Lameness in Newly Arrived Feedlot Calves

Project Title: Investigating P3 Necrosis in Feedlot Cattle

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Background

Lameness is the second most costly feedlot health issue after bovine respiratory disease. Aside from treatment and death losses, lame cattle eat less, grow less, convert feed to gain less efficiently, and are more prone to transport injuries. Lameness is also a significant animal welfare concern and has been incorporated into some on-farm welfare audit systems.

There are many different types and causes of lameness, ranging between genetics (e.g. conformation), nutrition (e.g. founder), the environment (e.g. frostbite), injuries and infection (e.g. footrot, hairy heel wart). Some types of lameness may have several causes, like toe tip necrosis syndrome (TTNS).

TTNS is a specific type of lameness that affects the outside claw of the hindfoot, and typically occurs within one to seven days after arriving at the feedlot. The coronary band at the top of the hoof splits, and the hoof wall often sloughs off after two to four weeks. Very excitable cattle may be more prone to this condition when they damage the soles of their feet while struggling in the chute, but cause and effect may be difficult to separate. It is unclear whether struggling causes the initial injury, or if animals struggle because the foot is already in pain. The disease is sporadic, but tends to cluster by truckload and feedlot pen. Extreme lameness results in a rapid loss in body condition and poor performance. Approximately half of the animals will recover; the remainder are euthanized because of poor performance and concerns about animal welfare.

TTNS is also referred to as P3 necrosis because the infection occurs in the corium surrounding the third phalangeal (i.e. P3) bone at the tip of the hoof. The P3 is comparable to the third bone at the end of a human finger.
Objectives

To identify the cause(s) of toe tip necrosis and develop a strategy to prevent and/or control it.

What they did

In October and November 2012, three commercial veterinary practices collected hind feet from 67 feeder heifers and steers (averaging 385 to 700 lbs) that died or were euthanized in commercial feedlots (on average within three weeks of feedlot arrival) and submitted them to the research team. For each TTNS-affected animal, hooves from an unaffected control animal were also collected.

At the university, researchers who were “blind” to whether an animal was diagnosed with TTNS analyzed 39 of the hind feet for presence or absence of apical white line separation (AWLS). Each hoof was then sliced lengthwise into thin sections. The location, nature and severity of the lesions were described and compared between TTNS-affected and control hooves.

Hooves from 6 affected cases that were not used in the above study were sliced lengthwise and photographed. Lines were drawn to subdivide the foot and which subdivision the lesions occurred in was recorded. The section with the most lesions was examined under a microscope for signs of inflammation.

What they learned

The white line between the hoof and sole had separated in all of the TTNS-affected hooves, but in only 3% of control hooves. The white line was also significantly thinner in TTNS-affected hooves than in control hooves. In TTNS-affected hooves, tissue in the toe tip was always visibly inflamed, the center of the toe was inflamed about two thirds of the time, and the upper section of the toe was only inflamed about 25% of the time. Microscopic examination only found dead (necrotic) tissue in areas that were also inflamed. There were no signs of smaller, isolated infections apart from the toe tip infections. Unlike foundered cattle, the P3 bone was not rotated in TTNS cases.

What it means

TTNS most likely moves from the outside in, not the inside out. This is significant because the prevailing belief is that the disease begins with cattle damaging the soles of the hooves. The damage to the hoof leads to thinning and weakening of the white line, and separation of the sole from the hoof wall. As previously noted, once the bacteria breach the white line, they travel into the foot to infect the P3 bone and other soft tissues. The infection doesn’t always end at the foot. Sometimes it spreads up the leg along the tendons and between the muscles, or it may enter the bloodstream and from there can spread to the lungs, liver, and kidneys.

The finding that the disease appears to move into the foot rather than starting at P3 and moving outwards may help us prevent it. Specifically, flooring should provide traction, but must not result in excessive wear. This typically occurs when animals are overcrowded and agitated in the chutes, resulting in hind feet being abraded by concrete flooring as they push against the animals ahead of them. Considering both the flooring and how the animals are being moved and handled may help prevent costly losses.

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