# Atypical myopathy

Atypical myopathy is a seasonal condition of non-exertional rhabdomyolysis seen in pastured horses. It occurs in the autumn and spring from ingestion of seeds and seedlings of sycamore (*Acer pseudoplatanus*) in Europe and box elder (*Acer negundo*) in North America. The toxins hypoglycin A and its homologue methylenecyclopropylglycine are metabolised to compounds that impair lipid metabolism, which primarily affect cardiac, respiratory and postural muscles. Initial signs are reluctance to move with muscle weakness and stiffness. Common signs are pigmenturia, hypothermia and pain of varying severity. Mortality is high and many animals succumb within 2–3 days. Treatment is intensive supportive care, including nutritional support to provide alternative energy substrates and vitamin and mineral supplementation. There is no specific antidote for atypical myopathy, so prevention is key.

https://doi.org/10.12968/ukve.2022.6.3.96

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**Key words:** poisoning | toxicology | sycamore | atypical myopathy | hypoglycin A

Atypical myopathy (also known as seasonal pasture myopathy, equine atypical myopathy and atypical myoglobinuria) is a rhabdomyolysis of horses that is not induced by exercise, and which has a high mortality rate. It was periodically reported in the veterinary literature at least as far back as the 1930s in Wales (Bowen and Craig, 1942) and has since been reported in most of Europe (Votion et al, 2020), North America (Pope and Heslop, 1960; Valberg et al, 2013), Australia (Irwin and Pulsford, 1951) and New Zealand (McKenzie et al, 2016).

## Circumstances of poisoning

Atypical myopathy is seasonal and occurs in horses at pasture that eat the seeds and seedlings of sycamore or box elder. Various factors may be involved but most cases occur in the autumn from ingesting the winged fruits (samaras) containing the seeds (Unger et al, 2014) (*Figure 1*). Stormy weather may lead to heavy contamination of pasture. Sterile fruits of box elder do not contain seeds and are free of hypoglycin A (Votion et al, 2019).

Cases also occur in the spring when horses ingest seedlings (Baise et al, 2016). In the spring, grass growth may be limited by cooler weather and seedlings may protrude above the grass, making ingestion more likely (Votion et al, 2019). Clusters of flowers (inflorescences) may also be sources of exposure and windy weather can lead to contaminated pastures. Cases of atypical myopathy in the summer and winter are uncommon (Durham, 2015). Other *Acer* species, such as the field maple (*Acer campestre*) and the Norway maple (*Acer platanoides*), do not contain detectable concentrations of hypoglycin A and are considered safe for horses (Westermann et al, 2016).

It has been shown that neither mowing nor spraying with herbicide reduces the toxicity of sycamore seedlings and pastures contaminated with sycamore should not be used to produce hay or silage (González-Medina et al, 2019).

Although there is a higher incidence in younger horses, older animals can also be affected (van Galen et al, 2012a). Younger animals may be at greater risk because they are more likely to eat unsuitable forage compared to older, more picky animals.

Atypical myopathy has also been reported in donkeys, zebras (van Galen et al, 2012a), deer (Bunert et al, 2018; Bochnia et al, 2021) and camels (Hirz et al, 2021). Humans are also susceptible to poisoning with hypoglycin A and its homologue methylenecyclopropylglycine, although clinical manifestations are different. It is the cause of Jamaican vomiting sickness, caused by ingesting unripe ackee fruit (*Blighia sapida*) (Kakpo et al, 2020) and toxic encephalopathy from ingesting lychee (*Litchi chinensis*) in association with undernutrition and prolonged fasting (Sarkar et al, 2020). These plants and *Acer* species all belong to the *Sapindaceae* plant family.

## Cause and mechanism

The cause of atypical myopathy was unknown for many years but has been shown to occur following ingestion of seeds and seedlings of some *Acer* species containing two cyclopropylamino acids, hypoglycin A and methylenecyclopropylglycine. Although the presence of cyclopropyl derivatives in these species was reported some time ago (Fowden and Pratt, 1973), it was not until 2012 that it was determined that box elder (*Acer negundo*) (*Box 1*) was present in the pastures of horses that developed pasture myopathy in the US and that hypoglycin A was present in the seeds (Valberg et al, 2013). A related tree, sycamore (*Acer pseudoplatanus)*, was identified in European pastures (Votion et al, 2014) and the seeds were found to contain hypoglycin A (Unger et al, 2014) *(Figure 1*).

