Clostridial disease in beet grazing systems

A new syndrome of deaths has emerged among beef cattle grazed on beet. **Jim Gibbs**, Senior Lecturer in Livestock Health and Production at Lincoln University, suggests some potential causes.

NEW ZEALAND CATTLE and sheep production systems are based almost exclusively on pasture. While dairy operations use appreciable supplementary feeds in addition to pasture, this is not true of the beef and sheep industries, with only one genuine exception – winter crop feeding in the south. Crop use in beef systems is commonly for cattle who are fattening for slaughter.

In recent years, more attention has been given to accelerated-feeding forage systems for young stock, driven by meat processors incentivising for greater continuity of supply across seasons, and the difficulties of marketing pasture-fed beef internationally when a carcass could be anywhere from 18 months to four years old. While several Gramineae forage systems can achieve winter growth liveweights suitable for a slaughter weight at 18 months, the Brassicaceae cannot, and lower dry matter (DM) yields per hectare and poor utilisation rates in wet, cold season weather limit most Gramineae finishing systems' profitability.

Fodder beet (Amaranthaceae) grazing systems have been developed in New Zealand in the past decade (Gibbs, 2011). The crop has emerged as the sole forage genuinely suited to use in beef finishing systems, and it has also grown as a ewe and hogget wintering feed. Crop features suit forage finishing systems: a high and stable metabolisable energy (ME) (12 MJ/kg DM); suitable protein content (11–13%); a high sugar content (c. 50% of DM); yields up to 40t DM/hectare in New Zealand; spring sown for autumn/winter use; palatable and straightforward to feed; and demonstrated profitability on farm because unusually high energy intakes can be achieved. As a result, it has been the fastest-uptake forage in New Zealand history.

Animal health impacts with appropriately managed beet grazing systems are very low, with published mortality and morbidity rates in beef <1% over 200 days (Gibbs et al., 2015). Rumen acidosis at introduction to the crop is the principal disease to be managed (Gibbs and Saldias, 2014). However, as a novel system, new disease syndromes are not unexpected, and the rise of clostridial deaths as the primary health challenge has emerged as wider farmer understanding of acidosis management has all but abolished that as a crop feature. Across New Zealand, sheep and cattle operations grazing the crop have had documented clostridial deaths.

In sheep, vaccination with standard five-in-one proprietary clostridial preparations prior to entry has proved effective, while unvaccinated young stock can have significant mortality (>1%) in the first two weeks. Persistent deaths after careful vaccination are not a feature of either lamb or hogget operations on beet beyond this period, so management has proved simple and uptake has been strong.

However, in beef finishing systems on beet, it appears a new syndrome of deaths has been emerging. While sporadic deaths on beet of single animals across the 130-day autumn and winter season are not uncommon in herds vaccinated with either various five-in-one or Covexin 10 (10-in-one), with no discernible improvement in mortality rate in a group of the larger beef operations in New Zealand, in the past three years seven larger operations have reported a different pattern of clustered, multiple deaths. Only one farm was a wintering dairy cow herd, and young (weaners) comprised more than 75% of all deaths on the others.

These operations are always experienced in beet feeding and have invested heavily in these systems, and therefore are achieving close to maximum intakes by assiduous grazing management and supplement use. I have never observed this pattern of deaths in recent-entry beet feeders. The deaths appear several weeks after transition to the crop is completed, which represents closely the time to achieve both maximum intake capacity and renal adaptation to the extreme water loads (>100mls urine/kg liveweight) (Prendergast and Gibbs, 2015; Jayasinghe and Gibbs, 2018).

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The only clinical sign is sudden death, typically in larger, better-condition steers, and the deaths are often clustered in both time and space. Gross postmortem changes I have observed tally with those reported from various practices, with minimal tissue haemorrhage or pleural, pericardial or peritoneal fluid, but rapid diffuse tissue oedema and putrefaction. Enteritis or the presence of fibrin adhesions has not been an identified feature. While glycosuria is reported as pathognomic in Type D intoxication, it is a difficult assessment with urine volumes two to three times above reference ranges, and is typically unrewarding. Only one animal from more than 50 deaths was observed by an owner as ill before death – a cow, who then died without convulsions inside two minutes after a mild epistaxis and collapse.

In every case, the deaths stopped approximately three to four weeks after they began, independent of any intervention. Within that period, neither re-vaccination nor altered diet – typically increased supplement – reduced the deaths. Affected farms included those feeding mineral licks and those that did not. The total mortality rates observed in the prescribed periods ranged from 2% to 4%. However, given the large herd sizes on these farms, those rates could amount to several dozen dead animals, so they were naturally alarming for the owners.

On three farms, a very similar pattern of deaths was observed in two consecutive years. In each case, local veterinary advice was to use Covexin 10-in-one in the second year rather than the five-in-one (from various manufacturers) used in the first year. On two farms, a three-shot programme with two-week interval was given before entry. In both cases, second-year deaths exceeded those of the previous year, with similar stock entering the crop, and the

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same beet cultivars and feeding regime. Interestingly, the dominant beef beet cultivar (Brigadier) typically associated with highest stock performance on beet, was never present on affected farms; in every case, medium DM cultivars were used, with correspondingly lower liveweight gains. So these deaths were not associated with elite performance farms, albeit with high performers.

