

Botulism in Cattle in the Northern Territory

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Botulism has a rapid onset and is usually a fatal disease of livestock in the northern regions of Australia, particularly in areas where pastures experience periods of protein and phosphorus deficiency. It is caused by the botulinum toxin produced by the bacterium *Clostridium botulinum*.

Before the widespread use of vaccination, botulism killed significant numbers of stock in the northern beef industry. In 2010, 78% of Northern Territory (NT) producers vaccinated all their cattle against botulism.¹

Although botulism usually affects unvaccinated herds, it can also infect improperly vaccinated herds. Losses of up to 25% due to botulism have been reported in some herds. The disease may also cause persistent but undetected low losses, which may reach 10 to 20% annually.



RISK TO PEOPLE

While humans can be infected by botulism, handling infected cattle does not usually pose a risk.

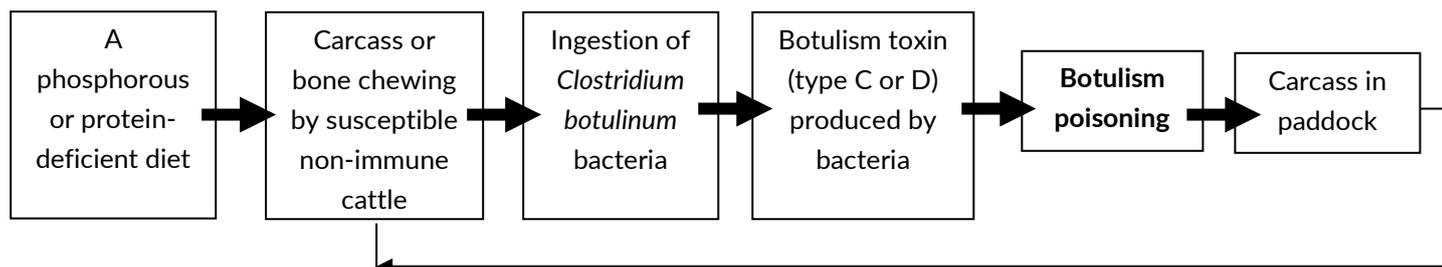
CAUSE

Clostridium botulinum is a bacteria commonly found in soil, water and marine sediments around the world. It is also a normal inhabitant of the intestinal tract of many healthy horses, cattle and poultry. The bacterium thrives in decaying animal or plant material and produces spores, which can survive in the environment for a long time. The spores germinate in moist, low-oxygen environments, such as rotting carcasses or decaying organic material. Under favourable conditions, they proliferate and produce botulinum toxin, which, if consumed, leads to intoxication.

There are several forms of the toxin, which are designated from botulinum A to G. However, almost all botulism in cattle in Australia is caused by botulinum C and D. The toxin is extremely potent and minute doses can be lethal. The toxin interferes with the interaction between nerves and muscles, causing paralysis. All species, including humans, can be affected although there is a wide variation in sensitivity and cattle are some of the most susceptible species.

Bones and carrion of decaying cattle and fly maggots are the main sources of the toxin. Infection has also been known to occur by drinking water or consuming feed that has been contaminated by animal carcasses and rotting feed (mouldy or decomposing hay or silage). Another form of botulism, known as toxic infectious botulism, occurs when animals consume actively-growing bacteria from the gastrointestinal tracts of cattle where the toxin is subsequently produced.

The chain of events most commonly necessary for botulism poisoning to occur in NT cattle is as follows:



An outbreak of botulism is likely to occur when unvaccinated cattle are kept on a protein and phosphorus-deficient diet, and where carcass chewing is common.

SUSCEPTIBILITY

The susceptibility of cattle to botulism in northern Australia depends on the following six factors:

1. Protein and phosphorous deficiency. Lactating cows and growing cattle have a higher need for protein and phosphorous. If such animals cannot obtain these needs from the feed, they will often develop a depraved appetite for carrion and bone chewing. The availability of phosphorous will vary depending on the soil type and seasonal conditions. Most NT soils are either phosphorous-deficient or are marginal. Even green pastures in the mid to late season are likely to be phosphorous-deficient. The protein in native pastures is often insufficient to maintain live-weight during much of the dry season. While 80% of NT properties provide some supplements¹, the cost and practical problems of supplementation reduce its effectiveness.

2. Carcass and bone chewing. This is commonly seen in the NT. Once cattle develop this habit, they may continue to chew bones even when dietary protein and phosphorous are adequate. Adequate supplementation will not cure this problem in deficient areas.

3. Bacterial distribution. Bacteria producing type C and type D toxins are present in all pastoral regions of the NT.

4. Toxin. Cattle are very susceptible to the botulism toxin. Toxin production occurs in an anaerobic environment, with moisture and an optimum temperature of around 23 °C (15-35 °C). All these conditions can be found in a rotting carcass. The toxin can last for a year at 30 °C and is rapidly inactivated at 37 °C. Therefore, the amount of available toxin is not constant. Although not all rotting carcasses are necessarily toxic, the proportion of toxic carcasses in tropical environments can be very high.

5. Access to carcasses. All decaying carcasses are potentially infective, including those of cattle, horses, donkeys, pigs, birds, wallabies and rodents.