These compounds are not toxic in themselves, but after ingestion need to be converted to the active metabolites methylenecyclopropyl acetic acid and (methylenecyclopropyl)formyl-CoA. Various metabolites can be detected in the blood of horses that

have ingested sycamore or box elder (Valberg et al, 2013; Votion et al, 2014). Methylenecyclopropyl acetic acid binds to coenzyme A forming (methylenecyclopropyl)formyl-CoA. This induces acquired multiple acyl-CoA dehydrogenase deficiency syndrome, affecting several mitochondrial dehydrogenases that use flavin adenine dinucleotide as a cofactor. This prevents the oxidation of fatty acids and, as a result, the production of energy in mitochondria. Adenosine triphosphate production is limited and there is a build up of toxic intermediates. Type 1 muscle fibres, which are more dependent on fatty acid oxidation for energy needs, are affected. These types of muscle fibres are found in cardiac, respiratory and postural muscles. Affected horses are unable to use lipids, the most efficient energy source, and become more reliant on anaerobic glycolysis, resulting in glycogen depletion and lactic acidosis. There is excessive lipid storage in muscle cells and abnormal production of acylcarnitines and urine organic acids (Westermann et al, 2008; Bochnia et al, 2015). It was the identification of this alteration in lipid metabolism that was key to identifying the cause of atypical myopathy in horses (Votion, 2016).

As a result of this mechanism of action, atypical myopathy has also been called acquired equine multiple acyl-CoA dehydrogenase deficiency (Karlíková et al, 2018).

## Toxic dose

The amount of hypoglycin A in sycamore seeds is very variable and has been measured as 3.6–252.9µg/seed (Unger et al, 2014) or 1.7–320 µg/g of seed (Bochnia et al, 2015). The concentration in box elder seeds in one study varied from 3–160µg/seed (Valberg et al, 2013). Based on the toxic dose in rats (Blake et al, 2006), the maximum tolerated dose in a 500kg horse is 26.5mg/horse (Valberg et al, 2013), and this would be equivalent to between 105 and more than 8000 sycamore seeds.

Approximately 20g of samaras, 50g of seedlings or 150g of inflorescences are the estimated maximum tolerated daily dose for a horse (Votion et al, 2019). Water is also a potential source of exposure, and it is estimated that 2 litres of water that has been in contact with seedlings is the maximum tolerated daily dose for a horse (Votion et al, 2019). Individual response is also known to be variable and other factors may be required for the disease to develop.

## Clinical signs

There may be a period of 4 days or more after the plant material is ingested before the onset of clinical signs (Rendle, 2016). Signs can occur after the horse has been removed from pasture (van Galen et al, 2008) and in some cases animals may also be found dead on pasture without having developed any apparent signs (van Galen et al, 2008).

Initial signs of atypical myopathy include a reluctance to move and a quiet demeanour. Signs then progress to pronounced muscle weakness (with stiffness, carrying the head low, dysphagia, recumbency and respiratory distress) and pain (sweating, depression, and gastrointestinal impaction). Tachycardia may result from cardiomyopathy and/or pain. Pigmenturia with dark red/brown urine occurs in more than 90% of cases (Rendle, 2016). Distended bladder and hypothermia may occur (Votion et al, 2007). The se-

*Figure 1. The paired winged fruits (samaras) of sycamore (*Acer pseudoplatanus*) containing two seeds. Only the seeds contain hypoglycin A.*

## Box 1. The trees

***Acer pseudoplatanus* (sycamore, sycamore maple)**

A large deciduous tree (up to 35m in height), native to central, eastern and southern Europe. It is thought to have been introduced into the UK in the Middle Ages but may have been earlier. The bark is dark pink-grey and smooth when young, but becomes cracked and develops small plates with age. Twigs are pink-brown and hairless. The palmate leaves measure 7–16cm and have five lobes. Leaf stalks of younger trees are characteristically red. The flowers are small, green-yellow and hang in spikes. After pollination by wind and insects, female flowers develop into distinctive winged fruits known as samaras. These trees are the main cause of atypical myopathy in Europe.

