

**Vanselow BA** (1997) Vascular damage and heat stress related to low antioxidant levels in feedlot cattle rations. *Recent Advances in Animal Nutrition in Australia 1997* p 239

## **Vascular damage and heat stress related to low antioxidant levels in feedlot cattle rations**

**B A Vanselow**

NSW Agriculture Beef Centre, University of New England, Armidale NSW 2351

Investigations into a previously unexplained condition of leg swelling in feedlot cattle have demonstrated a relationship with low vitamin E levels and have revealed one of the predisposing causes of death from heat stress.

This study began when a summer-associated condition of swelling of the hind legs was reported in feedlot cattle (>2 months on feed) in NSW and Queensland. The condition occurred during prolonged hot weather; improvement occurred after the onset of cooler weather. It was first observed as lameness, fluid swelling of the hock joint, progressing to generalised swelling down to the hoofs and sometimes up to the groin. The skin directly above the hoof and around the pastern was reddened, swollen and tight. Microscopic examination of the lower leg revealed marked changes in small blood vessels with a resultant outpouring of fluid into surrounding tissues. The changes were similar to those described in other animal species, in particular pigs and poultry with antioxidant deficiency. Antioxidants, especially vitamin E, stabilise membranes protecting them from free radical attack. Endothelial cells of blood vessels can be a target for free radicals with resultant vessel damage, fragility and leakage.

As a consequence, antioxidant levels in feed, serum and tissue, as well as other possible contributing factors, were studied. Feedlot cattle with leg swelling or death from heat stress consistently had nil or low vitamin E supplementation and low serum/tissue levels. Secondary dietary factors which may have exacerbated the condition were high levels of fat (tallow or whole cotton seed), rancidity, high levels of carbohydrate and lactic acidosis, fungal contamination, nitrites, ionophors and an imbalance of omega 3 and omega 6 fatty acids. From our studies, feedlot cattle have very low levels of linolenic acid (an omega 3 fatty acid) compared with grazing animals (up to 100 fold difference). One of the roles of vitamin E is to control the formation of prostaglandins, although omega 3 fatty acids are significantly more effective in this role by effectively decreasing the capacity for the synthesis of leukotrienes and prostaglandins: compounds which can increase vascular permeability.

Response trials in swollen-legged cattle showed a positive response to dietary vitamin E. The disease has apparently been prevented from recurring by increasing dietary supplement with vitamin E (to 500IU vitamin E per beast per day).

During investigations into leg-swelling, 2 feedlots in northern NSW and one in southern Queensland suffered many deaths from heat stress during days of prolonged hot, humid weather. These feedlots were feeding nil or low levels of vitamin E. Leg swelling was observed over the 2-3 months prior to the deaths. At the Queensland feedlot, it was shown that animals less than 2 months on feed were significantly less at risk, and that there was no association with body weight and fat cover. It thus appeared that the deaths were associated with a physiological defect in heat dissipation. Most heat is lost through the skin either by evaporative cooling, that is, sweating or conduction. The body has an ingenious mechanism of bringing hot blood from the body core to the skin surface. Small vessels in the skin called arteriovenous anastomoses connect arteries directly to veins. When needed they open up and blood bypasses the slow capillary network, thus allowing a more rapid flow of blood to the surface, and quicker dissipation of heat from the superficial veins. This mechanism is most important in the distal limbs, hence the common observation that hot or fevered animals will stand in dams. In very hot climatic conditions it seems likely that damage to these small vessels will impair their function in heat regulation with a resultant rise in body temperature, irretrievable tissue damage and death.