It is worth sketching out why these deaths are likely to be *Clostridium perfringens* Type D intoxications secondary to intestinal overgrowth, rather than *C. sordellii*, which has sometimes been claimed.

C. perfringens as a group is documented as producing more than two dozen toxins, and Type D intoxication includes at least three independent toxins, with separate but overlapping pathophysiology, each with variants between strains, and with differing effects between bovine, ovine and caprine stock. The best evidenced pathophysiology of Type D disease initiation is intestinal overgrowth of *C. perfringens* populations secondary to changed diets, where high energy intakes are a feature. *C. perfringens* populations increase growth and activity, and reduce sporulation in environments of high sugars, including glucose and fructose, and these substrates are typically increased in diets where disease has been observed. It is notable that there exists considerable confusion across time and geography on both the class of species and pathologies expected with *C. perfringens* toxin-derived disease, so caution is required in interpreting the older literature.

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While there has been significant recent work in isolating and typing these toxin-proteins, the production of antibodies from any supplied antigen in a vaccine will not entirely represent the spectrum that stock are challenged with in the field. This appears to be the case with *C. perfringens*. The considerable cross-specificity documented strengthens the efficacy of available clostridial vaccines, but beet grazing systems represent an unusual challenge.

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Strip grazing, and the peculiar intake patterns seen with beet, ensure that animals spend about six hours daily in a small area at the hot wire (Saldias and Gibbs, 2016), far longer than comparable crop use, and faecal contamination of grazed feed is therefore high. However, utilisation of the crop is unusually high (Saldias and Gibbs, 2016), so faecal intakes are likely to be high also. Winter crop feeding is associated with high soil intakes, ensuring soil clostridial spores are also consumed. Beet intakes are higher than comparable winter feeds (Prendergast and Gibbs, 2015), and because the crop has low (6-15%) DM, unusually high water loads 'eaten' are a feature. Recent studies have shown that peak intake is achieved after about a month on the crop (Jayasinghe and Gibbs, 2018).

This intake of water has many effects, but extreme rumen passage rates are a key feature (Prendergast and Gibbs, 2015). As beet has a uniquely high sucrose content, ample sugars are spilled into the small intestines with the fluid passage, presumably with both faecal clostridium and soil spores. These odd features appear to lend themselves to *C. perfringens* Type D toxin production in a manner that is not achieved with other feeds, with only a few exceptions (lambs on lucerne or clover, as examples).

For most beet grazing animals, any overgrowth of *C. perfringens* in these circumstances, and the variation in antigen this presents, is likely managed by the cross-specificity of vaccination. However, the challenge is so high that some are presumably overwhelmed and present as clinical cases. It may be that the deaths eventually stop due to the natural process of improved immunity from exposure to these presumably variant toxin antigens. The timeframe seems to support this: with no change to any component of the system, deaths cease after about four weeks.

The usual question I'm asked is how to prevent these deaths. I am persuaded by experience and the available literature that extra vaccination approaches are not effective. Cereal-grain-fed finishing cattle in a US study – as close as New Zealand will get to a high ME intake equivalent – did not have a reduced death rate (0.24% across 90-day feeding) with additional entry vaccination for *C. sordellii* or *C. perfringens* Type C and D after the standard programme (De Groot et al., 1997).

Further, there is no reliable evidence in the literature that demonstrates a protective effect against sudden death syndrome of multiple vaccination beyond two administrations with multi-vaccines for clostridial disease.

Commonly, some reference to rumen acidosis is also claimed, and roughage supplement increases have been used in a number of cases, to no effect. This is no surprise, for two reasons. First, rumen pH is not associated with clostridial disease at all. Second, even if it were, rumen function in unrestricted *ad libitum*-fed cattle with <20% supplement has been repeatedly studied for a decade now, and almost two dozen experiments have consistently demonstrated with in situ assessments that rumen pH is stubbornly high (Gibbs et al., 2018). Intake pattern, not daily ME intake, governs rumen pH. Restrict feeding beet encourages competitive feeding and always crashes rumen pH, but few experienced beet operators do this anyway. Additionally, as supplement changes are typically made well into the beet grazing period when intake patterns are firmly set, supplement increases are taken up by a small proportion of the herd, leaving the high-challenge cattle unchanged. So, for several robust reasons this approach has been a complete failure in altering death rates.

What does work? One approach that requires more assessment but has worked is changing the grazing sites, and then waiting it out (the deaths always stop). It is noted above that often deaths occur clustered in location. In the admittedly small number of cases seen to date, at least some appeared associated with paddock areas. Certain sites (eg, stock camps) will have higher clostridial spore levels. In the balance of factors that tip animals into disease, avoiding grazing them at critical windows may be effective, and can be done rapidly without upending the system.

A future investigation that seems warranted is to look at the value of vaccines with expanded antigen variation. Also, the association of certain crop cultivars with the presence or absence of this disease syndrome in beet grazing stock may represent a longer-term solution, although at present no explanation for any such association is available. Finally, targeted histopathological examinations of tissues used in research applications of clostridial intoxication, such as cerebral oedema, may assist in providing a clearer picture of this new syndrome. VS

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