

Public Health Update

Saturday, March 05, 2005

Tularemia

Note: The following material is excerpted and edited from materials provided by CDC.

There were at least two cases of Tularemia within the NPS system last year.

Tularemia is a potentially serious illness that occurs naturally in the United States. It is caused by the bacterium *Francisella tularensis* found in animals (especially rodents, rabbits, and hares).



The causative agent of tularemia is one of the most infectious pathogenic bacteria known, requiring inoculation or inhalation of as few as 10 organisms to cause disease.

Symptoms of tularemia could include:

- sudden fever
- chills
- headaches
- diarrhea
- muscle aches
- joint pain
- dry cough
- progressive weakness

People can also catch pneumonia and develop chest pain, bloody sputum and can have trouble breathing and even sometimes stop breathing.

Other symptoms of tularemia depend on how a person was exposed to the tularemia bacteria. These symptoms can include ulcers on the skin or mouth, swollen and painful lymph glands, swollen and painful eyes, and a sore throat.



How Does Tularemia Spread?

People can get tularemia many different ways:

- being bitten by an infected tick, deerfly or other insect
- handling infected animal carcasses
- eating or drinking contaminated food or water
- breathing in the bacteria, *F. tularensis*

Tularemia is not known to be spread from person to person. People who have tularemia do not need to be isolated. People who have been exposed to the tularemia bacteria should be treated as soon as possible. The disease can be fatal if it is not treated with the right antibiotics.

How Soon Do Infected People Get Sick?

Symptoms usually appear 3 to 5 days after exposure to the bacteria, but can take as long as 14 days.

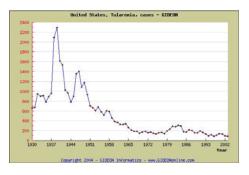
Geographic Distribution and Human Exposures

Tularemia occurs throughout much of North America and Eurasia. In the United States, human cases have been reported from every state except Hawaii; however, most cases occur in south-central and western states (especially Missouri, Arkansas, Oklahoma, South Dakota, and Montana). In Eurasia, the disease is also widely endemic, although the greatest numbers of human cases are reported from northern and central Europe, especially Scandinavian countries and those of the former Soviet Union. Tularemia is almost entirely a rural disease, although urban and suburban exposures occasionally do occur.

Throughout its range, *F tularensis* is found in widely diverse animal hosts and habitats and can be recovered from contaminated water, soil, and vegetation. A variety of small mammals, including voles, mice, water rats, squirrels, rabbits, and hares, are natural reservoirs of infection. They acquire infection through bites by ticks, flies, and mosquitoes, and by contact with contaminated environments. Although enzootic cycles of *F tularensis* typically occur without notice, epizootics with sometimes extensive die- offs of animal hosts may herald outbreaks of tularemia in humans.

Incidence

The worldwide incidence of tularemia is not known, and the disease is probably greatly underrecognized and underreported. In the United States, reported cases have dropped sharply from several thousand per year prior to 1950 to less than 200 per year in the 1990s. Between 1985 and 1992, 1409 cases and 20 deaths were reported in the United States, for a mean of 171 cases per year.



Persons in all age groups were affected, but most were children younger than 10 years and adults aged 50 years or older.

Most cases occur in June through September, when arthropod-borne transmission is most common. Cases in winter usually occur among hunters and trappers who handle infected animal carcasses. In the United States, cases are mostly sporadic or occur in small clusters; in Eurasia, waterborne, arthropod-borne, and airborne outbreaks involving hundreds of persons have been reported.

Prior to the advent of antibiotics, the overall mortality from infections with the more severe type A strains was in the range of 5% to 15%, and fatality rates as high as 30% to 60% were reported for untreated pneumonic and severe systemic forms of disease.^{74, 78} Currently, the overall casefatality rate of reported cases in the United States is less than 2%.^{34, 49} Type B infections are rarely fatal.