***Acer negundo* (box elder, ashleaf maple, box elder maple, Manitoba maple)**

A fast growing and short-lived medium-to-large tree native to North America. It is a species that was introduced to Europe. It is known as Box Elder as the wood is white like Box, and the leaf resembles that of Elder (making this species unique among Maples). Unlike other *Acer* species, *A. negundo* is dioecious with separate male and female trees. The flowers are small, drooping racemes and the seeds are paired samaras. They drop in autumn but may persist through winter. These trees are the main cause of atypical myopathy in North America.

verity of pain can be variable (Votion et al, 2007) but severe pain has been reported occasionally (Pope and Heslop, 1960; Brandt et al, 1997). Appetite is usually maintained (Votion et al, 2007).

Cardiac effects reported in affected horses include mildly increased heart rate, heart murmurs (Votion, 2016), ventricular premature depolarisations, prolonged corrected QT intervals and abnormal myocardial wall motion on echocardiograph (Verheyen et al, 2012). In one study, an affected horse still had ventricular premature depolarisations and prolonged corrected QT intervals at follow up after 10 weeks (Verheyen et al, 2012).

Secondary complications are common in horses with atypical myopathy. These include head oedema, buccal ulceration, gastric ulceration, choke, diarrhoea, renal dysfunction and paraphimosis (van Galen et al, 2012a). Recumbent horses are also prone to pressure sores and corneal ulceration.

Signs are caused by acute degeneration in postural and respiratory muscles and sometimes the myocardium (Votion et al, 2014). Death can occur from cardiac failure caused either by extensive degeneration of the myocardium or indirectly by asphyxia, which results from diffuse pulmonary oedema caused by congestive heart failure and reduced ventilation that is associated with respiratory muscle necrosis (Cassart et al, 2007).

## Laboratory findings

Hyperlipidaemia and hyperglycaemia occur as a result of stress, catabolism, impaired lipid metabolism, reliance on carbohydrate metabolism and increased hepatic gluconeogenesis. Muscle enzymes are increased with elevated creatine kinase, lactate dehydrogenase and aspartame transferase. Increased cardiac troponin I concentrations occur as a result of myocardial damage (Votion et al, 2007; Verheyen et al, 2012).

Hypocalcaemia is common in horses with atypical myopathy (van Galen et al, 2008). There may also be respiratory alkalosis, lactic acidosis or a combination of these (van Galen et al, 2013). Liver dysfunction occurs in some cases (van Galen and Votion, 2013b). Pigmenturia is caused by myoglobinuria and should be distinguished from haematuria and haemoglobinuria (van Galen et al, 2008). Urine and serum concentrations of acyl carnitine and acyl glycines are elevated. Hypoglycin A can be detected in blood and urine (Bochnia et al, 2015; Baise et al, 2016) and can confirm a horse's exposure.

## Pregnancy and lactation

Newborn foals are rarely affected with atypical myopathy but uthe risks are not currently understood. A foal born to a mare that had been diagnosed with the condition when 6 months pregnant was seen to develop signs within 6 hours of birth. At this time, the mare had no signs but was grazing on pasture near sycamore trees. Analysis showed the foal had concentrations of acylcarnitines and methylenecyclopropyl acetic acid-carnitine in the blood consistent with a diagnosis of atypical myopathy. It was unclear if exposure had been placental or was via the mare's milk (Karlíková et al, 2018).

In a second case, a foal developed signs when it was 7 days old. Analysis found that the foal had concentrations of the toxic compounds associated with atypical myopathy insufficient to explain the clinical signs. However, it did have inhibition of the acyl-CoA enoylhydrase, suggesting a genetic disorder resulting in extensive loss of function of this enzyme and inability to metabolise fatty acids. Therefore, the signs that developed resembled atypical myopathy which is also caused by inability to oxidise fatty acids. It has been suggested that vertical transfer of the toxic compounds associated with atypical myopathy is low, except where there are other risks factors related to the mechanism of the disease, as in this case (Sander et al, 2021a). Both the foals described above were euthanised because of the severity of clinical signs.

Hypoglycin A, methylenecyclopropyl acetic acid and their metabolites have been detected in the milk of mares exposed to sycamore (Sander et al, 2021b; Renaud et al, 2021). Although concentrations are usually low, exposure to these compounds in the milk the foals ingest while also grazing will increase the risk of atypical myopathy. Poisoning in foals that are exclusively milk-fed cannot be ruled out. The risk to people that drink the milk of exposed mares has also not been determined.

