Effects of dietary beta-agonist treatment, Vitamin D₃ supplementation and electrical stimulation of carcasses on meat quality of feedlot steers
Introduction

• The current classification system is utilized for:
  - The description of specific requirements when purchasing carcasses
  - The utilisation of variety in the market with a view to optimum consumer satisfaction
  - utilisation of price differences and to determine selling prices.

• The current system classifies meat by:
  - Age of the animal
  - Fatness
  - Conformation
A-age animals are considered to be the most tender and meat from A-age animals is sold for more.

In reality this is not always the case as many other factors other than age can affect tenderness.

These factors include both pre- and post-slaughter manipulation:
- use of beta-agonists
- Supplementation
- controlled electrical stimulation of carcasses
Most South African feedlots supplement with a beta adrenergic-agonist (hereafter referred to as beta-agonists).

The beta-agonist zilpaterol is one of the most commonly utilised beta-agonists in commercial beef production.

Beta-agonists improve rate of gain, feed efficiency and increase carcass meat yield efficiency.

Both the producer and consumer could benefit as meat becomes less expensive to produce.
Beta agonists are known to affect the aging potential of beef muscle negatively by increasing the activity of the enzyme calpastatin.

This results in the production of tougher meat.

Both controlled electrical stimulation of carcasses as well as the supplementation of ultra-high levels of vitamin D₃, for short periods before slaughter, could enhance the aging potential of beef and alleviate this problem.
Materials and Methods

• One hundred and twenty Bonsmara steers.

• Six treatment groups:
  - Control group which received the feedlot diet only.
  - Five remaining groups were all supplemented with zilpaterol hydrochloride (0.15mg/kg live weight for 30 days and withdrawn 4 days prior to slaughter).
  - One of the five groups only received zilpaterol.
  - The remaining four groups received zilpaterol and vitamin D₃ at the following levels and durations prior to slaughter:
    • $7 \times 10^6$ IU/animal/day for 3 days (3D7M)
    • $7 \times 10^6$ IU/animal/day for 6 days (6D7M)
    • $7 \times 10^6$ IU/animal/day for 6 days followed by 7 days of no supplementation (6D7M7N)
    • $1 \times 10^6$ IU/animal/day for 9 days prior to slaughter (9D1M)
Carcasses were split and the left sides electrically stimulated (ES) for 30s within 30 min of killing and the right sides were not electrically stimulated (NES).

All samples were collected from the *M. Longissimus lumborum*.

pH and temperature measurements were taken every hour for 4 hours and a final measurement was taken at 18 hours *post mortem*. 
The following tests were conducted:

- Warner Bratzler shear force (WBSF) measurements at 3 and 14 days post mortem.

- Myofibril fragment length (MFL) at 3 and 14 days post mortem.

- $\mu$-calpain, m-calpain and calpastatin activity at 1 hour and 24 hours post mortem.

- Sarcomere lengths at 24 hours post mortem.
Fig. 1. Interaction between treatment and ES in relation to WBSF ($P = 0.003$). (Bars with different superscripts differ significantly).
• **Fig. 2.** Interaction between treatment and ES in relation to calpastatin activity \((P = 0.015)\). (Bars with different superscripts differ significantly).
• **Fig. 3.** Interaction between treatment and *post mortem* aging in relation to WBSF ($P < 0.001$). (Bars with different superscripts differ significantly).
• **Fig. 4.** Interaction between treatment and time of measurement in relation to μ-calpain activity ($P < 0.002$). (Bars with different superscripts differ significantly).
• **Fig. 6.** Interaction between stimulation and time of measurement in relation to μ-calpain activity ($P < 0.001$). (Bars with different superscripts differ significantly).
Fig. 1a. Interaction between treatment (Control, 3D7M, 9D1M, 6D7M7N, 6D7M, Zilpaterol), electrical stimulation (NES and ES) and post mortem aging (3 and 14 days) for Warner Bratzler shear force.
Fig. 1c. Interaction between treatment (Control, 3D7M, 9D1M, 6D7M7N, 6D7M, Zilpaterol), electrical stimulation (NES and ES) and time of measurement post mortem (1 h and 24 h) for Calpastatin activity.
Fig. 1d. Interaction between treatment (Control, 3D7M, 9D1M, 6D7M7N, 6D7M, Zilpaterol), electrical stimulation (NES and ES) and time of measurement *post mortem* (1 h and 24 h) for μ-calpain activity.
Conclusion

- High levels of vitamin D$_3$ supplementation does not seem to be a viable option for improving meat tenderness in beta-agonist treated beef.

- Only a shorter but higher dose (3D7M) and a longer but lower dose (9D1M) of vitamin D$_3$ showed small but significant improvements in tenderness, under conditions of no electrical stimulation.
• The benefit of using electrical stimulation on its own should be less costly and show better results on improving beta-agonist treated beef compared to any vitamin D$_3$ treatment with no stimulation.

• Furthermore, with electrical stimulation, no added advantage of feeding vitamin D$_3$ is achieved.
While age of the animal is still an important contributing factor to tenderness and meat quality, alone it is not an accurate predictor of meat quality and other pre- and post harvest factors also need to be taken into account.
Thank You