INTRODUCTION

The genus *Prunus* contains more than 200 species, a number of named hybrids and innumerable varieties and cultivars (1). The *Prunus* family includes both trees and shrubs. These plants are especially important for their agriculturally-harvested, stone fruits, including: cherries, peaches, plums, nectarines, apricots and almonds. Even though this fleshy fruit is commonly and safely consumed by humans, the leaves, shoots, bark and pits of the fruit contain cyanogenic glycosides that can cause poisonings in livestock.

Canada has seven native *Prunus* tree and shrub species. The most common wild species in Ontario are:

- Choke cherry – *Prunus virginiana*
- Pin cherry – *Prunus pensylvanica*
- Black cherry – *Prunus serotina*
- Canada plum – *Prunus nigra*
- American Plum – *Prunus americana*

In a “wild” environment, pin cherry and black cherry probably pose the greatest risk for accidental ingestion by livestock (1). Peach, apricot, plum, sweet cherry and sour cherry can sometimes be found growing wild along pastures, due to the spread of seed from fruit-cultivated orchards.

For *Prunus*, young rapidly growing leaf tissue and the seed tend to contain the highest concentrations of the potential toxin (Figure 1) (2). Wilted and damaged leaves that blow into pastures are also of concern because, when water is initially lost from the leaf, the cyanide becomes more concentrated. However, since cyanide is volatile, as drying continues, cyanide levels decrease (2,3,4). Even though leaves and pits contain the highest concentrations, all parts of the plant, including twigs, dormant buds, branches, and bark, can contain cyanogenic glycosides and should, therefore, be kept away from all animals.

THE TOXIN

The toxic principles of the *Prunus* family are the cyanogenic glycosides prunasin, prulaurasin and amygdalin (2,3). These cyanogenic glycosides are typically stored in the vacuole of the plant cell where they do not harm the normal metabolic processes of the plant. However, if the vacuole membrane is broken due to damage or stresses (e.g., chewing, crushing, wilting, freezing), certain enzymes in the plant cell and micro-organisms in the ruminal stomach hydrolyze the glycosides into hydrogen cyanide (HCN) (2,4). Horses and pigs are less susceptible to poisoning since they have acidic stomachs, which inactivate these enzymes, preventing hydrolysis of the glycosides (4). Once free HCN is produced, it is toxic to both the plant cells in contact with the cyanide and to the animals that ingest it. This process is primarily a plant defence mechanism against herbivory and pathogen infection. Increased production and concentration of cyanogenic glycosides have been experimentally shown to occur within the plant during cool moist growing conditions, nitrate fertilization, low phosphorous soil levels, frost, drought and following application of 2, 4-D herbicides (2,4).

The choke cherry (*P. virginiana*) on average contains 143 mg of HCN per 100 g of fresh leaves. However, a sample of western choke cherry (*P. virginiana* var. *melanocarpa*) leaves was documented to contain as much as 368 mg of HCN per 100 g of fresh leaves (2,3). Ruminants consuming 0.25% of their body weight in these leaves are
likely to suffer fatal cyanide poisoning. This would mean that a 400-kg (880-lb.) cow would only have to consume 1 kg (2.2 lbs.) of fresh leaves (2). The black cherry (P. serotina) is considered the most dangerous of the eastern wild cherries and averages 212 mg of HCN per 100 g of leaves (3). At this concentration, 0.11 kg (¼ lb.) of leaves would kill a 45-kg (100-lb.) animal (3). The lethal dose of HCN for cattle and sheep is about 2.0 mg/kg of body weight (3, 4). For humans, the lethal dose is between 0.35 and 0.5 mg/kg (3, 4).

IDENTIFYING PRUNUS SPECIES
The most commonly found wild cherry species growing along livestock pastures in Ontario are choke cherry, pin cherry and black cherry (described below). Seeds from these species are often distributed by birds after eating the mature fruit, and depositing the seed while perched on pasture fences.

PRUNUS SPECIES COMMONLY FOUND IN ONTARIO

<table>
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<tr>
<th>Tree</th>
<th>Blossom</th>
<th>Leaf and/or Bark</th>
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**Choke Cherry**
*P. virginiana*
(“Shubert” cultivar with dark red-purple leaves)

The choke cherry is a coarse, conical, deciduous shrub or small tree that can reach 30 ft. tall and 25 ft. wide, and often suckers from the base (4, 7). The bark is gray and non-aromatic (4, 7). Leaves are ovate to obovate to elliptical in shape, sharply serrate along the margins, thin, mid to dark green, glossy and approximately 10 cm (4 in.) long (4, 7). The flowers develop in late spring, usually after the expanding of the leaves, appearing in dense racemes as small, white and cup-shaped (4, 7). The fruit is red/red-purple in colour, spherical and astringent (4, 7). In late summer and fall, the brown hairs on the underside of the leaf appear as tufts along the mid-vein where each side vein meets the mid-vein.