## Postmortem findings

A postmortem examination of affected animals may be non-specific but findings can include discoloured muscles, including the myocardium, subcutaneous oedema from neck and sternum to hindlimbs (Finno et al, 2006), congested lungs (van Galen et al, 2008) and discoloured urine in the bladder (Whitwell et al, 1988). The main histopathological findings in atypical myopathy are myodegenerative changes (Whitwell et al, 1988) and lipid accumulation in type 1 muscle fibres (Finno et al, 2006; Cassart et al, 2007). Myocardial necrosis may be seen (Finno et al, 2006). Seeds of the suspect plants may be found in the stomach in some cases (Żuraw et al, 2016).

## Diagnosis

Diagnosis of atypical myopathy is based on history, clinical signs, laboratory findings and exclusion of other causes of clinical signs (*Box 2*), particularly rhabdomyolysis.

## Prognosis

Mortality in horses with atypical myopathy is high and many die within 2–3 days of the onset of clinical signs (van Galen, 2008; Votion, 2012). The mortality in individual incidents is variable and has been reported as 56–96% (Brandt et al, 1997; Puyalto-Moussu et al, 2004; Finno et al, 2006; van Galen et al, 2012a; González-Medina et al, 2017; Dunkel et al, 2020). The survival rate is generally around 25%, but it is worth noting that this includes cases that occurred before the mechanism of poisoning was understood and targeted treatment was applied (van Galen et al, 2010, 2012a).

In survivors, there is usually deterioration over 48–72 hours before improvement is seen. Animals that survive beyond 5 days have a better prognosis (van Galen et al, 2010) and there appear to be no long-term sequelae (Valberg, 2014; Rendle, 2016).

Survival has been associated with defaecation, remaining standing most of the time, normal mucous membranes and normothermia (van Galen, 2008). A poor prognostic indicator in clinical studies is dyspnoea with severely lowered arterial partial pressures in oxygen ($PaO_2$) levels. Non-survivors are more likely to be recumbent (van Galen, 2008) but recumbent animals can survive. Of

note, serum creatine kinase activity does not seem to be of prognostic value (Votion et al, 2007), but declining creatine kinase is a positive sign (van Galen and Votion, 2013b).

## Treatment

Treatment of atypical myopathy is supportive and should be started as soon as atypical myopathy is suspected. Any affected horse with suspected atypical myopathy and all herdmates should be removed from pasture where they might be exposed to sycamore or box elder.

The aims of therapy in horses with atypical myopathy are limiting further muscle damage, restoring circulating volume, correcting acid-base and electrolyte disturbances, providing alternative energy substrates to muscle cells and analgesia (Votion, 2016).

## Monitoring

The muscle groups should be inspected and palpated for signs of weakness and stiffness. Renal, liver and respiratory function, triglycerides, creatine kinase, lactate dehydrogenase and aspartame transferase, electrolytes and glucose should be monitored. An ECG and troponin concentrations should be monitored for evidence of myocardial dysfunction (van Galen and Votion, 2013a).

Subclinical toxicosis is common and the creatine kinase should be measured in herdmates of a horse with suspected atypical myopathy (Durham, 2015).

## Fluids and nutrition

Fluid therapy is important in horses with atypical myopathy as it will correct dehydration and acid-base disturbances, and protect the kidneys from myoglobin-induced injury and non-steroidal anti-inflammatory drug (NSAID)-induced renal effects (van Galen and Votion, 2013a; Fabius and Westermann, 2018). Intravenous rehydration is preferred over oral administration as it is less stressful and more rapid (Fabius and Westermann, 2018). The choice of fluids should be based on specific metabolic disturbances (van Galen and Votion, 2013a), but lactated Ringer's solution is suitable in most cases, with further supplementation as required based on electrolyte and blood gas parameters (Carslake, 2019). Fluid therapy should be given until the urine is yellow and the animal is no longer dehydrated.

In atypical myopathy there is impaired lipid metabolism and a shift from aerobic to anaerobic metabolism. Metabolites from anaerobic metabolism derange homeostasis. Therefore, nutritional support should focus on providing an alternative energy source (such as carbohydrates) in affected animals and providing a low-fat diet. This is done with a glucose infusion (*Table 1*) but with monitoring of blood glucose as hyperglycaemia occurs in horses with atypical myopathy (probably because of stress) and this may cause osmotic diuresis and exacerbate dehydration. An insulin infusion may be required to maintain blood glucose (Fabius and Westermann, 2018). Carbohydrate-rich foods include grass, good quality hay, alfalfa, grains, molasses, carrots and apples. Food should be given little and often to prevent large fluctuations in glucose, with free access to grass, hay or alfalfa (van Galen et al, 2012b). In the anorexic or dysphagic horse, a stomach tube may be required (van Galen et al, 2012b).

