

Viruses and Diseases

Tetanus is a medical term indicating a prolonged contraction of striated muscle. If the condition is caused by exposure to certain bacteria, a serious and often fatal [disease](#) may result. Tetanus is the primary symptom caused by the [neurotoxin tetanospasmin](#) which is produced by the [Gram-positive](#), obligate [anaerobic bacterium *Clostridium tetani*](#). Infection usually originates from a contaminated wound, often a cut or deep puncture wound. Common symptoms are muscle spasms in the jaw (hence the common name **lockjaw**), followed by difficulty swallowing and general muscle stiffness in other parts of the body. Infection can be prevented by proper immunization, as well as by post-exposure [prophylaxis](#). [Tetany](#) refers to a state of muscle tension

Encephalomyelitis is a general term for [inflammation](#) of the [brain](#) and [spinal cord](#), describing a number of disorders:

- [acute disseminated encephalomyelitis](#) or *postinfectious encephalomyelitis*, a [demyelinating disease](#) of the brain and spinal cord, possibly triggered by vaccination or [viral](#) infection;
- *encephalomyelitis disseminata*, a synonym for [multiple sclerosis](#);
- *equine encephalomyelitis*, a potentially fatal [mosquito](#)-borne viral disease that infects [horses](#) and humans;
- [myalgic encephalomyelitis](#), a syndrome involving inflammation of the central nervous system with symptoms of muscle pain and fatigue; the term has sometimes been used interchangeably with [chronic fatigue syndrome](#), though there is still controversy over the distinction.
- [experimental autoimmune encephalomyelitis](#) (EAE), an animal model of brain inflammation.

Horse flu (or **Equine influenza**) refers to varieties of [Influenzavirus A](#) that are [endemic](#) in [horses](#). Horse flu viruses were only isolated in [1956](#). There are two main types of virus called equine-1 ([H7N7](#)) which commonly affects horse heart muscle and equine-2 ([H3N8](#)) which is usually more severe. Horse flu is endemic throughout the world.

The disease has a nearly 100% infection rate in an unvaccinated horse population that has not been previously exposed to the virus. The incubation time is one to five days.

Horses with horse flu can run a fever, have a dry hacking cough, have a runny nose, and become depressed and reluctant to eat or drink for several days but usually recover in 2 to 3 weeks.

"Vaccination schedules generally require a primary course of 2 doses, 3-6 weeks apart, followed by boosters at 6-12 month intervals. It is generally recognised that in many cases such schedules may not maintain protective levels of antibody and more frequent administration is advised in high-risk situations." ^[1]

Equine herpesvirus 4 (EHV-4) is a [virus](#) of the family *Herpesviridae* that cause [rhinopneumonitis](#) in [horses](#). It is the most important viral cause of respiratory infection in foals.^[1] Like other herpes viruses, EHV-4 causes a lifelong latent infection in affected animals. These horses are usually the source for new infection for foals over two months old, weanlings, and yearlings. Symptoms include fever, loss of appetite, and discharge from the nose. Most infected animals recover in one to three weeks,^[2] but death can occur in environments with overcrowding and other stress factors. There is a vaccine available.

Strangles is a [contagious, upper respiratory tract infection](#) of horses and other [equines](#) caused by a [bacterium](#), *Streptococcus equi*. Strangles is [endemic](#) in domesticated [horse](#) populations worldwide.

Clinical symptoms are characterised by [fever](#), [nasal](#) discharge or pus, and swollen or enlarged [mandibular lymph nodes](#) - around the neck and face. Affected animals may also stop eating and have a dull affect.

Possible complications include the horse possibly becoming a chronic carrier of the disease, asphyxia due to enlarged lymph nodes compressing the larynx or windpipe, bastard strangles (spreading to other areas of the body), pneumonia, guttural pouches filled with pus, purpura hemorrhagica, and heart disease. The average length for the course of this disease is 23 days.

The disease is spread when the nasal discharge or material from the draining abscess contaminates pastures, barns, feed troughs, etc. Isolation of new horses for 4 to 6 weeks, immediate isolation of infected horses, and disinfection of stalls, water buckets, feed troughs, and other equipment will help prevent the spread of strangles.

Equines of any age may contract the disease, although younger and elderly equines are more susceptible, owing to a weaker immune system.

Vaccinations are available. The initial vaccination is followed by a booster in three weeks and a third booster in six weeks from the initial vaccine. Annual re-vaccination is given thereafter.

[Mortality](#) is expected to be up to 1 in every 10 horses affected with strangles. The disease is very contagious and [morbidity](#) is high. Precautions to limit the spread of the illness are necessary and those affected are normally isolated. An isolation period of 6 weeks is usually necessary to ensure that the disease is not still incubating before ending the isolation.

