# *Cestrum Parqui* L'Herit (Green Cestrum) Toxicity in Cattle

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## Introduction

*Cestrum parqiu* L'Herit (green cestrum), is a perennial shrub which has become naturalized in Australia. *C. parqui* has been reported to be toxic to many species including cattle. Clinical signs of toxicity in cattle include included dullness, anorexia, increased heart rate, rumenal stasis, dehydration, constipation and a tendency to lie down. In severe cases staggering gait, generalised muscular tremors, lateral recumbency and terminal tonic and clonic convulsions are seen. This report details the clinical and pathological findings in which eight animals died of a herd of thirty five cattle, believed to been caused by *C.parqui* poisoning.

*Cestrum parqiu* L'Herit (green cestrum), is a perennial shrub native to Chile, South America. It has naturalized in Australia and a common weed in coastal Queensland south of the tropics and in Northeast New South Wales[1]. It has also been recorded in Victoria[2], and areas of South Australia[1, 3]. It is commonly found on rubbish dumps, vacant allotments, along fence lines, around farm buildings, under trees and shady creek banks[2]).

*C. parqui* has been reported to be toxic to many species including cattle[2]. Previous reports [4, 5] identified that the shrub causes fever, anorexia, abdominal pain, and haemorrhage into the gastrointestinal tract and many other body systems. Later studies has identified that the shrub primary site of injury is the liver [6-8]).
The following report documents the death of 7 cattle attributed to the consumption of *C. parqui* .

**Abbreviations** DPI, Department of Primary Industries; AST, aspartate transaminase; PT, Prothombin time

## Case report

The clinical cases occurred on a dairy property in Northern Victoria. A mob of 35 cattle, consisting of rising 2 year old pregnant Holstein Frisian heifers, cross bred heifers, beef heifers and steers and a young Holstein Friesian bull. These animals were moved on to vacant out block property to clean up around an old farm house, sheds and rubbish dumps.

The pasture around the sheds consisted predominately of mature ryegrass, kikuya, old tobacco plants and an unidentified shrub that grew under pepper corn trees.

The veterinarian was called to the property on the morning of Sunday 13th May, to examine and investigate four heifers that had died suddenly overnight. The stock were observed the evening before and all appeared to be in good health.

Of the heifers that died, 3 were cross bred and one beef, identified as #01201, #00942, #01202 and baldy heifer, respectively. It was advised that the remaining stock be moved off that paddock prior to the veterinarians' arrival.

An examination of the carcasses was performed. Three of the four carcasses had been scavenged. All heifers were bloated in lateral recumbency, and the environment around them did not appear to be disturbed. All heifers had defecated loose brown faeces prior to death, three heifers had clotted blood from rectum and one heifer had clotted blood from eyes and nose.

A walk of the property and inspection of the remaining stock was conducted and all stock appeared to be unaffected. It was noted in the surrounding environment there was an old farm house with lead based paint, a number of rubbish deposits beside a machinery shed and the unidentified shrub, with yellow flowers (Figure 1a) and black berries (Figure 1b), had clearly been eaten and was present along the fence line, around the shed and under the peppercorn trees.

A field necropsy was carried out on the 17 month old heifer, #01201. Findings revealed clotted blood from rectum, eyes and on muzzle, grossly distended rumen with gas and mixed plant material (pH of 7), small intestines were grossly distended however contents appeared to be normal, liver


Figure 1. *a) Yellow flowers b) berries of the green cestrum shrub*

grossly enlarged and congested, the spleens capsule had paint brush haemorrhages present and meaty on cut section. There was some interstitial emphysema and congestion present in some areas of the lungs however predominantly normal and the heart had ecchymotic haemorrhages over the pericardium and endocardium. It was also found that the heifer was 18 weeks pregnant. The following samples were taken, aqueous humor, plain blood, ingesta, urine, faeces, rumen fluid, fixed and fresh tissue samples of liver, kidneys, spleen, lung and heart. Pasture samples and samples from the unidentified shrub were taken.

Differentials considered at this stage included, lead toxicity, kikuyu grass toxicity, nitrate poisoning, metabolic disease, cardiac glycoside toxicity and toxicity from the unidentified shrub. A number of tests were conducted at the veterinary practice. The aqueous humor and plant material were tested for nitrates and it was negative, faecal examination was unremarkable and the urine pH was 8. The unidentified

shrub was later identified as *cestrum parqui* (green cestrum) by T. Lonsdale of the Department of Primary Industries (DPI).