### Box 2. Differential diagnosis and diagnosis

**Differential diagnosis**

- Colic
- Lameness
  - Arthritis
  - Laminitis
- Endotoxaemia
- Neurological disease
  - Tetanus
  - Botulism
  - Rabies
  - Spinal cord disease
  - Grass sickness
  - Meningitis
- Haematuria or haemoglobinuria of any cause
  - Trauma
  - Exercise
  - Cystitis
  - Calculi
  - Systemic haemolysis
  - Urethral defects
  - Bladder tumour
  - Renal haemorrhage
- Post-anaesthetic myopathy
- Polysaccharide storage myopathy
- Vitamin E or selenium deficiency
- Poisoning
  - Ionophores
  - Organophosphate insecticides
  - Carbamates
  - Strychnine
- Immune-mediated myositis
- Prolonged recumbency

**Diagnosis**

- History
  - Previously healthy horses at pasture
  - Sudden onset of signs
  - Multiple animals affected
  - Possible access to seeds or seedlings of *Acer* species
  - Time of year
  - Poor weather
  - Previous reports at same location
- Exclusion of other conditions/causes of rhabdomyolysis
- Clinical signs
  - Myoglobinuria
  - Muscle weakness and stiffness
  - Depression
  - Recumbency
  - Sweating
  - Muscle tremors
  - Rapid deterioration
- Laboratory findings
  - Large increase in creatine kinase
  - Hypocalcaemia
  - Hypoglycaemia
  - Hyperlipidaemia
  - Methylenecyclopropyl acetic acid-carnitine in blood
- Muscle biopsy (shoulder muscle)

**Table 1. Drug doses in the treatment of horses with atypical myopathy**

| Treatment | Dose |
|---|---|
| Activated charcoal | 1–4g/kg orally as a slurry |
| Vitamin E | 5000IU/day orally |
| Vitamin C | 5 mg/kg/day orally; 5000mg intravenously in 5 litres of lactated Ringer's solution |
| Selenium | 1mg/day orally |
| Carnitine | 100mg/kg orally; 18–22mg/kg intravenously |
| Vitamin B2 (riboflavin) | 44µg/kg/day intravenously or orally |
| Glucose | Up to 40ml/kg/day of a 5% solution |
| Insulin | 0.4U/kg/day subcutaneously or 0.07U/kg/h constant rate infusion with monitoring of blood glucose concentrations |
| Meloxicam | 0.6mg/kg orally |
| Ketoprofen | 2.2mg/kg intravenously |
| Acepromazine | 0.02–0.06mg/kg intravenously; 0.03–0.10mg/kg intramuscularly |

Adapted from van Galen and Votion, 2013a, 2013b; Fabius and Westermann, 2018

Vitamin E and selenium may also be helpful and vitamin E supplementation appears to have a positive effect on outcome in horses with atypical myopathy (Finno et al, 2006; van Galen et al, 2012b). Carnitine supplementation may aid detoxification and stimulate glucose metabolism (Fabius and Westermann, 2018). Vitamin B2 (riboflavin) is part of flavin adenine dinucleotide and has been effective in humans with multiple acyl-CoA dehydrogenase deficiency and, therefore, is recommended in horses (Fabius and Westermann, 2018). Vitamin B2, vitamin C, vitamin E and carnitine have been beneficial in horses with atypical myopathy (van Galen et al, 2012b).

## Muscle relaxants

Muscle relaxants may be used to relieve muscle tremors, cramps and pain but it should be noted that they may potentiate muscle weakness (van Galen and Votion, 2013b). Acepromazine is recommended (Fabius and Westermann, 2018). Methocarbamol should be used with caution as it is potentially nephrotoxic (van Galen and Votion, 2013b).

## Supportive care

Animals with atypical myopathy should be kept warm (Votion et al, 2007) and stress limited where possible. Slings have been used in some cases but attempting to place an affected horse in a sling may add to its stress (van Galen and Votion, 2013a).

Activated charcoal has been shown to bind hypoglycin A in vitro and could be considered if ingestion was recent (Krägeloh et al, 2018), depending on the clinical condition of the animal. It should not be given within 2 hours of any oral medication. Laxatives can be given but are only likely to be useful if ingestion was recent (Fabius and Westermann, 2018).