In ulceroglandular tularemia, the form that arises from handling a typically contaminated carcass or following an infective arthropod bite, a local cutaneous papule appears at the inoculation site at about the time of onset of generalized symptoms, becomes pustular, and ulcerates within a few days of its first appearance. The ulcer is tender, generally has an indolent character, and may be covered by an eschar. Typically, one or more regional afferent lymph nodes may become enlarged and tender within several days of the appearance of the papule. Even with antibiotic treatment, the affected nodes may become fluctuant and rupture. In oculoglandular tularemia, which follows direct contamination of the eye, ulceration occurs on the conjunctiva, accompanied by pronounced chemosis, vasculitis, and lymphadenitis. Glandular regional tularemia is characterized lymphadenopathy without an ulcer.

What Can I Do To Prevent Becoming Infected with Tularemia?

Tularemia occurs naturally in many parts of the United States. Use insect repellent containing DEET on your skin, or treat clothing with repellent containing permethrin, to prevent insect bites. Wash your hands often, using soap and warm water, especially after handling animal carcasses. Be sure to cook your food thoroughly and that your water is from a safe source.

Note any change in the behavior of your pets (especially rodents, rabbits, and hares)

or livestock, and consult a veterinarian if they develop unusual symptoms.

Avian Influenza

Note: The following information is excerpted and edited from material provided by the World Health Organization



The disease in birds: impact and control measures

Avian influenza is an infectious disease of birds caused by type A strains of the influenza virus. The disease, which was first identified in Italy more than 100 years ago, occurs worldwide.

All birds are thought to be susceptible to infection with avian influenza, though some species are more resistant to infection than others. Infection causes a wide spectrum of symptoms in birds, ranging from mild illness to a highly contagious and rapidly fatal disease resulting in severe epidemics. The latter is known as "highly pathogenic avian influenza". This form is characterized by sudden onset, severe illness, and rapid death, with a mortality that can approach 100%.

Fifteen subtypes of influenza virus are known to infect birds, thus providing an extensive reservoir of influenza viruses potentially circulating in bird populations. To date, all outbreaks of the highly pathogenic form have been caused by influenza A viruses of subtypes H₅ and H₇.

Migratory waterfowl – most notably wild ducks – are the natural reservoir of avian influenza viruses, and these birds are also the most resistant to infection. Domestic poultry, including chickens and turkeys, are particularly susceptible to epidemics of rapidly fatal influenza.

Direct or indirect contact of domestic flocks with wild migratory waterfowl has been implicated as a frequent cause of epidemics. Live bird markets have also played an important role in the spread of epidemics.

The quarantining of infected farms and destruction of infected or potentially exposed flocks are standard control measures aimed at preventing spread to other farms and eventual establishment of the virus in a country's poultry population. Apart from being highly contagious, avian influenza viruses are readily transmitted from farm to farm by mechanical means, such as by contaminated equipment, vehicles, feed, cages, or clothing. Highly pathogenic viruses can survive for long periods in the environment, especially when temperatures are low. Stringent sanitary measures on farms can, however, confer some degree of protection.



A constantly mutating virus: two consequences

Influenza viruses lack mechanisms for the "proofreading" and repair of errors that occur during replication. As a result of these uncorrected errors, the genetic composition of the viruses changes as they replicate in humans and animals, and the existing strain is replaced with a new antigenic variant. These constant, permanent and usually small changes in the antigenic composition of influenza A viruses are known as antigenic "drift".

The tendency of influenza viruses to undergo frequent and permanent antigenic changes necessitates constant monitoring of the global influenza situation and annual

adjustments in the composition of influenza vaccines.

Influenza viruses have a second characteristic of great public health concern: influenza A viruses, including subtypes from different species, can swap or "reassort" genetic materials and merge. This reassortment process, known as antigenic "shift", results in a novel subtype different from both parent viruses. As populations will have no immunity to the new subtype, and as no existing vaccines can confer protection, antigenic shift has historically resulted in highly lethal pandemics. For this to happen, the novel subtype needs to have genes from human influenza viruses that make it readily transmissible from person to person for a sustainable period.

Conditions favourable for the emergence of antigenic shift have long been thought to involve humans living in close proximity to domestic poultry and pigs. Because pigs are susceptible to infection with both avian and mammalian viruses, including human strains, they can serve as a "mixing vessel" for the scrambling of genetic material from human and avian viruses, resulting in the emergence of a novel subtype. Recent events, however, have identified a second possible mechanism. Evidence is mounting that, for at least some of the 15 avian influenza virus subtypes circulating in bird populations, humans themselves can serve as the "mixing vessel".

