This information is a professional communication for the equine industry. The OAHN group is a dedicated group of veterinarians from primary care practices, academia, government and laboratories, who meet regularly to discuss equine disease and health issues. It is the intent of this program to monitor and protect the health of horses in Ontario.

Ontario Animal Health Network (OAHN)
Equine Expert Network
Quarterly Owner Report – July to September 2016

July-September 2016 Report #6

Highlights
Key Points
Facts about Red Maple Toxicity
Looking Ahead – Equine Proliferative Enteropathy

Your OAHN Equine Network Team:

Practitioners
Dr. Drew Hunnisett
(Network Co-Lead)
Dr. Chris Grossenbacher
Dr. Melissa McKee
Dr. Jessica Peatling
Animal Health Lab
Dr. Murray Hazlett
Ontario Vet College
Dr. L. Memo Arroyo
OMAFRA
Dr. Alison Moore
(Network Co-Lead)
Dr. Cathy Furness
Tim Pasma

Ontario Equine Disease Surveillance (July to September) - Key points

- There was an increase in horses with colic in July and August reported by survey respondents as well as network members.
- Equine influenza was diagnosed in a racehorse during an outbreak of respiratory disease.
- Potomac Horse Fever was diagnosed in a few horses with diarrhea.
- Two horses infected with West Nile Virus were identified in the Toronto area and Middlesex County respectively. OMAFRA's equine neurological surveillance map can be found here.

The maple leaf has been a Canadian emblem since the 18th century, but did you know if can be toxic to horses?

Facts about Red Maple Toxicity (*Acer* species)

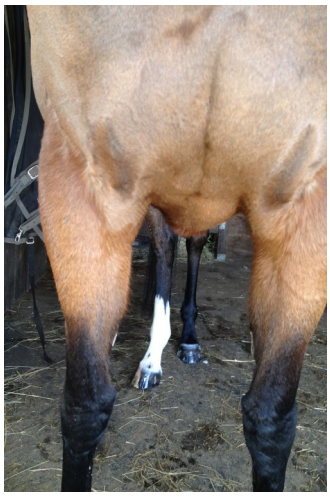

Red Maple (*Acer rubrum)*

- Autumn is the time when Red Maple toxicity in horses is diagnosed in Ontario. Toxicity occurs from ingestion of dried or wilted (NOT fresh) red maple leaves. Other members of the Acer family have also caused toxicosis after ingestion.
- Dried leaves can remain toxic for 4 weeks[1].
- Horses develop anemia due to red blood cell and hemoglobin damage caused by gallic acid, an oxidant present in red maple leaves. Methemoglobin, a form of hemoglobin incapable of carrying oxygen in the blood, is formed.
- Horses may show signs of weakness, lethargy, icterus (yellowing of the mucous membranes and whites of the eyes), rapid heart rate, dark red/brown/black urine due to the presence of methemoglobin or sudden death.
- There is no age, sex or breed that is affected more frequently.
- Treatment includes supportive therapy: intravenous fluids, non-steroidal anti-inflammatory medication, whole blood transfusion, ascorbic acid, furosemide and mineral oil.
- Fences should be placed so not to allow horses to come in contact with wilted maple leaves.[1]

[1] http://www.extension.umn.edu/agriculture/horse/pasture/maple/
http://www.omafra.gov.on.ca/english/livestock/horses/facts/06-109.htm

Looking Ahead—Diarrhea and low protein blood levels in weanlings and yearlings. Could it be Equine Proliferative Enteropathy (EPE)?

- Equine Proliferative Enteropathy (EPE) is a syndrome in weanlings and yearlings caused by the bacterium *Lawsonia intracellularis.* This organism causes thickening of the intestinal lining leading to a syndrome of fever, lethargy, edema (fluid) around the jaw, legs and/or in the girth area, and/or diarrhea.
- Low protein, particularly a protein called albumin, found on testing the blood is a hallmark finding.
- EPE affects 2-13 month olds but is diagnosed most frequently in 6-8 month old horses. It has been diagnosed in adult horses up to 17 years of age although adult horses usually have an underlying disease[1]. Colts are more frequently diagnosed than fillies[2].
- In North America, cases of EPE usually occur from August to January[3] and the bacterium can live in the environment for up to 14 days in manure at 5-15 C[o5].



Horse with edema in the girth area

Risk Factors

- The stress of weaning and heavy parasite burdens have been identified as risk factors on the farm for the development of EPE[1]. Weanlings in Ontario have died due to EPE and severe cyathostome (small strongyle) infection occurring at the same time[4].
- Weaning foals after September may lower the likelihood of them developing EPE than foals weaned earlier in the year[2].

Clinical Signs

- The most common signs of EPE are; weight loss, lethargy, fever (>38.5°C), edema, diarrhea, colic.
- A severe form of EPE can occur which causes diarrhea and a bleeding disorder (called disseminated intravascular coagulation or DIC) with a rapid progression and poor prognosis[1] .
- Some foals may appear normal but will have a low protein/albumin level.

Diagnosis

- Low protein and particularly albumin may be one of the most consistent and earliest indicators of EPE. Typically, a DNA test performed on fresh feces is most often used to diagnose infection with *L. intracellularis.* A serum test (serum IPMA) is also available that indicates exposure to *L. intracellularis* and may be used in conjunction with other tests to identify affected horses. Your veterinarian may use an ultrasound to identify thickened loops of small intestine as part of their workup.
- Other diagnoses your veterinarian may consider include bacterial infections, viral causes of diarrhea, intestinal ulceration and parasitism.

Treatment

- Antibiotics are the common medication used to treat EPE. Other treatments may include intravenous fluids, plasma (as a source of protein) and antiulcer medication.

Prevention

- Your veterinarian may use an off-label swine vaccine but only on farms where EPE is a problem.
- Monthly protein levels may be monitored to identify at-risk horses and treat early.

Biosecurity

- Rabbits also carry the bacterium *L. intracellularis*, can transmit it to horses making them ill, and may act as a reservoir for the organism on the farm[5]. Therefore rabbits and other wildlife should have limited access to pastures and food.
- Horses with EPE should be isolated from the herd until 1 week after antibiotic therapy has finished to reduce environmental contamination[3].

Prognosis for EPE in Racehorses

- In one study, foals affected with EPE sold for less at yearling sales than non-affected yearlings from the same stallion.
- There were no differences in race earnings between groups[6].

[1] Slovis, NM In Practice. 2016 June, 38: 287-292

[2] Page AE et al. Equine Vet J. 2015 Nov;47 (6):655-61

[3] *Page AE, Slovis NM, Horohov DW. Vet Clin North Am Equine Pract. 2014 Dec;30(3):641-58*

[4] Arroyo LG et al. Can Vet J. 2013 Sep;54(9):853-8.

[5] *Vannucci FA, Gebhart CJ. Vet Pathol. 2014 Mar;51 (2):465-77.*

[6] Frazer M, Abstract ACVIM , JVIM . 2011 Jun: 677-678

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