[\[edit\]](#)

Treatment

As with many streptococcal infections, [penicillin](#) or penicillin-derivative antibiotics are the most effective treatments.

Rabies (from a Latin word meaning *rage*) is a [viral](#) disease that causes acute [encephalitis](#) in [animals](#) and people. It can affect most species of warm-blooded animals, but is rare among herbivores. In unvaccinated humans, rabies is almost invariably fatal once full-blown symptoms have developed, but prompt post-exposure [vaccination](#) usually prevents symptoms from developing.

The stereotypical image of an infected ("rabid") animal is a [dog](#) foaming at the mouth; however [cats](#), [ferrets](#), [raccoons](#), [skunks](#), [foxes](#), [wolves](#), [coyotes](#), [bears](#), and [bats](#) can also become rabid. [Squirrels](#), [chipmunks](#), other [rodents](#) (except [beavers](#)) and [rabbits](#) are very seldom infected, perhaps because they would not usually survive an attack by a rabid animal. Rabies may also be present in a so-called "paralytic" form, rendering the infected animal unnaturally quiet and withdrawn.

The virus is usually present in the [saliva](#) of a symptomatic rabid animal; the route of infection is nearly always by a bite. By causing the infected animal to be exceptionally aggressive, the virus ensures its transmission to the next host. Transmission has occurred via an [aerosol](#) through mucous membranes; transmission in this form may have happened in people exploring caves populated by rabid bats. Transmission from person to person is extremely rare, though it can happen through [transplant surgery](#) (see below for recent cases), or even more rarely through bites or kisses.

After a typical human infection by animal bite, the virus directly or indirectly enters the [peripheral nervous system](#). It then travels along the [nerves](#) towards the [central nervous system](#). During this phase, the virus cannot be easily detected within the host, and vaccination may still confer cell-mediated immunity to pre-empt symptomatic rabies. Once the virus reaches the [brain](#), it rapidly causes [encephalitis](#) and symptoms appear. It may also inflame the spinal cord producing [myelitis](#).

The period between infection and the first [flu](#)-like symptoms is normally 3–12 weeks, but can be as long as two years. Soon after, the symptoms expand to [cerebral dysfunction](#), [anxiety](#), [insomnia](#), [confusion](#), [agitation](#), abnormal behaviour, [hallucinations](#), progressing to [delirium](#). The production of large quantities of [saliva](#) and tears coupled with an inability to speak or swallow are typical during the later stages of the disease; this can result in "[hydrophobia](#)". Death almost invariably results 2–10 days after the first symptoms; the handful of people who are known to have survived the disease were all left with severe brain damage, with the recent exception of [Jeanna Giese](#) (see below).

[\[edit\]](#)

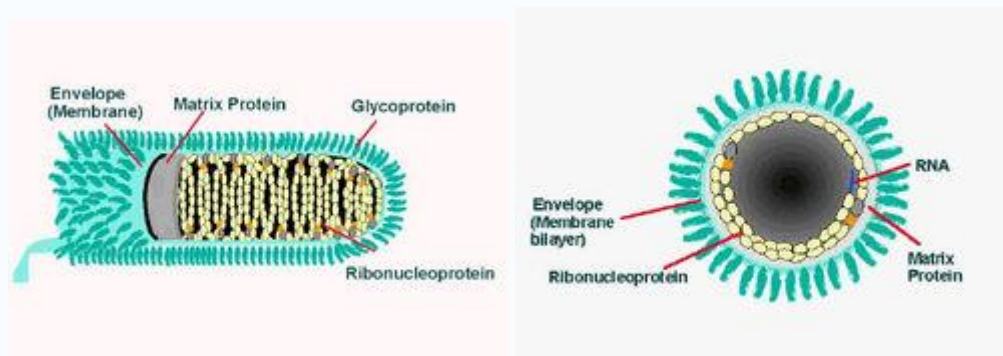
The virus

The rabies virus is a [Lyssavirus](#). This genus of [RNA viruses](#) also includes the [Aravan virus](#), [Australian bat lyssavirus](#), [Duvenhage virus](#), [European bat lyssavirus 1](#), [European bat lyssavirus 2](#), [Irkut virus](#), [Khujand virus](#), [Lagos bat virus](#), [Mokola virus](#) and [West Caucasian bat virus](#). Lyssaviruses have [helical](#) symmetry, so their infectious particles are approximately cylindrical in shape. This is typical of plant-infecting viruses; human-

infecting viruses more commonly have cubic symmetry and take shapes approximating [regular polyhedra](#).

