumonic Tularemia**

The following are clinical syndromes that can appear similar to the pneumonic form of tularemia:

- bacterial pneumonia (*Mycoplasma*, *Staphylococcus*, *Streptococcus*, *Haemophilus*, *Klebsiella*, *Moraxella*, *Legionella*)
- *Chlamydia* infection
- Q fever
- tuberculosis
- inhalational anthrax
- pneumonic plague
- fungal pulmonary disease (histoplasmosis, coccidioidomycosis)
- Viral pneumonia (influenza, hantavirus, RSV, CMV)
- severe acute respiratory syndrome (SARS)
- other causes of atypical or chronic pneumonias

### **Differential: Glandular and Ulceroglandular Tularemia**

The following are clinical syndromes that can appear similar to the glandular and ulceroglandular forms of tularemia:

- pyogenic bacterial infections
- cat-scratch disease (*Bartonella*)
- syphilis
- chancroid
- lymphogranuloma venereum
- tuberculosis
- nontuberculosis mycobacterial infection
- toxoplasmosis
- sporotrichosis
- rat-bite fever
- anthrax
- plague
- herpes simplex virus infection
- adenitis or cellulitis (*Staphylococcus* or *Streptococcus*)
- *Pasteurella* infections
- rickettsial infections
- orf virus infection

### **Differential: Oculoglandular Tularemia**

The following are clinical entities that can appear similar to the oculoglandular form of tularemia:

- pyogenic bacterial infections
- adenoviral infection
- syphilis
- cat-scratch disease
- herpes simplex virus infection
- varicella-Zoster virus infection
- sporotrichosis
- coccidioidomycosis
- tuberculosis

### **Differential: Oropharyngeal Tularemia**

The following are causes of syndromes that appear similar to the oropharyngeal form of tularemia:

- *Streptococcus* pharyngitis
- infectious mononucleosis
- adenoviral infection
- diphtheria
- GI anthrax

### **Differential: Typhoidal Tularemia**

The following are causes of syndromes that can appear similar to typhoidal forms of tularemia:

- *Salmonella* spp. infection
- brucellosis
- endocarditis
- leptospirosis

- *Legionella* infection
- *Chlamydia* infection
- Q fever
- disseminated mycobacterial or fungal infection
- rickettsial infections
- malaria
- meningococemia
- septicemic plague
- septicemia caused by other gram-negative bacteria
- *Staphylococcus* or *Streptococcus* toxic shock syndrome
- other causes of prolonged fever without localizing signs

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## LABORATORY DIAGNOSIS

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The diagnosis of tularemia requires a high index of suspicion because the disease often presents with nonspecific symptoms and nonspecific results of routine lab tests.

Although recommended, microscopy and culture are difficult and often not fruitful. The organism is rarely seen on stained clinical specimens and is difficult to isolate using routine culture media and conditions. However, isolation is possible from a variety of clinical specimens if culture conditions are optimized. Even still, some strains may require up to a week to develop visible colonies, especially if the patient has been placed on bacteriostatic antibiotic therapy. Because of the need for nonroutine laboratory methods and because *F. tularensis* is a risk to laboratory personnel, clinicians should notify the laboratory when tularemia is suspected.<sup>3, 21</sup>

**If you are testing or considering testing for tularemia, you should:**

- **IMMEDIATELY notify SFDPH Communicable Disease Control (24/7 Tel: 415-554-2830).** SFDPH can authorize and facilitate testing, and will initiate the public health response as needed.
- **Inform your lab that tularemia is under suspicion. *F. tularensis* may pose a risk to lab personnel.**

Diagnosis is most commonly confirmed by serologic testing. Antibody detection assays include tube agglutination, microagglutination, hemoagglutination, and enzyme-linked immunosorbent assay (ELISA). Significant antibodies appear around the end of the 2nd week of illness, peak at 4-5 weeks, and can persist indefinitely. A single titer of 1:160 or greater (by tube agglutination) or 1:128 or greater (by microagglutination) is a presumptive positive; a four-fold rise in titer is required for definitive serologic diagnosis.<sup>3, 21</sup>

Although rapid diagnostic tests are not widely available, the public health laboratory system may be able to provide timely testing of certain clinical specimens (e.g., polymerase chain reaction (PCR) testing).

