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On farm factors increasing dark cutting in pasture finished beef cattle

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ABSTRACT

The on-farm factors increasing the incidence of dark cutting were studied in 3145 pasture raised cattle consigned in 66 lots. Animal, environmental and farm management factors were recorded and pasture quantity, quality and mycotoxin concentrations were measured. The relative risk of dark cutting decreased by 26% in cattle grazing pastures with magnesium concentrations exceeding 0.24%. There was a 50% increase in relative dark cutting risk of cattle drinking from dams compared to drinking from troughs. Feeding supplements (hay/silage) in the last 7 days prior to slaughter reduced the relative risk of dark cutting by 25%. A high prevalence of mycotoxins was detected in pastures across all farms. In this case pasture ergot alkaloid concentrations above 600PPB increased the relative risk of dark cutting by 45%, while the presence of FumonisinB1 increased risk by 58%. In contrast the presence of 3acetyldeoxynivalenol reduced the relative dark cutting risk by 37%. Sex also affected the incidence of dark cutting, with heifers less likely to cut dark than steers by 47%.

1. Introduction

Meat with a high ultimate pH (> 5.7 for Meat Standards Australia (MSA)) is typically dark in colour and is known as ‘dark cutting’ or ‘dark, firm and dry beef’ (DFD). Inferior meat from dark cutting carcasses graded by MSA costs the Australian beef industry up to \$55 m per annum (Jose, McGilchrist, Perovic, Gardner, & Pethick, 2015) and is estimated to cost beef producers around \$7.09 per carcass graded by MSA (McGilchrist, Alston, Gardner, Thomson, & Pethick, 2012). Of the 3.2 million Australian cattle graded in the 2014–15 financial year, the annual dark cutting rate was approximately 5.9%, however seasonal and geographical variation can push average rates up to 35% in pasture based cattle (unpublished data). Dark cutting is primarily caused by low muscle glycogen concentration at slaughter, which is a function of glycogenesis due to on-farm nutrition and glycogenolysis due to stress and exercise in the pre-slaughter period. The highest incidences of dark cutting occur at the transition phases of the pasture growing when pasture is old and poor quality or when pasture is young, short and rapidly growing (McGilchrist, Perovic, Gardner, Pethick, & Jose, 2014). However there is still considerable variability in dark cutting rates between beef producers at certain times of the year which cannot be explained by fluctuations in pasture metabolisable energy and protein alone.

Perennial ryegrass (*Lolium perenne*) is the mainstay of temperate

perennial pasture based beef systems in southern Australia, estimated to be sown over approximately 6 million hectares, predominately in regions with high winter rainfall (Foot, 1997). Perennial ryegrass can have high mycotoxin concentrations and mineral imbalances during winter when the pasture is short, lush and rapidly growing. These factors have known effects on health and productivity in livestock however the relationship with dark cutting has not been previously explored (Diaz, 2005; Fink-Gremmels, 2008; Fink-Gremmels & Diaz, 2005). Random sampling of Australian perennial ryegrass pastures identified very high infection rates with the naturally occurring fungal endophyte *Neotyphodium lolii*, with mean frequencies of 78% and up to 90% in older ryegrass cultivars (Reed et al., 2000; Reed & Moore, 2009). Concentrated in the basal leaf sheaths and flowering stems and seeds, the endophyte forms a symbiotic relationship with the ryegrass providing vigour and pest resistance via the production of mycotoxins and secondary alkaloid metabolites (DiMenna, Mortimer, Prestidge, Hawkes, & Sprosen, 1992; Keogh, Tapper, & Fletcher, 1996; Lane, 1999). *N. lolii* produces hundreds of different alkaloids, of which many are potentially toxic to grazing livestock and result in an array of adverse effects including neurological and behavioural changes, increased muscle contraction, reduced feed intake and growth rate, immunosuppression and lameness (Lean, Golder, Loudon, & McGilchrist, 2016), all of which could impact glycogen metabolism. The concentration of *N. Lolii* hyphae in the plant fluctuates seasonally, as do