[Biopsy](#) shows typical [Negri bodies](#) in the infected neurons.

The virus has a bullet-like shape with a length of about [180 nm](#) and a cross-sectional diameter of about [75 nm](#). One end is rounded or conical and the other end is planar or concave. The lipoprotein envelope carries knob like spikes, composed of Glycoprotein G. Spikes do not cover the planar end of the virion. Beneath the envelope is the membrane or matrix (M) protein layer which may be invaginated at the planar end. The core of the Virion consists of helically arranged ribonucleoprotein. The genome is unsegmented linear negative sense RNA. Also present in the nucleocapsid are RNA dependent RNA transcriptase and some structural proteins.



Longitudinal and cross-sectional schematic view of Rabies virus

[\[edit\]](#)

Prevention

There is no known cure for symptomatic rabies, but it can be prevented by [vaccination](#), both in humans and other animals. Virtually every infection with rabies was historically a death sentence, until [Louis Pasteur](#) developed the first rabies vaccination in [1885](#). This vaccine was first used on 9-year old [Joseph Meister](#), on [July 6, 1885](#), after the boy was badly mauled by a rabid dog.

Pasteur's vaccine consisted of a sample of the virus harvested from infected (and necessarily dead) rabbits, which was weakened by allowing it to dry. Similar nerve tissue-derived vaccines are still used today in developing countries, and while they are much cheaper than modern cell culture vaccines, they are not as effective and carry a certain risk of neurological complications.

The human diploid cell rabies vaccine (HDCV) was started in 1967. Human diploid cell rabies vaccines are made using the attenuated Pitman-Moore L503 strain of the virus. Human diploid cell rabies vaccines have been given to more than 1.5 million people worldwide [as of 2006](#). Newer and less expensive purified chick embryo cell vaccine, and

purified Vero cell rabies vaccine are now available. The purified Vero cell rabies vaccine uses the attenuated Wistar strain of the rabies virus, and uses the Vero cell line as its host.

[\[edit\]](#)

Post-exposure prophylaxis

Treatment after exposure (known as [post-exposure prophylaxis](#) or "PEP") is highly successful in preventing the disease if administered promptly, within 14 days after infection. In the United States, the treatment consists of a regimen of one dose of [immunoglobulin](#) and five doses of rabies vaccine over a 28-day period. Rabies immunoglobulin and the first dose of rabies vaccine should be given as soon as possible after exposure, with additional doses on days 3, 7, 14, and 28 after the first. The vaccinations are relatively painless and are given in one's arm, in contrast to previous treatments which were given through a large needle inserted into the abdomen. In case of animal bites it is also helpful to remove, by thorough washing, as much infectious material as soon as possible. Since the development of effective human vaccines and immunoglobulin treatments, the US death rate from rabies has dropped from 100 or more annually in the early 20th century, to 1–2 per year, mostly caused by bat bites, which may go unnoticed by the victim and hence untreated.

PEP is effective in treating rabies because the virus must travel from the site of infection through the [peripheral nervous system](#) (nerves in the body) before infecting the [central nervous system](#) (brain and spinal cord) and glands to cause lethal damage. This travel along the nerves is usually slow enough that vaccine and immunoglobulin can be administered to protect the brain and glands from infection. The amount of time this travel requires is dependent on how far the infected area is from the brain: if the victim is bitten in the face, for example, the time between initial infection and infection of the brain is very short and PEP may not be successful.

[\[edit\]](#)

Pre-exposure prophylaxis

Currently pre-exposure immunization has been in domesticated and wild animal populations. In many jurisdictions, domestic dogs, cats, and ferrets are required to be vaccinated. A new, orally active, genetically recombinant virus vaccine for [raccoon](#) rabies awaits licensing by the U.S. [Department of Agriculture as of 2006](#). A gene that produces a protein in the rabies virus outer coat was inserted into a live [vaccinia virus](#) using recombinant DNA technology. When the modified vaccinia virus infects a wild animal, it produces the antigenic protein normally made by the rabies virus. The wild animal's body recognizes the protein as foreign, and the animal develops active immunity. The plan for immunization of wild populations involves dropping bait containing food wrapped around a small dose of the live virus. The bait would be dropped by helicopter concentrating on areas that have not been infected yet.

A pre-exposure vaccination is also available for humans, most commonly given to veterinarians and those traveling to parts of the world where the disease is common, such as India. (Most tourists would not need such a vaccination, just those doing substantial outdoor non-urban activities.) However, should a vaccinated person be bitten by any animal possibly having rabies, they **must** have subsequent post-exposure treatment. Failure to do so could be fatal. Such post-exposure treatment is far less extensive than what would normally be required by someone with no pre-exposure vaccination.

