

# **EHV in Massachusetts: Equine Herpes Spreads from New Jersey**

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**April 6, 2010** - The Massachusetts Department of Agricultural Resources has alerted horse owners, breeders, and veterinarians in this state that four horses here are known to have been exposed to the EHV-1, a contagious virus, in New Jersey. The horses were subsequently transported to Massachusetts. One of the horses has since tested positive for the virus and is exhibiting

clinical signs associated with the disease.

Agricultural officials are urging horse owners to take precautions to curb possible spreading of the virus among the state's horse population. EHV-1 is a contagious disease of horses, which can cause respiratory illness, spontaneous abortion, neurological disease, and death



## **Botulism: Should you vaccinate?**

Botulism is recognized by many as an illness that comes from eating contaminated food. For horses, botulism can be deadly. The silent killer has left a Rusk County family reeling.

In March 2010, the field in front of Bob and Bonnie Rosolowski's house was full of galloping horses. Now, just 3 remain. "The first one we lost was my old mare; she was 33; we had her for 31 years," said Bonnie tearfully. In a matter of weeks, Bob and Bonnie lost 5 horses. They were up around the clock, fighting to save the animals they loved so much. "Sometimes almost all night long," added Bob, "trying to figure out what was going on, what we could do for them. Nobody around here had any experience with it."

After area farmers began reporting similar problems with cattle, a veterinarian was able to identify the illness as botulism. The toxins in botulism are found in soil and in decaying plant or animal matter. Bonnie suspects the snow melt and warmer weather may have created the perfect environment for the spores to grow onto the hay that's fed to the horses. "The toxin is always there," said Bonnie. "It just takes the right set of circumstances to activate the spores."

The disease works quickly, attacking the nervous system. Eventually the animal loses muscle control, and suffocates. "Even though they are paralyzed, and they cannot motivate their muscles themselves, they feel everything," continued Bonnie. "They feel all the pain."

An anti-toxin exists, but is not widely available and is very expensive. The Rosolowski's were able to get the medicine in time to save their remaining 3 horses, but the emotion of losing five is still fresh. Hay from the farm is now being tested to find out whether that is where the horses contracted botulism. For more information about the illness, or vaccine, contact

your local veterinarian.

[The account of the horses and nearby cattle being affected lends credibility to the thought that the melting snows may have uncovered the spores or the bacterium.]

Differences in antigenicity among the toxins produced by different strains of botulism-causing organisms allow for separation of the organisms into 7 distinct types (A-G). Types A, B, and E are the toxins most often responsible for disease in humans, whereas types C and D only cause disease in other animals (e.g., nonhuman mammals, birds, fish). In rare instances, a single strain of organism may produce 2 toxins. C botulinum is distributed widely throughout the environment and can be found in soil, freshwater and saltwater sediments, household dust, and on the surfaces of many foods. The toxins produced are cytoplasmic proteins (mass = 150 kDa) released as cells lyse. While the spores survive 2 hours at 100 C (but die rapidly at 120 C), the exotoxin is heat labile and becomes inactive after one minute at 85 C or 5 minutes at 80 C.

Although the mode of entry of toxin may differ between the different forms of diseases, once the toxin enters the bloodstream, it acts in a similar manner to produce the clinical signs and symptoms. The toxin binds to receptors on presynaptic terminals of cholinergic [neuron] synapses, is internalized into vesicles, and then is translocated to the cytosol. In the cytosol, the toxin mediates the proteolysis of components of the calcium-induced exocytosis apparatus (the SNARE proteins) to interfere with acetylcholine release. Blockade of neurotransmitter release at the terminal is permanent, and recovery only occurs when the axon sprouts a new terminal to replace the toxin-damaged one.