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individual metabolite concentrations however all typically rise in the spring and when the plant is stressed and then drop off in late autumn (DiMenna et al., 1992). Peak pasture mycotoxin levels correspond with a time when the nutritional quality of mediterranean perennial ryegrass is lowest, thus autumn ill-thrift and poor performance is often mistakenly attributed solely to poor nutrition (Lean et al., 2016). Thus it is hypothesised that cattle grazing pastures with high mycotoxin concentrations will have an increased incidence of dark cutting.

Temperate Australian pastures during autumn and winter are typically grass dominant, young, short and rapidly growing. Pastures are typically lush with a high water content, high protein and high potassium content but low in calcium and magnesium concentrations. These parameters can result in impaired magnesium intake and absorption in grazing cattle and cause a metabolic disease known as hypomagnesaemia (HypoMg) or grass tetany (Schonewille, 2013). Magnesium is an essential dietary mineral and cofactor for numerous physiological and biochemical functions including nerve conduction, muscle contraction and adrenaline release. HypoMg can result in the animal appearing nervous or excited, with muscle twitching and in severe cases can lead to clinical tetany and death (Mayland, 1988). HypoMg reduces an animal's feed intake (Mayland, 1988) thus reduces energy intake and muscle glycogenesis. Furthermore HypoMg is likely to increase glycogenolysis due to increased neuromuscular hyperexcitability and increased adrenaline responsiveness to stress (Mayland, 1988; Schonewille, 2013). The grass tetany index or tetany ratio calculated using the equation potassium/calcium + magnesium expressed in milliequivalents (Kemp, & t Hart, 1957) is a commonly used indicator of whether cattle grazing pastures are at risk of developing HypoMg. Therefore we hypothesised that cattle grazing pastures with a higher grass tetany index will have an increased incidence of dark cutting.

2. Materials and methods

Groups (n = 66) of *Bos Taurus* pasture raised cattle (n = 3145) were sourced from commercial beef properties on King Island, Tasmania. The cattle were predominately British breeds, (Angus, Hereford & Murray Grey), typically < 24 months, of varying sex, and were hormone and anti-biotic free. Cattle were transported via ship to mainland Tasmania for slaughter at the same processing plant between March and June 2015. The 66 groups were slaughtered on 13 kill days with between 3 and 6 groups harvested per kill day. Stock were loaded into stock trailers at the property of origin on a Sunday and remained in these trailers until unloading at the processing plant on Monday. Trucking distance on King Island from farm gate to port ranged from 15 to 90 km. Shipping-time between King Island and mainland Tasmania was approximately 8 to 10 h, and trucking distance on mainland Tasmania was approximately 100 km. The cattle did remain in the stock trailers on the ship between arriving in Tasmania and the Devonport port opening at 6 am. Fitting in with MSA requirements, all stock were slaughtered within 36 h of leaving the farm gate.

All cattle transported on the same day were also slaughtered on the same day in successive lots. Carcasses were dressed to AUS-MEAT carcass standards and hot standard carcass weight (HSCW) measured in kilograms at the end of the processing chain. Carcasses were chilled overnight and were graded by qualified Meat Standards Australia graders 18 to 24 h post-mortem. Carcass traits assessed by graders included;

- gender (steer/heifer)
- eye muscle area (EMA) of the *longissimus thoracic et lumborum* at the carcass quartering site in square centimeters, measured using a standardised AUS-MEAT grid (AUS-MEAT, 2005)
- ultimate pH (pHu) of the *longissimus thoracic et lumborum* (AUS-MEAT, 2005)
- rib fat depth measures millimeters of subcutaneous fat of the

longissimus thoracic et lumborum at the quartering site

- ossification score is the amount of spinous process calcification of the thoracic, lumbar and sacral vertebrae. It is measured on a scale between 100 and 590 (AUS-MEAT, 2005, Romans et al., 1994)
- MSA marbling score is the amount of fat between muscle fibres in the *longissimus thoracic et lumborum* muscle at the quartering site (AUS-MEAT, 2005)
- MSA index is calculated using a predictive model of eating quality (Polkinghorne, et al, 2008)