[\[edit\]](#)

Prevalence



Countries with Rabies-Free status (in red), as of January 2006


More than 99% of all human deaths from rabies occur in Africa, Asia and South America; India alone reports 30,000 deaths annually.^[1]

Dog licensing, killing of stray dogs, muzzling and other measures contributed to the eradication of rabies from the [United Kingdom](#) in the early [20th century](#). More recently, large-scale vaccination of cats, dogs and ferrets has been successful in combating rabies in some developed countries.

Rabies virus survives in widespread, varied, rural wildlife reservoirs. However, in Asia, parts of Latin America and large parts of Africa, dogs remain the principal host. Mandatory vaccination of animals is less effective in rural areas. Especially in developing countries, animals may not be privately owned and their destruction may be unacceptable. Oral vaccines can be safely distributed in baits, and this has successfully impacted rabies in rural areas of [France](#), [Ontario](#), [Texas](#), [Florida](#) and elsewhere. Vaccination campaigns may be expensive, and a cost-benefit analysis can lead those responsible to opt for policies of containment rather than elimination of the disease.

Rabies was once rare in the United States outside the [Southern states](#), but [raccoons](#) in the mid-Atlantic and northeast United States have been suffering from a rabies epidemic since the [1970s](#), which is now moving westwards into [Ohio](#).^[2]



 Cases of animal rabies in the United States in 2001

The particular variant of the virus has been identified in the southeastern United States raccoon population since the [1950s](#), and is believed to have traveled to the northeast as the result of infected raccoons being among those caught and transported from the southeast to the northeast by hunters attempting to replenish the declining northeast raccoon population.^[3] As a result, urban residents of these areas have become more wary of the large but normally unseen urban raccoon population. It has become the common assumption that any raccoon seen in daylight is infected; certainly the reported behavior of most such animals appears to show some sort of illness, and autopsies usually confirm rabies. Whether as a result of increased vigilance or just the normal avoidance reaction to any animal not seen in the course of day to day life, such as a raccoon, there has only been one documented human rabies case as a result of this variant.^[4] ^[5] This does not include, however, the greatly increasing rate of prophylactic rabies treatments in cases of possible exposure, which numbered fewer than 100 persons annually in New York State before [1990](#), for instance, but rose to approximately 10,000 annually between [1990](#) and [1995](#). At approximately \$1500 per course of treatment, this represents a considerable public health expenditure. Raccoons do constitute approximately 50% of the approximately 8,000 documented animal rabies cases in the United States.^[6] Domestic animals constitute only 8% of rabies cases, but are increasing at a rapid rate.^[6]



 A rabid dog, with saliva dropping out of the mouth

In the midwestern United States, [skunks](#) are the primary carriers of rabies, comprising 144 of the 237 documented animal cases in [1996](#). The most widely distributed reservoir of rabies in the United States, however, and the source of most human cases in the U.S., are [bats](#). Nineteen of the 22 human rabies cases documented in the United States between [1980](#) and [1997](#) have been identified genetically as bat rabies. In many cases, victims are not even aware of having been bitten by a bat, assuming that a small puncture wound found after the fact was the bite of an insect or spider; in some cases, no wound at all can be found, leading to the hypothesis that in some cases the virus can be contracted via inhaling airborne [aerosols](#) from the vicinity of a bat or bats. For instance, the [Centers for Disease Control and Prevention](#) warned on [May 9, 1997](#), that a woman who died in October, [1996](#) in [Cumberland County, Kentucky](#) and a man who died in December, [1996](#) in [Missoula County, Montana](#) were both infected with a rabies strain found in silver-haired bats; although bats were found living in the chimney of the woman's home and near the man's place of employment, neither victim could remember having had any contact with them. This inability to recognize a potential infection, in contrast to a bite from a dog or raccoon, leads to a lack of proper prophylactic treatment, and is the cause of the high mortality rate for bat bites.

In case of an attack by a possibly rabid animal, most states in the United States allow the killing of the attacking animal. Because a rabies diagnosis requires that the brain tissue be preserved, it is recommended that rabid animals are not to be shot in the head.

[Australia](#) is one of the few parts of the world where rabies has never been introduced. However, the Australian Bat Lyssavirus occurs naturally in both insectivorous and fruit eating bats (flying foxes) from most mainland states. Scientists believe it is present in bat populations throughout the range of flying foxes in Australia.

Many territories, such as the [United Kingdom](#), [Ireland](#), [Hawaii](#), and [Guam](#), are free of rabies (although there may be a very low prevalence of rabies among bats in the UK; see below).

