

Lead Toxicity in Cattle

Lead toxicity in cattle is one of the most common forms of poisoning seen in cattle practice.

The common sources of lead which cattle gain access to include lead paint (from old buildings), lead plumbing materials, lead-acid batteries and waste motor oil. In regions with high industrial pollution levels, gradual poisoning is also possible.

Calves (<6 months of age) are most frequently affected by lead poisoning due to their inquisitive nature.

Once lead is ingested it sits in animal's reticulum (stomach 1) and is progressively transformed into toxic salts which is then absorbed into the blood stream. Once absorbed into the blood stream it is transported around the body which:

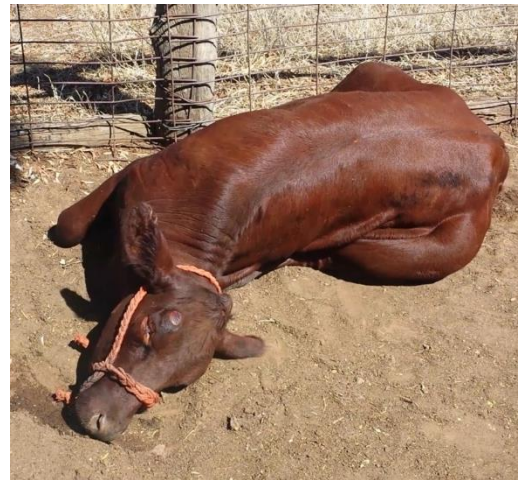
- Damages nervous tissues (including the brain cells)
- Damages internal organs (particularly the liver and kidneys),
- Damages bone marrow and red blood cells (leading to anaemia).

Lead ingestion also can have an impact on fertility and can result in abortions and foetal death.

The clinical signs observed in cattle that have ingested lead vary based on the source and the amount consumed. Some animals will be found dead and others will develop a chronic illness with poor performance.

In acute lead poisoning cases, the clinical signs observed include:

- Sudden death/ found dead
- Depression
- Lethargy
- Loss of appetite
- Apparent blindness
- Diarrhoea
- Stagers
- Excitable
- Unusual behaviours including grinding teeth, jaw chomping, bobbing head, twitching eyes/ ears



- Frothing at mouth
- Muscle tremors
- Convulsions

In chronic poisoning cases, the initial clinical signs observed include weakness, lethargy, anorexia, weight loss, diarrhoea and anaemia, which then may progress to the acute lead poisoning clinical signs outlined above.

Diagnosis of lead poisoning in the live animal is often made based on history of access to a lead product, the typical clinical signs observed and whole blood-lead concentrations. At post-mortem, lead compounds may be found in the gastrointestinal tract, otherwise testing for the lead level in the kidneys and liver is diagnostic.

If you suspect lead poisoning in a group of cattle, the first thing to do is to move the group from the suspected lead poisoning location.

In acute poisoning cases, treatment is rarely successful. If treatment is elected, Thiamine hydrochloride (vitamin B1) injections are utilized to reduced lead deposition in tissues and assist in binding and elimination of lead from the body. Other supportive treatments include oral drenching with activated charcoal and magnesium sulphate (Epsom salt) to bind lead in the gastrointestinal tract. The chelating agent Calcium-EDTA has been used IV but often it's use is prohibited due to it often being unavailable and it is expense.

The most important prevention is to prohibit access to lead-based products and batteries on farm.

Lead poisoning is a notifiable disease under section 7 of the Livestock Disease Control Act 1994 (LDCA), therefore any suspected cases must be reported to the Victorian Chief Veterinary Officer within 7 days. The meat and milk from cattle that have experience lead poisoning or have been exposed to lead are not fit for human consumption. Under Agriculture Victoria's policy for the management of lead residues in grazing livestock, all at risk animal are blood tested to assess level of exposure to lead. There may be restrictions put in place to prevent the sale of exposed livestock. Usually, exposed animals will have repeated blood tests over time until the level of lead in the blood drops below the permitted maximum limit. After, this time the animals maybe sold for slaughter but parts of there offal will be prevented from entering the food chain.

Reference material:

Agriculture Victoria. 2022. Lead exposure and poisoning in livestock. Accessed 7th July 2022, from: <https://agriculture.vic.gov.au/livestock-and-animals/livestock-health-and-welfare/lead-exposure-and-poisoning-in-livestock>

Parkinson T J, Vermunt J J & Malmö J (2019) Diseases of Cattle in Australasia: A Comprehensive Textbook. New Zealand Veterinary Association Foundation for Continuing Education, USA. pp 442-445 & 665-685.