



# **SOUTH WILLAMETTE VETERINARY CLINIC**

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## **Potomac Horse Fever Outbreak in Pacific Northwest**

By Wes Violet, DVM      12-1-2008

### **Introduction**

Potomac Horse Fever was originally diagnosed in the Potomac River region of Maryland over 25 years ago causing cases of colic and severe diarrhea. A rickettsial organism, *Neorickettsia risticii* was eventually isolated as the causative agent. Serologic antibody surveys have suggested exposure of horses to the organism in over 40 states and in Canada, South America, and Europe. However, detection of DNA of the organism by polymerase chain reaction (PCR) from actual clinical cases has proven much more rare. This may be due to the high number of false positives found by serology.

### **Epidemiology**

Determining the life cycle of *N. risticii* was difficult. Like many other rickettsia, it was initially thought to involve a tick as an insect vector. Proof of tick transmission was unrewarding. Since the organism is similar to the causative agent of salmon poisoning in dogs and equine cases usually occur along streams, rivers and irrigated pastures, freshwater snails were suspected as vectors. In 2005, John E. Madigan and N. Pusteria of UC Davis established that the rickettsia is carried in trematodes (flukes) to freshwater snails that serve as primary intermediate hosts. Subsequently, the organism is carried to the aquatic larval stages of certain insects including caddis flies, mayflies, stoneflies and dragonflies.

Horses become infected by ingesting dead insects in pastures, streams and pond water, or contaminated feed. Peak season of infection is commonly mid summer to early fall when insect activity is highest.

### **Clinical Signs**

After ingestion of insect parts, affected horses may exhibit mild depression and anorexia within 10-14 days. Soon after, transient biphasic fevers of 102 – 107 F develop, followed by a more persistent fever 3-7 days later. Many exhibit congested, injected mucous membranes. The organism has a predilection for the mucosa of the colon and cecum so ileus, or a loss of intestinal motility is a very common early clinical sign. Diarrhea develops in ~ 60% of untreated cases, and may become severe and projectile in 24-48 hrs. Dehydration can occur and laminitis develops in ~ 25-30% of PHF cases. Mortality rates vary from 5 – 30%. Abortions can occur in infected pregnant mares.

## **Diagnosis**

Differential diagnoses of PHF include other enterocolitis diseases in horses. These include parasitic causes such as small strongyles, acute salmonellosis, clostridial colitis, and drug toxicities.

Laboratory testing for PHF is available, but is not always accurate.

Serologic testing using fluorescent antibodies are performed at the Oregon State Veterinary Diagnostic Laboratory for most of the suspected cases from Washington, Idaho, and Oregon.

Serologic accuracy is sometimes questioned because of unpredictable antibody levels achieved in infected animals and the large number of strains of *N. risticii* isolated.

At Corvallis, any acute titers > 1:160 are presumptive for PHF diagnosis in face of clinical signs and treatment is advised. If titers are < 1:160, retesting is recommended in 7-10 days for confirmation with convalescent serum.

Other testing for PHF can be performed at UC Davis utilizing PCR to detect *N. risticii* DNA in whole blood or feces. Although somewhat more expensive, the test has fewer false positives and additional PCR assays can be performed to diagnose salmonella or clostridial organisms.

## **Treatment**

It is important that a horse showing early signs of PHF be treated with IV oxytetracycline before colitis worsens and diarrhea becomes life threatening. Flunixin meglumine (Banamine) therapy is also recommended to control endotoxemia and prevent laminitis. This often requires making a presumptive diagnosis based solely on clinical signs for horses in endemic areas. Supportive lab results are usually obtained later. Horses that develop severe diarrhea often require referral for intensive care and IV fluid maintenance.

## **Re- emergence in Pacific Northwest**

The first major outbreaks of PHF occurred in the Oregon, Washington, and Idaho in the late 1980's prompting many equine practices to recommend vaccination for horses in endemic areas. Since that time, positive cases dropped to roughly 6-12 / year for the entire region. Routine vaccination fell out of favor.

From July to December of 2008, 30 horses with enterocolitis tested positive for PHF at the Oregon State Veterinary Diagnostic Lab with 2 additional positives confirmed at UC Davis. Ten of these animals eventually were referred to the Veterinary Teaching Hospital for intensive care. It is unknown how many other positive samples were submitted to other labs or how many cases were treated without testing. Oregon's Lane and Jackson counties lead with 5 positive cases each.

It is common especially for vector borne diseases to vary in incidence in endemic areas.

Many factors are involved including climatic conditions, numbers of requisite vectors, and the immune status of the susceptible equine population. This autumn in the Northwest was particularly mild and caddis fly and mayfly hatches were observed as late as mid November. Concurrent with the PHF outbreak, horses here were also experiencing the worst Pigeon Fever epidemic in recent memory. Pigeon Fever is caused by a *Corynebacterium sp* spread by biting flies. In summer and fall 2009, it may become evident that our region is endemic for these diseases once more.

## **Prevention**

There are currently two killed vaccines available for the prevention of PHF in healthy horses. There has been somewhat mixed results with their success in practice. Vaccine failure to protect animals has sometimes been reported in endemic areas. This may be attributed to poor antibody response, different strains of *Neorickettsia* causing PHF, or inadequate timing or frequency of vaccination. Although vaccinates may still become infected, it is generally thought that the severity of clinical signs is reduced. Cell mediated immunity simulated by vaccination may play an important role.

Horses in affected regions should be vaccinated with a two dose primary series 3-4 weeks apart, ideally in late spring/ early summer. Boosters may be required at 4-6 month intervals in problem areas.

Other control measures can include limiting grazing access to pastures along creeks and ponds during aquatic insect hatches. Dimming stable lights that attract insects at these times can also be helpful.

Horses that recovered from PHF have a natural immunity that lasts ~ 20 months.

Infected horses are not a risk to other stabled animals and a carrier state has not been documented.