Human infection with avian influenza viruses: a timeline

Avian influenza viruses do not normally infect species other than birds and pigs. The first documented infection of humans with an avian influenza virus occurred in Hong Kong in 1997, when the H5N1 strain caused severe respiratory disease in 18 humans, of whom 6 died. The infection of humans coincided with an epidemic of highly pathogenic avian influenza, caused by the same strain, in Hong Kong's poultry population.

Extensive investigation of that outbreak determined that close contact with live infected poultry was the source of human infection. Studies at the genetic level further determined that the virus had jumped directly from birds to humans. Limited transmission to health care workers occurred, but did not cause severe disease.

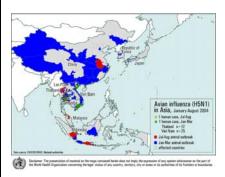
Rapid destruction – within three days – of Hong Kong's entire poultry population, estimated at around 1.5 million birds, reduced opportunities for further direct transmission to humans, and may have averted a pandemic.

That event alarmed public health authorities, as it marked the first time that an avian influenza virus was transmitted directly to humans and caused severe illness with high mortality. Alarm mounted again in February 2003, when an outbreak of H5N1 avian influenza in Hong Kong caused 2 cases and 1 death in members of a family who had recently travelled to southern China. Another child in the family died during that visit, but the cause of death is not known.

Two other avian influenza viruses have recently caused illness in humans. An outbreak of highly pathogenic H7N7 avian influenza, which began in the Netherlands in February 2003, caused the death of one veterinarian two months later, and mild illness in 83 other humans. Mild cases of avian influenza H9N2 in children occurred in Hong Kong in 1999 (two cases) and in mid-December 2003 (one case). H9N2 is not highly pathogenic in birds.

The most recent cause for alarm occurred in January 2004, when laboratory tests confirmed the presence of H5Ni avian influenza virus in human cases of severe respiratory disease in the northern part of Viet Nam.

Why H5N1 is of particular concern



Of the 15 avian influenza virus subtypes, H₅N₁ is of particular concern for several reasons. H₅N₁ mutates rapidly and has a documented propensity to acquire genes from viruses infecting other animal species. Its ability to cause severe disease in humans has now been documented on two occasions. In addition, laboratory studies have demonstrated that isolates from this virus have a high pathogenicity and can cause severe disease in humans. Birds that survive infection excrete virus for at least 10 days, orally and in faeces, thus facilitating

further spread at live poultry markets and by migratory birds.

The epidemic of highly pathogenic avian influenza caused by H5N1, which began in mid-December 2003 in the Republic of Korea and is now being seen in other Asian countries, is therefore of particular public health concern. H5N1 variants demonstrated a capacity to directly infect humans in 1997, and have done so again in Viet Nam in January 2004. The spread of infection in birds increases the opportunities for direct infection of humans. If more humans become infected over time, the likelihood also increases that humans, if concurrently infected with human and avian influenza strains, could serve as the "mixing vessel" for the emergence of a novel subtype with sufficient human genes to be easily transmitted from person to person. Such an event would mark the start of an influenza pandemic.

Influenza pandemics: can they be averted?

Based on historical patterns, influenza pandemics can be expected to occur, on average, three to four times each century when new virus subtypes emerge and are readily transmitted from person to person. However, the occurrence of influenza pandemics is unpredictable. In the 20th century, the great influenza pandemic of 1918–1919, which caused an estimated 40 to 50 million deaths worldwide, was followed by pandemics in 1957–1958 and 1968–1969.

Experts agree that another influenza pandemic is inevitable and possibly imminent.

Most influenza experts also agree that the prompt culling of Hong Kong's entire poultry population in 1997 probably averted a pandemic.

Several measures can help minimize the global public health risks that could arise from large outbreaks of highly pathogenic H5N1 avian influenza in birds. An immediate priority is to halt further spread of epidemics in poultry populations. This strategy works to reduce opportunities for human exposure to the virus. Vaccination of persons at high risk of exposure to infected poultry, using existing vaccines effective against currently circulating human influenza strains, can reduce the likelihood of co-infection of humans with avian and influenza strains, and thus reduce the risk that genes will be exchanged.

Workers involved in the culling of poultry flocks must be protected, by proper clothing and equipment, against infection. These workers should also receive antiviral drugs as a prophylactic measure.