At grading, the *longissimus thoracis* must be < 12 °C, have a pH ≤ 5.7 and/or meat colour ≤ 3 to be eligible for MSA grading. Group 33 (n = 25head cattle) was not MSA graded after slaughter so was eliminated from the data set as no carcass characteristics including pH and colour were recorded thus incidence of DFD could not be determined. Percentage of DFD was calculated for each group.

%DFD = number of DFD per group/total number in group

Animal and management factors for each group of cattle were recorded including water source (dam or trough), grazing type (rotational or set stocked), pasture type (rye grass dominant, clover or alfalfa), supplementary feed offered (yes or no), lifetime yardings, lifetime truckings, average age, weaning age, weaning method (yard or paddock), days since last yarding, trace element supplementation in the last 6wks (yes or no), hours of curfew off feed prior to shipping and trucking distance from farm gate to King Island port. Environmental factors for each shipment were recorded including weather forecast, maximum and minimum temperature, wind speed and direction, sea state, swell height, direction and period.

Pasture availability in kilograms of dry matter per hectare was calculated using the average of fifteen 0.1 m² quadrants cut to grazing height (3 cm above ground level) and oven dried. A 500 g sample of pasture sward and supplementary feed for each group was collected for pasture quality, mineral and mycotoxin analysis. Pasture was plucked to grazing height to represent what the animal was eating from 20 random locations across the paddock, avoiding high traffic areas, fence lines and watering areas. Supplementary feed (silage and hay) samples were taken at 12 random sites from fresh unloaded material. Samples were mixed thoroughly in a clean plastic bucket then 500 g selected for analysis, vacuum packed and frozen to prevent respiration and fermentation changes. Samples were then freeze-dried and cut into 5–8 cm pieces before laboratory analysis. Pasture quality and minerals were analysed using near infra-red and wet chemistry (Dairy One, Ithaca, New York, USA). Forage quality included metabolisable energy, crude protein, dry matter, acid detergent fibre, effective neutral detergent fibre, in vitro true digestibility, trace element and mineral concentrations (magnesium, potassium, calcium, Sodium, Chloride (Cl), Copper, Molybdenum) using Near Infra-Red and Wet Chemistry (Dairy One, Ithaca, New York, USA). The grass tetany index was calculated using the following equation which converts % diet dry matter to milliequivalents (MEq) (Kemp, & t Hart, 1957). Indices > 2.2 suggest an increased risk of HypoMg (Schonewille, 2013).

Grass tetany index = (% potassium × 256)/[(% calcium × 499) + (% magnesium × 823)].

Mycotoxin analysis was conducted by Biomin at Romer Labs, Singapore. The method was a high-performance liquid chromatography-electrospray ionization-mass spectrometry (HPLC-ESI-MS/MS) using an Eksigent ultraLC100-XL HPCL coupled to an Applied Biosystems 5500 Qtrap mass spectrometer (Waltham, MA, USA). The results were interpreted by BIOMIN Holding GmbH (Getzersdorf, Austria). The major mycotoxins and their families tested were Ochratoxin-A, Zearalenone, Fumonisin, Aflatoxins, Ergot Alkaloids, β-trichothecenes and α-trichothecenes. The individual mycotoxins analysed included; 15AcetylDeoxynivalenol, 3AcetylDeoxynivalenol, AflatoxinB1, AflatoxinB2, AflatoxinG1, AflatoxinG2, Deoxynivalenol, Diacetoxyscirpenol, FumonisinB1, FumonisinB2, FumonisinB3,

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