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## TREATMENT AND PROPHYLAXIS

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These recommendations are current as of this document date. SFDPH will provide periodic updates as needed and situational guidance in response to events ([www.sfcddcp.org](http://www.sfcddcp.org)).



## Treatment

First-line treatment for tularemia is streptomycin or gentamicin.<sup>2, 20, 22, 23</sup> Flouroquinolone antibiotics have also been effective<sup>24, 25</sup>. Other alternatives are tetracyclines and chloramphenicol. However these drugs are bacteriostatic and their uses have produced more relapses than treatment with aminoglycosides.<sup>20, 26</sup> Clinicians should be aware that *F. tularensis* strains released intentionally may be resistant to antimicrobials.<sup>3</sup>

Supportive care, including fluid management and hemodynamic monitoring, should be considered in all patients. Intensive care with respiratory support may be necessary in patients with complications.<sup>2</sup>

**Contained casualty setting:** The Working Group recommends parenteral antimicrobial therapy when individual medical management is available (**Table 1**). Therapy may be switched to oral antimicrobials when clinically indicated.<sup>3</sup>

**Mass casualty setting:** Use of oral antibiotics may be necessary if the number of patients exceeds the medical care capacity for individual medical management (**Table 2**).<sup>3</sup>

| <b>TABLE 1. TREATMENT OF TULAREMIA IN THE CONTAINED CASUALTY SETTING<sup>3</sup></b>   |  |
|--|--|
| <b>Patient Category</b>  | <b>Therapy Recommendation*</b>   |
| <b>Adults:<br/>Preferred Choices</b>   | Streptomycin, 1 gm IM BID for 10 days†‡§ <b>OR</b><br>Gentamicin, 5 mg/kg IM or IV QD for 10 days†‡  |
| <b>Adults:<br/>Alternative Choices</b>   | Doxycycline, 100 mg IV BID for 14-21 days† <b>OR</b><br>Chloramphenicol, 15 mg/kg IV QID for 14-21 days** <b>OR</b><br>Ciprofloxacin, 400 mg IV BID for 10 days†   |
| <b>Children:<br/>Preferred Choices</b>   | Streptomycin, 15 mg/kg IM BID (max 2 gm/day) for 10 days‡ <b>OR</b><br>Gentamicin, 2.5 mg/kg IM or IV TID for 10 days‡   |
| <b>Children:<br/>Alternative Choices</b>   | Doxycycline,<br>>45 kg, give adult dosage for 14-21 days<br><45 kg, give 2.2 mg/kg IV BID for 14-21 days <b>OR</b><br>Chloramphenicol, 15 mg/kg IV QID for 14-21 days** <b>OR</b><br>Ciprofloxacin, 15 mg/kg IV BID (max 1 gm/day) for 10 days |
| <p>* These treatment recommendations reflect those of the Working Group on Civilian Biodefense and may not necessarily be approved by the Food and Drug Administration.</p> <p>† Acceptable for pregnant women.</p> <p>§ Streptomycin is not as acceptable as gentamicin for use in pregnant women because irreversible deafness in children exposed in utero has been reported with streptomycin use.</p> <p>‡ Aminoglycosides must be adjusted according to renal function.</p> <p>** Concentration should be maintained between 5 and 20 µg/mL; concentrations &gt;25 µg/mL can cause reversible bone marrow suppression.</p> |  |

**TABLE 2. TREATMENT OF TULAREMIA IN THE MASS CASUALTY SETTING AND FOR POSTEXPOSURE PROPHYLAXIS\*<sup>3</sup>**

| Patient Category                  | Therapy Recommendation*   |
|-----------------------------------|---|
| Adults (Including Pregnant Women) | Doxycycline, 100 mg PO BID for 14 days <sup>‡</sup> <b>OR</b><br>Ciprofloxacin, 500 mg PO BID for 14 days <sup>‡</sup>  |
| Children                          | Doxycycline,<br>>45 kg, give adult dosage for 14 days<br><45 kg, give 2.2 mg/kg PO BID for 14 days <b>OR</b><br>Ciprofloxacin, 15 mg/kg PO BID (max 1 gm/day) for 10 days |

\* These treatment recommendations reflect those of the Working Group on Civilian Biodefense and may not necessarily be approved by the Food and Drug Administration.

