



## Lecture title: Rabies

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**Summary:** Rabies is a deadly, zoonotic, neurologic disease caused by a bullet-shaped, enveloped RNA virus that belongs to the genus *Lyssavirus* (from *Lyssa*, the Greek goddess of madness, rage, and frenzy).

The virus is fragile in the environment, and readily inactivated by a variety of disinfectants, soap, ultraviolet light, and heat. It can survive up to 3 to 4 days in carcasses at 20°C, and longer with refrigeration.<sup>2,3</sup> Freezing tissues at temperatures less than -20°C may prolong survival of the virus for years.

### Signs and Their Pathogenesis

After inoculation into the subcutaneous tissues and muscle, rabies virus replicates locally within cells and then attaches to peripheral nerve endings. Local replication around the bite site can continue for months before the virus enters peripheral sensory and motor nerve endings, with the nicotinic acetylcholine receptor being the main receptor for the virus.

Once the virus is within the CNS, there is massive viral replication, with cell-to-cell transmission of virus across synaptic junctions. The spinal cord, medulla oblongata, periaqueductal gray matter, brainstem, and cerebellum are particularly affected. The virus also moves outwards from the CNS into peripheral and autonomic nerves and is deposited in a variety

of tissues, including cardiac and skeletal muscle, the eye, the kidney, pancreas, nerves around hair follicles, and the salivary glands. Production of new virions through budding from the plasma membrane occurs primarily within the salivary glands, which results in the shedding of virus that can be transmitted to other hosts. Thus, the presence of virus in the saliva indicates that the CNS has been infected. In domestic animals, death occurs before the saliva becomes infected. Virus is shed by some dogs for up to 10 days before the onset of clinical signs.



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The clinical presentations of rabies virus infection have been divided into excitatory (“furious”) and paralytic (“dumb”) forms. Three phases have been described in the progression of the disease, the prodromal, furious, and paralytic phases, but the stages are variable and may overlap, and signs may be atypical . Infrequently, there is a history of a wound, or wounds are still present at the time neurologic signs occur. Unfortunately, rabies is infrequently suspected as the possible diagnosis at the time rabid animals are examined by veterinarians.

When it occurs, the prodromal phase lasts 2 to 3 days in dogs and 1 to 2 days in cats, and is characterized by a variable fever, licking or chewing at the bite site, and behavioral changes. Dogs and cats may become lethargic, anorexic, apprehensive, restless, or reclusive, and vomiting may occur. Pupillary dilation, sometimes with decreased pupillary light reflexes, may occur.

The furious phase occurs in approximately two thirds of affected cats and dog and lasts to 7 days. Clinical signs result from forebrain involvement and include irritability, anxiousness or excitability, hyperesthesia, hypersalivation, vocalization, roaming, and aggression. Affected animals may try to eat foreign objects, which may become lodged within the gastrointestinal tract, or they may attack their surroundings or moving objects.

Some animals develop ataxia, vestibular signs, and seizures. Tremors; staring, or a wild, spooky, or blank look in the eyes; increased vocalization; and compulsive running can occur cats, which also often develop aggressive behavior.

The paralytic phase develops 1 to 10 days after the onset of clinical

signs and is characterized by flaccid paralysis, which is ascending and often initially involves the bitten extremity. Neurologic examination reveals flaccid paralysis with absent segmental reflexes. Laryngeal paralysis may lead to a change in the sound or pitch of a dog’s bark or cat’s meow.

Hypersalivation results from paralysis of the pharyngeal muscles, and a “dropped jaw” can occur as a result of masticatory muscle paralysis, especially in dogs, which may appear to owners as if they are choking