6. Susceptible cattle. Unvaccinated and improperly vaccinated cattle can be assumed to be fully susceptible. Even properly vaccinated cattle can succumb if the amount of toxin is high enough to overpower the body's immune system. Natural immunity can develop in cattle that have been exposed to the natural toxin and have recovered.

SIGNS

Cattle of all ages can be affected by botulism. The toxin binds to nerve endings and prevents nerve impulses to muscles. This leads to a floppy or flaccid paralysis, which usually progresses throughout the body. Typical signs include hindlimb weakness progressing to paralysis and collapse (downer cattle). Other signs include paralysis of the



muscles of the face, jaw or tongue, inability to eat or drink and drooling. Death usually results from respiratory failure or between one and four days following the onset of clinical symptoms, but can take up to 14 days.

The development of signs and the progress of the disease depend on the amount of toxin ingested. Signs are usually seen within two to six days after consuming contaminated material. In an infected herd, most deaths will occur within the first week. However, this can continue for up to three weeks. In unvaccinated extensive pastoral herds, botulism may be suspected when unexpected numbers of cattle are found dead or are missing at mustering.

DIAGNOSIS

Diagnosis of botulism is largely based on clinical signs and on exclusion of other causes. In some cases, where the animal is still alive, the tongue will remain extended when pulled. However, this is not always observed. If a post-mortem is conducted, evidence of carrion consumption may be found in the rumen or reticulum (honeycomb) in the form of hide, bone or maggot pupae.



Botulinum toxin levels in the blood of clinically affected cattle are usually too low to be detected with current laboratory tests. Antibody testing of surviving exposed cattle during and after an outbreak of the disease is of limited value and is mainly used to assess the status of vaccination.

TREATMENT

Once an animal has absorbed the toxin, there is nothing that can be done to improve recovery. Most cases develop quickly and death nearly always follows. Cattle that are mildly affected may recover with good nursing and veterinary care. Very early cases may respond to purging of the intestinal tract to remove the toxin. If nursing is attempted, the animal must not be drenched through the mouth as it cannot swallow.

Most cattle affected by botulism die of respiratory failure. However, complications can develop in cattle that cannot stand and sometimes the kindest option may be euthanasia.

During an outbreak, vaccination may reduce the potentially harmful effects of consumed bacteria that may release toxin. However, vaccination is not effective in cattle that have consumed the toxin. It is also important to prevent further access to skeletons, carcasses, rubbish tips and burn piles during an outbreak.

PREVENTION

Three steps are recommended for the prevention of botulism poisoning:

1. Vaccination with bivalent (type C and D) botulism vaccine following a recommended program.
2. Supplementary feeding of cattle with phosphorous and protein.
3. Removal of all carcasses and bones.

Vaccination

Vaccination with the bivalent (type C and D) botulism vaccine is the most effective long-term prevention strategy. A range of botulism vaccines is available and involves either an initial two-shot program, one month apart, or a single shot followed by an annual booster shot. An alternative long-acting vaccine consists of an initial single shot followed by a booster shot every three years. Both vaccines provide a similar level of protection. Which vaccine to select will depend on cost and management practices. All vaccines require booster shots to maintain immunity.

Calves can be vaccinated from one month of age to produce effective immunity. Properties that conduct early weaning (60 kg+) should ensure that calves are vaccinated at this early age. Protective maternal antibody levels are depleted by six months of age.

Vaccination failure

Vaccine failures do occur and it is important to store and administer the vaccine carefully. In 2015, a survey of 19 NT properties found that protection in cattle vaccinated against botulism varied from 0% to 100%, with an average of only 67%. This means that over 30% of the supposedly vaccinated animals were not protected against botulism.

The factors which may prevent adequate protection after vaccination include stress at vaccination, a poor vaccination procedure, inadequate attention to the 'cold chain' process, the choice of vaccine, cattle missed in mustering, lack of vaccine boosters, concurrent diseases, such as pestivirus, poor nutritional status of the animals and stock being vaccinated too young (antibodies transferred from the cow, called maternal antibodies, can interfere with vaccination). Often, the vaccination history of imported cattle is overlooked. Botulism vaccination should therefore take place along with other vaccinations before cattle are released into the herd.

To reduce the risk of vaccine failure, you should follow all manufacturer instructions prior to use, such as storage, handling and vaccination recommendations. Station storage of vaccine should be in a cool room at 4 °C. Vaccine should be kept in an esky with ice in the yards. Vaccine should be injected under the skin on the neck or behind the shoulder. It is important to avoid sites close to the rumen, particularly the rumen fossa (triangular area under the hip) as the vaccine is destroyed if injected into the rumen.

Supplementation

Supplementation with non-protein nitrogen (e.g. urea) and phosphorous is a recognised management practice. However, even the best supplementation programs will not completely prevent carcass or bone chewing.

Carcass removal

The removal of carcasses is not always an option under extensive range conditions where paddocks are large and the checking of stock is infrequent. However, the removal of all carcasses from areas of stock congregation, such as watering points, is important. Carcasses can be burnt, buried, locked up in the turkey nest enclosure or at least taken a considerable distance away.

ACKNOWLEDGMENTS

This Agnote updates and replaces the original produced by Kevin DeWitte in 1996.

¹ Cowley, T., Oxley, T. MacDonald, N. Cameron, A. G., Conradie, P., Collier, C. and Norwood, D. (2015). The 2010 Pastoral Industry Survey – Northern Territory Wide. Northern Territory Government, Australia

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