Later that morning the farmer sought further veterinary consultation for a cross bred heifer # 1563, which was down and showing clinical signs of dypsnoea. On examination the heifer was in lateral recumbency,
respiratory rate was elevated with harsh lung sounds, elevated heart rate (160 beats per minute) and the temperature was 38.4 degrees Celsius. The heifer then begun to paddle, appeared to be hyperaesthetic and demonstrated an expiratory grunt. Administration of the following treatments was performed, 200g of activated charcoal orally via a stomach tube, 500mls of 4 in 1 intravenously (24.5g/L calcium borogluconate, 4.7g/L magnesium, 12.1g/L hypophosphite, 181.5g glucose) and 1250mg of vitamin B1 intravenously. The heifer then displayed tonic and clonic convulsions, opistotonous and died several minutes later. The remaining cattle were yarded and all but 3 were drenched orally with approximately 200 grams of activated charcoal made into a mixture. One beef heifer went down on the track with respiratory difficulties, muscle tremors and then became very aggressive.

Over the next 72 hours the farmer reported animals that appeared dull, anorexic and several having a staggering gait. During this time another 2 Friesian heifers and 1 beef steer died. Five days after the initial incident the farmer reported that no more further deaths had occurred and the remaining cattle appear to be healthy and showing no clinical signs. A total of 8 cattle died from the original 35. The estimated cost to the producer was $12,500.

Laboratory results revealed that the kidney tissue lead levels were <10 µmol/kg wet weight, considered within the normal reference values of the laboratory, and bacterial cultures of blood, liver and lungs were unremarkable. Histopathology was performed on liver, spleen, heart, lung and kidney. The histopathology results revealed marked and uniform pooling of blood in periacinar areas accompanying bridging necrosis of periacinar hepatocytes (Figure 2), apoptotic changes were present in many leukocytes in the spleen, subepicardial haemorrhages and unremarkable changes present on the lung and kidney samples.

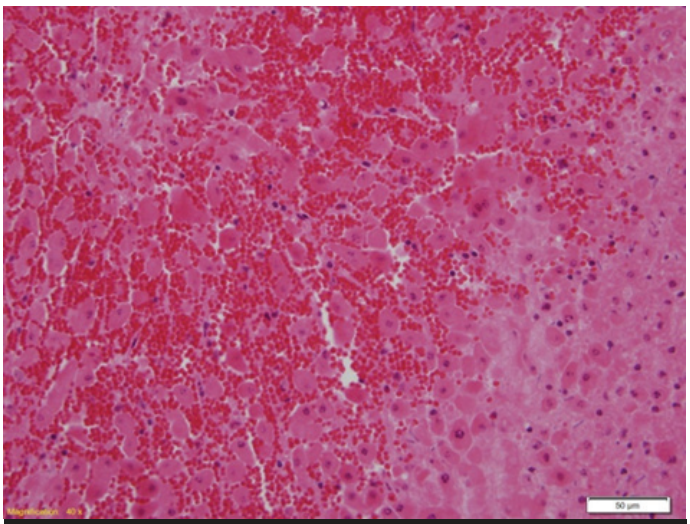

**Figure 2.** ***Heifer #01201. Liver showing necrosis and loss of periacinar (centrolobular) hepatocytes and pooling of blood into these areas (H & E, x 40).***

The diagnosis that was made was acute hepatic failure due to severe hepatic necrosis consistent with green cestrum poisoning. The haemorrhaging from the gastrointestinal tract was attributed to depletion of clotting factors in acute hepatic failure, by the veterinary pathologist.

**Discussion**

*Cestrum parqui* is an upright woody shrub with few to many stems growing from the base, leaves are yellow with a green tinge spearhead shaped that alternate on the stem. The flowers are yellow, tubular and usually are in clusters. The fruits of the shrub are shiny purple to black, egg-shaped fleshy berries [1, 2, 9]. The flowering and fruit seasons are in summer and autumn.

*C.parqui* contains parquin and carboxyparquin, which both are diterpenoid (kaurene) glycosides. All part of the plant contain these toxins[9, 10]. The primary lesion in poisoned cattle is severe periacinar coagulation necrosis of hepatocytes [6, 8, 11]. There are other plant species that produce similar lesions and those genera include Trema, Xanthium, Myoporum and Hetia as well as poisoning from cyanobacteria [9, 10, 12]. *C. parqui* plants are considered unpalatable, however hungry cattle will consume the plant, generally when feed is scant, such as in a drought. Poisoning cases have occurred during all months of the year in Australia[9].

McLennan *et al* experimentally demonstrated *C. parqui* toxicity in cattle. One animal dosed orally with 30g (wet weight) of fresh plant developed mild signs initially within 24 hours. These clinical signs included dullness, anorexia, increased heart rate, rumenal stasis, dehydration, constipation and a tendency to lie down. However this animal died 26 hours after the onset of mild clinical signs. With the last 3 hours before death the animal displayed a staggering gait, generalised muscular tremors, lateral recumbency and terminal tonic and clonic convulsions[8]. The latter clinical signs are consistent with the cross bred heifer # 1563 that died when receiving veterinary attention.