**Pin Cherry**
*P. pensylvanica*

The pin cherry, also called wild red cherry, bird cherry, and fire cherry, is typically a spreading deciduous tree or large shrub (up to 30 ft. high and 30 ft. wide), with smooth reddish-colour bark that is often peeling and red banded (4, 7). The leaves are oblong to lanceolate in shape, doubly serrate along the margins, with a bright green surface and rough underside (4, 7). The leaves are approximately 11 cm (4.5 in.) long and tend to turn yellow and/or red in fall (4, 7). The small, white, cup-shaped flowers on the pin cherry usually appear before or with the leaves, and present themselves in an umbel (4, 7). The fruit develops after the flower as small, red, spherical, drupes approximately ¼ inch in diameter (4).
The black cherry, also called wild rum cherry, is a deciduous, broadly columnar tree that can reach 60 ft. tall and 30 ft. wide (4, 7). The bark is rough and dark on the outer exposed surface while the inner bark is reddish and very aromatic (4, 7). The leaves of the black cherry are oblong to lanceolate in shape, serrate with incurving teeth along the margins; the upper surface is glossy and dark green while the underside is often lighter and duller (4, 7). The leaves often reach 13 cm (5 in.) in length and turn yellow and/or red in the fall (7). The small, white, fragrant, bowl-shaped flowers appear in racemes after the emergence of the leaves and mature into small red drupes that ripen to black (4, 7). In late summer and fall, there is a distinguishable solid line of brown hairs along the mid-vein on the underside of the leaf.

CLINICAL SIGNS
When toxic amounts of plant material are ingested, the signs of HCN intoxication can typically be seen within 10 to 20 minutes (1, 4). Initially, the animal will exhibit apprehension and distressed behaviour, which is quickly followed by staggering, gross muscle tremors, severe weakness, ataxia and rapid, laboured respiration (1, 4). Kicking and titanic-type seizures, as well as attempts to urinate, are also common in the earlier stages (1). The animal will then slow or stop breathing. A very slow heart rate may also be observed (1). Eventually the animal becomes comatose and there may be a period of brief paddling of the feet with convulsions before death (1). Cyanosis, the blue colouration that results from deoxygenated blood, is not a good sign of HCN poisoning since the blood remains red and well-oxygenated (1, 5). This colouration is observed because the oxygen release from haemoglobin to the cells is blocked (1, 5). This entire sequence of events can occur within 2 to 3 minutes or may take 1 to 2 hours, depending on the size of animal and dosage. However, if the animal survives the first hour, prognosis for recovery is usually favourable (1, 4).

In 2001, investigations into losses of foals and embryos in female horses, a syndrome now called Mare Reproductive Loss Syndrome (MRLS), originally implicated cyanide poisoning as the potential cause of
the problem. It was thought that tent caterpillars, which heavily contaminated pastures, feed troughs and water bowls, concentrated cyanide when they fed on leaves of *Prunus* species (Figure 2). The tent caterpillars were accidentally consumed by horses and were thought to result in absorption of cyanide (6). This theory has been replaced by other hypotheses.

**TREATMENT**

Treatment for cyanide intoxication is often too late since the disease usually progresses rapidly (1). The treatment is aimed at unblocking the cellular respiration. A solution consisting of sodium nitrite and thiosulfate (5 g of sodium nitrite, 15 g sodium thiosulfate in 200 mL of water for cattle and 1 g sodium nitrite, 3 g sodium thiosulfate in 50 mL of water for sheep) is recommended (1, 4). This antidote works best when administered intravenously, since it is not absorbed when given orally (1). Oral therapy with glucose, molasses, or glyceraldehyde may provide a benefit. These products act as slow antagonists by tying up free HCN into cyanhydrin. However, due to the slow nature of this detoxification, they should not be used as a primary treatment (1). They can be used in conjunction with the intravenous antidote or as a possible short-term preventative (1).

To prevent cyanide poisoning in livestock, consider the following. The pH in the stomach plays a major role in the hydrolysis of the cyanogenic glycoside (1). Lowering the stomach pH by feed management and supplements reduces the risks from cyanogenic forages (1). Sulphur supplements in salt can also be used to increase the rate of natural detoxification and resistance (1, 4).

It is extremely useful to identify all tree and shrub species growing near your house and livestock pastures in order to determine the safety or risk each may pose to livestock that may come into contact with them.

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**REFERENCES**


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Photos of Pin Cherry Blossom and Bark courtesy of Glen P. Lumis, University of Guelph, Guelph, Ontario