When cases of avian influenza in humans occur, information on the extent of influenza infection in animals as well as humans and on circulating influenza viruses is urgently needed to aid the assessment of risks to public health and to guide the best protective measures. Thorough investigation of each case is also essential. While WHO and the members of its global influenza network, together with other international agencies, can assist with many of these activities, the successful containment of public health risks also depends on the epidemiological and laboratory capacity of affected countries and the adequacy of surveillance systems already in place.

Cumulative Number of Confirmed Human Cases of Avian Influenza A/(H5N1) since 28 January 2004

2 February 2005

Country/ Territory	Total cases	Deaths
Cambodia	1	1
Thailand	17	12
Viet Nam	37	29
Total	55	42

Notes

Total number of cases includes number of deaths. WHO reports only laboratoryconfirmed cases.

A Variety of Contributions: Highlighting the Work of Public Health Service Officers Assigned to NPS

CAPT Alan Schroeder, Assigned to the Facilities Management Program (361) 949-8173 ext. 234

CAPT Schroeder is responsible for the NPS Service-Wide Hazardous Waste Operations Ist Responder and Weapons of Mass Destruction Trainer/Course.

On a FY basis, he instructs (12-15) 24- Hour Ist Responder Operations Training Courses; (1-2) 40- Hour Site Technician Training Course; and either instructs or coordinates (15-20) 8- Hour Hazardous Waste Operations Annual Refresher

Training Courses to ensure that NPS personnel do not loose their professional credentials at either the 24-Hour or 40-Hour OSHA/NFPA certification levels.

To date, this training program has resulted in over 1600 NPS trained and certified Spill/Terrorism Incident Intervention Personnel.



The training sessions outlined above ensure NPS compliance with OSHA 29 CFR 1910.120 (Hazardous Waste Operations and Emergency Response (HAZWOPER) Regulations and NFPA 472 (Professional Competencies for Ist This training when Responders). completed by NPS Law Enforcement, Maintenance, Park Police, Wildland Fire & Natural Resources Personnel allows them to respond to small spills of known chemicals/fuels; perform contaminated site work; or respond to larger chemical or fuel spills as part of a coordinated Hazardous Materials Spill Response Team. Weapons of Mass Destruction (WMD) training component is designed for LE Rangers, Park Police, Emergency Medical and Public Works Personnel who are mandated to have a minimum of 8- Hours of WMD training to meet Homeland Security and Department of Justice Training requirements as outlined in HSPD5 former (Patroit Act).

CAPT Schroeder also developed NPS training courses on the Incident Command System and Clandestine Drug Lab Hazards. He is currently certified as an OSHA, FEMA, NIMS, Homeland Security, and Department of Justice Trainer for all of the course that are presented at National Parks.

Secondary duties include Fueling System Audits of National Parks that may spill fuel during water transport and Service-Wide PMIS Project Review for Park & Regional Hazardous Waste Management Funding Requests.

Regional Public Health Consultants Northeast

CAPT Barry Hartfield (978) 970-5033

National Capital CAPT Richard Durrett (202)619-7070 Southeast CDR Brian Cagle (404) 562-3124 ext 549

Midwest CDR Robert Reiss (402) 221- 3786

Intermountain CAPT John Collins (303) 969- 2922 CAPT Joe Winkelmaier (505) 988- 6040 LT George Larsen (307) 344- 2273 LTJG Adam Kramer (929) 226- 0168

Pacific West CDR Paul Robinson (510) 817-1375 CDR John Leffel (206) 220-4270

Alaska CDR John Leffel (206) 220- 4270

WASO Staff

Director, Office of Public Health CAPT Chuck Higgins (202) 513-7217

Deputy Director, Office of Public Health LCDR Jason Thomas (202) 513-7226

Program Analyst Sonya Coakley (202) 513-7215

Individual Park Public Health Staff GATE LT Craig Ungerecht (718) 354-4693 SEKI Paul Schwarz (559) 565-3144 LAME J. Shannon Swann (702) 293-8985 YOSE Bernice Dommer (209) 379-1033





In Partnership for nearly 100 years, the National Park Service and the United States Public Health Service have worked together to protect the health of visitors in Americas Parks!