<sup>‡</sup> Although fetal toxicity may occur with doxycycline use, the Working Group recommended doxycycline or ciprofloxacin for postexposure prophylaxis of pregnant women or for treatment of infection of pregnant women in the mass casualty setting.

### **Postexposure Prophylaxis**

Antibiotic prophylaxis should begin as soon as possible and preferably within 24 hours after exposure to an infectious aerosol containing *F. tularensis* (**Table 2**). Postexposure prophylactic antibiotic treatment of close contacts of tularemia patients is not recommended because human to human transmission of *F. tularensis* is not known to occur.<sup>3</sup>

### **Vaccination**

A live, attenuated vaccine was developed and has been used in the United States to protect laboratory personnel who work with *F. tularensis*. This vaccine is currently under review by the Food and Drug Administration and is unavailable.<sup>3</sup> Clinical trials to develop a new tularemia vaccine are underway but it is not likely that a vaccine will be widely available in the near future.

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## **COMPLICATIONS AND ADMISSION CRITERIA**

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Disease manifestations and complications are typically related to the portal of entry of *F. tularensis*. Pneumonic tularemia may result in severe pneumonia, lung abscess, or acute respiratory distress syndrome (ARDS). Glandular and ulceroglandular tularemia may progress to lymph node suppuration and secondary pneumonia. Oculoglandular tularemia can cause localized lymph node suppuration, whereas oropharyngeal tularemia has been associated with mesenteric lymphadenitis, gastrointestinal (GI) ulceration, and GI bleeding. Typhoidal tularemia not uncommonly progresses to secondary pneumonia. All forms of human tularemia carry the potential for hematogenous dissemination of the organism to other organs such as bone, pericardium, and peritoneum, and for progression to sepsis and multiorgan failure.<sup>2-4</sup>

Admission to the hospital is generally advisable for patients with any form of tularemia, in order to administer antibiotics intravenously and to monitor for disease progression.

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## INFECTION CONTROL

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These recommendations are current as of this document date. SFDPH will provide periodic updates as needed and situational guidance in response to events ([www.sfcddcp.org](http://www.sfcddcp.org)).

Clinicians should notify local public health authorities, their institution's infection control professional, and their laboratory of any suspected tularemia cases. Public health authorities may conduct epidemiologic investigations and implement disease control interventions to protect the public. Both the Hospital Infection Control Practices Advisory Committee (HICPAC) of the CDC and the Working Group for Civilian Biodefense recommend **Standard Precautions** for patients with tularemia in a hospital setting without the need for isolation. Routine laboratory procedures should be carried out under Biosafety Level 2 (BSL-2) conditions; however, manipulation of cultures or other activities that may produce aerosol or droplets (e.g., centrifuging, grinding, vigorous shaking) require BSL-3 conditions.<sup>3</sup>

### **Decontamination**

Contaminated surfaces can be disinfected with commercially available bleach or a 1:10 dilution of household bleach and water. All persons exposed to an aerosol containing *F. tularensis* should be instructed to wash body surfaces and clothing with soap and water.<sup>3</sup>

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## PEARLS AND PITFALLS

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1. The onset of tularemia is usually abrupt, with fever, headache, chills and rigors, generalized body aches, and coryza. A pulse-temperature dissociation has been noted in as many as 42% of patients.<sup>3</sup>
2. Clinicians should familiarize themselves with the local epidemiology of tularemia. The occurrence of human cases may follow a local tularemia epizootic (outbreak of disease in an animal population). Occurrence of pneumonic tularemia in a low-incidence area should prompt consideration of bioterrorism.<sup>3</sup>
3. The diagnosis of tularemia relies heavily on clinical suspicion. Routine laboratory tests are usually nonspecific. The organism is usually not apparent on gram-stained smears or tissue biopsies and usually does not grow on standard culture plates. However, *F. tularensis* may be recovered from blood and body fluids using special supportive media. Because of this and its potential hazards to laboratory personnel, the laboratory should be notified if tularemia is suspected.

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