Analgesia may be required and is recommended in horses with recumbency, as pain relief may allow the animal to stand. NSAIDs are preferred over opioid analgesics because of the adverse effects associated with the latter (Fabius and Westermann, 2018). As stated previously, intravenous fluid therapy is important to protect against NSAID-induced renal effects. Emptying the bladder (manually by catherisation) may help alleviate pain in some cases (Votion et al, 2007). Massage of affected muscles and physiotherapy may ease pain and stimulate blood flow (Rendle, 2016).

Regular turning of recumbent animals is required to reduce exacerbation of myopathy and avoid decubital ulcers (van Galen and Votion, 2013b). Sternal recumbency is preferred over lateral recumbency. Artificial tears may be required to prevent corneal ulceration (van Galen and Votion, 2013b).

Oxygen may be required in animals with respiratory distress or evidence of severe hypoxaemia (van Galen and Votion, 2013b). The head should be elevated to prevent head oedema and aid breathing. Respiratory stimulants are not indicated since they will not improve respiratory muscle dysfunction (van Galen and Votion, 2013b). Antibiotics are recommended for suspected aspiration pneumonia.

## Prevention

Prevention is important as there is no specific treatment for atypical myopathy (Votion et al, 2020):

- Identify trees within and close to grazed fields (*Box 3*)
- Test for hypoglycin A in suspect trees

### Box 3. Resources on atypical myopathy

**Identification of sycamore and other common maples**

- A guide on identification of sycamore, Norway maple and field maple: https://orbi.uliege.be/bitstream/2268/242221/1/Maple-English.pdf

**Further information**

- Royal Veterinary College: https://www.rvc.ac.uk/equine-vet/information-and-advice/fact-files/atypical-myopathy
- The British Horse Society: https://www.bhs.org.uk/advice-and-information/horse-health-and-sickness/atypical-myopathy
- Atypical Myopathy Alert Group, University of Liège, Belgium: https://www.myopathie-atypique.uliege.be/cms/c_7997923/en/myopathie-equines

**Tests to carry out (samples from UK and Europe)**

- A rapid test for hypoglycin A and its principal metabolite methylenecyclopropyl acetic acid-carnitine
- Urine organic acid and plasma acyl carnitine profile testing, which support the diagnosis
- Samples of sycamore seeds, seedlings and leaves can be tested for horse owners who have concerns about trees on their properties
- Muscle biopsy: https://www.rvc.ac.uk/research/laboratories/comparative-neuromuscular-diseases-laboratory/diagnostic-services#panel-atypical-myopathy-testing-for-vets-and-owners

## KEY POINTS

- A seasonal, non-exercise-related rhadomyolysis of horses with a high mortality rate.
- The cause is ingestion of hypoglycin A in seeds or seedlings of sycamore in Europe and box elder in North America.
- Effects are prominently on the respiratory, postural and cardiac muscles as a result of induced impaired fatty acid metabolism.
- Initials signs are non-specific with rapid deterioration; many horses die within 48–72 hours of onset.
- Intensive supportive care is required in affected horses.
- Fluid therapy and nutritional support are vital.
- Preventing ingestion is key.

- Collect seeds, remove seedlings or prevent access to affected areas, using electric fencing or stabling
- Be aware that stormy autumn weather or felling of trees may result in heavy contamination of pasture with seeds or inflorescences
- Supplementary feeding (with hay, straw, oats or barley) throughout the year decreases the risk of atypical myopathy (Votion et al, 2020)
- Provide a salt block
- Provide access to mains water rather than natural sources of water (van Galen et al, 2008; Votion et al, 2020).
- Restrict grazing in risk areas during spring and autumn
- Limiting grazing time to less than 6 hours per day reduces the risk (van Galen et al, 2012a)
- Only graze affected pasture in the summer or winter months.
- Regularly deworm and vaccinate horses

Resources for owners and veterinary professionals are listed in *Box 3*.

## Conclusions

Atypical myopathy is a seasonal disease of horses resulting from impaired lipid metabolism caused by ingestion of toxic amino acids found in some species of *Acer* trees, particularly sycamore and box elder. Mortality rates are high, with many horses succumbing within 2–3 days of onset of signs. There is no antidote and prompt recognition with early and intensive supportive treatment is essential. EQ

### Conflicts of interest

The author declares that there are no conflicts of interest.

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