In the same experiment McLennan *et al* also orally dosed two other animals with 11 and 17 g of plant / kg body weight. These animals also showed signs of dullness, anorexia, increased heart rate, rumenal stasis, dehydration, constipation, normal body temperatures and a tendency to lie down. However these animals recovered within 28 hours [8]. In this outbreak the farmer did report other affected animals displaying signs of dullness, anorexia and a staggering gait, of which 2 animals died.

The clinical signs that were observed in this outbreak were consistent with those poisoning by Lavers, Lopez and McLennan *et al*. However as mentioned previously the body temperature of the heifer that died remained normal, which was observed by McLennan *et al*. However Lavers described that the body temperature was increased in the animals affected and Lopez described the body temperature to be sub-normal.

Lopez et al also reported that a dose of 29 g of fresh plant / kg was capable of causing death and that central nervous irritation was a pronounced feature. However the beef heifer that went down on the track displaying signs of respiratory difficulties, muscle tremors and aggression made a full recovery.

McLennan *et al* describe plasma biochemical and hematological changes in the animals that survived and the

animal that died from the poisoning. The main biochemical changes included elevations in aspartate transaminase (AST) values, increase in pro-thrombin (PT) times, total bilirubin was moderately elevated in all animals and the conjugated bilirubin increased substantially only in the animal that died. These biochemical changes are consistent with liver damage which is a major feature in cattle poisoned with *C. parqui*. The only hematological change noted was the total leucocytes count increased only in the animal that died, however it occurred shortly before death. It was also noted that the values for fibrin degradation products were unaffected[8].

The gross hepatic changes of slightly swollen pale liver described by McLennan *et al* [8]were similar to those observed in the heifer #01201 that the post mortem was conducted on. Brevis *et al* describe the gross liver changes as a reddish-orange and "nutmeg" appearance. Bervis *et al* also noted that the gallbladder wall was oedeamatous and contained hemorrhages[11].

Other gross pathology lesions found by McLennan *et al* included sub-serosal haemorrhages in the thoracic aorta and coronary vessels[8]. Whereas Brevis *et al* described a more generalized effect of hemorrhages in the pericardium, epicardium, endocardium, intestine, thymus and skeletal muscle and also found congestion and oedema of the lungs [11]. In the post mortem of the heifer #01201, there appeared to be a more generalized affect, similar to that described by Brevis et al. The haemorrhagic changes in the gastrointestinal tract may be due to irritation of the mucosa by the toxin or its metabolites[8]. Haemorrahage elsewhere may be contributed to a coagulopathy due to hepatic failure and increased consumption and decreased production of clotting factors[8]. One feature on this outbreak was that three of the four heifers had clotted blood from rectum and one of the heifers had clotted blood from eyes and nose, presumably due to the same mechanism mentioned above. This clinical feature was also consistent with Lavers findings [5].

McLennan *et al* found that the only histologically significant change was in the liver. In the liver of the animals that recovered, there was periacinar necrosis with neutrophil and macrophage infiltration. The liver changes were similar in the animal that died however there was more of a haemorrhagic component and lacked any appreciable cellular reaction. Brevis *et al* described the histological changes of the liver as centrilobular necroses and irregular vacuolation of the cytoplasm by peripheral hepatocytes of the affected areas. It was also found that there was a slight proliferation of the biliary ducts[11]. In the post mortem of the heifer #01201, the liver histological changes resembled components of both McLennan *et al* and Brevis *et al*.

McLennan *et al* conclude that the differences in gross pathology changes and hepatic changes may be due to material used in Brevis et al study had a higher toxin content [8]. However according to all studies and also the described clinical case hepatic failure as the cause of death with hepatic periacinar necrosis [5-8, 11].

Treatment for affected animals is often unrewarding and there is no specific treatment available. However general decontamination measures are recommended[9].

Prevention of *C. parqui* poisonings is to inhibit animal access to the shrub and farmer awareness of the poisonous shrub. The shrub can easily be destroyed either by physical removal or chemical treatment,[2] such as a mixture of 250g/L Amitrole and 220g/L Ammonium thiocyanate[13] or 300g/L 2,4-D and 75g/L picloram present as the triisopropanolamine salt[14]. For either method reoccurrence is likely and it may be necessary to perform further treatments[2].

It may be recommended not to graze cattle or livestock in areas around old farm buildings, sheds or rubbish dumps due to the potential risk of poisonings, not only in this case of *C. parqui* but other would-be poisonous plants or toxins.

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