

## CHAPTER 7: CONCLUSION

Vitamin D<sub>3</sub> has the potential to increase plasma calcium levels and therefore increase levels of calcium in the muscles, resulting in more calcium being available to activate the calcium dependant proteinases. Results regarding this effect of vitamin D<sub>3</sub> are however varied. One reason for this is the homeostasis mechanism in place to regulate plasma calcium levels which would have to be overcome by the correct dose and duration of vitamin D<sub>3</sub> supplementation. Even so, previous studies have shown that increased plasma calcium does not always lead to an increase in muscle calcium or any effect on the calcium dependent proteinase system. In addition, studies on vitamin D's ability to counteract the negative effects of beta-agonist supplementation are sparse.

In general electrical stimulation has its advantages in that it can counteract cold shortening where carcasses are chilled quickly. It can also result in more tender meat at an early stage without the prolonged aging. In this study we looked at the effect that both these methods had on the tenderness of meat from beta-agonist treated steers which would be tougher (when compared to control animals) due to an increased level of calpastatin activity, inhibiting the calcium dependent proteinase system. Another aim was to study the combined effects of these two treatments together (e.g. vitamin D supplementation in combination with electrical stimulation of carcasses) on meat tenderness.

In this study we have shown that the main reason for the decrease in tenderness of meat from beta-agonist supplemented animals is due to an increase in the levels of calpastatin, which is an inhibitor of the calpains. Vitamin D<sub>3</sub> proved not to be the most effective method of counteracting this negative effect on tenderness. Only the short, high dose (3D7M) and the low dose over a long period of time (9D1M) showed significant but small improvements in tenderness, but only under conditions where no electrical stimulation was applied. The best positive effect on calpastatin was recorded for 6D7M7N at 1h *post*

*mortem*, yet in all the scenarios the calpastatin activities for vitamin D<sub>3</sub> treatments were still significantly higher than for the control group. Despite elevated levels in serum calcium for 3D7M, 6D7M7N and 9D1M, it probably had little effect on the free cytosolic calcium to activate sufficient  $\mu$ -calpain to neutralize the effect of raised calpastatin activities caused by the beta-agonists. Vitamin D<sub>3</sub> also had only a small effect on the other quality traits (drip loss and colour) which were negatively affected by beta-agonist supplementation.

Electrical stimulation improved loin tenderness of both beta-agonist supplemented and non-supplemented animals and improved loin tenderness in general by the early onset of rigor by triggering the activity of the calpains. In addition electrical stimulation advanced the tenderisation process of steaks from the zilpaterol supplemented group of steers by reducing the activity of calpastatin and can therefore be implemented to improve meat tenderness of meat from zilpaterol supplemented steers. Electrical stimulation did however increase drip loss which also resulted in paler meat.

A significant interaction occurred between treatment and electrical stimulation. Electrical stimulation had very little effect on the tenderness of the control group, but had a significant effect on all the other treatment groups in particular on Z, 6D7M7N and 6D7M. Electrical stimulation reduced the variation between mean values for tenderness of all groups supplemented with zilpaterol. Furthermore, with electrical stimulation, no added advantage of feeding vitamin D<sub>3</sub> was achieved.

Electrical stimulation proved to be far more effective in improving meat tenderness compared to vitamin D<sub>3</sub> supplementation. The benefit of using electrical stimulation on its own is that it is less costly and easier to implement than vitamin D<sub>3</sub> supplementation. It also avoids the possible negative effects that vitamin D<sub>3</sub> can have on production due to a decrease in feed intake. However neither vitamin D<sub>3</sub> supplementation nor electrical

stimulation could completely counteract the effects of the beta-agonists and it is therefore advisable not to use zilpaterol if tenderness is the main focus of production.

## CHAPTER 8: CRITICAL EVALUATION

The aim of this research project was to study the effects of dietary beta-agonist treatment of feedlot cattle in combination with vitamin D<sub>3</sub> supplementation and electrical stimulation of the carcasses on meat quality parameters. Although these effects have all been studied separately before, there are no studies on the combined effects of these treatment combinations on meat quality of South African feedlot cattle. This experiment also explored various doses and durations of vitamin D<sub>3</sub> supplementation and in addition was a large trial allowing us to properly study the interactions between treatments, therefore allowing us to conclude which treatments or combination of treatments could be best utilized to improve meat quality. The experiment also focussed on the calcium dependant proteinase system giving a better picture of the effect of each treatment (and their combinations) on meat tenderness.

Regarding cattle breed type, the Bonsmara breed was chosen as a model because it is a typical medium frame animal and the most common breed found in feedlots throughout the country. This breed is also representative of a large portion of South Africa's feedlot stock. There would however be some breed differences regarding zilpaterol supplementation in that its use would not be recommended for later maturing breeds as their carcasses would become too heavy before fattening occurs. Beta-agonist treatment is also not recommended in breeds such as the Brahman, which already produce tougher meat due to inherently higher levels of calpastatin activity. The results of the present study with Bonsmara type feedlot cattle, provides a good indication of what can be expected in the South African beef industry.

As far as the diet fed is concerned, the cattle in our trial were fed a commercial feedlot diet typical of the diet fed in many feedlots. This was to try and eliminate diet as a variable as much as possible. There were however differences between the site of the

experiment when compared with conditions at a commercial feedlot in that a commercial feedlot would have less space available per animal, less bunk space for feeding, and therefore there is a chance that shy feeders would have less intake of a particular supplement (in this case vitamin D<sub>3</sub>). This is however an indication that the lack of response to vitamin D<sub>3</sub> supplementation in our trial, under controlled conditions, provides even stronger evidence that the current use of vitamin D<sub>3</sub> in commercial feedlots is a huge expense failure.

There are some changes or improvements that could have been made to the project. Firstly the trial could have included another treatment group of vitamin D<sub>3</sub> supplementation only. This could have shown if vitamin D<sub>3</sub> had any effect on improving meat tenderness when not under the challenge of zilpaterol supplementation. Muscle calcium could also have been measured in addition to the plasma calcium levels that were measured. This would possibly have given a better indication of whether increased plasma calcium levels led to an increase in calcium in the muscle and therefore to more calcium being available for the calcium dependent proteinase system.

It can once again be mentioned that so far no feed additive or pre- or post-slaughter practice has been able to improve tenderness of meat from steers supplemented with zilpaterol hydrochloride to the level of that of meat from steers not treated with the beta-agonist. A number of producers have already stopped the practice of supplementing with a beta-agonist which has resulted in the production of more tender meat being sold at a premium price. In South Africa however, the bulk of producers will continue to supplement with a beta-agonist to produce more meat per animal and therefore sell meat at a cheaper price to a population where red meat is often a luxury. It would therefore be beneficial to not only look at other methods of improving tenderness of meat from beta-agonist supplemented animals, but also to study the actual molecule more closely and gain a better knowledge of its mechanisms of action, thereby potentially being able to modify the molecule so that it

achieves the same results as a repartitioning agent but without the negative effects on meat tenderness.

This study ultimately showed that supplementation of vitamin D<sub>3</sub> had very little or no effect on meat quality. Consumers have shown that consistency in the quality of a product is important and with many feedlots across South Africa already supplementing vitamin D<sub>3</sub>, in an attempt to improve meat tenderness, the results from this study are beneficial in proving that this expensive practice is doing little to improve meat quality and more importantly has shown that supplementation with vitamin D<sub>3</sub> leads to inconsistencies in meat quality. We have also shown that further research needs to be conducted to find other methods (other than or which could be additive to electrical stimulation) to improve tenderness of meat from zilpaterol supplemented steers in our country.