# EFFECTS OF VITAMIN D<sub>3</sub> ON LIVE ANIMAL PERFORMANCE AND CARCASS TRAITS OF FEEDLOT CATTLE

By

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# NOMENCLATURE

1α-OH-D <sub>3</sub>	$1\alpha$ -hydroxyvitamin D <sub>3</sub>
1,24,25-(OH) <sub>3</sub> D <sub>3</sub>	1,24,25-trihydroxyvitamin $D_3$
1,25-(OH) <sub>2</sub> D <sub>3</sub>	1,24-dihydroxyvitamin D <sub>3</sub>
1,25,26-(OH) <sub>3</sub> D <sub>3</sub>	1,25,26-trihydroxyvitamin $D_3$
25-OH-D <sub>3</sub>	25-hydroxyvitamin D <sub>3</sub>
°C	degrees Celsius
Ca <sup>++</sup>	Ionized Calcium
CaBP	Calcium Binding Protein
CON	control
d	days
EDTA	Ethylenediamine tetracetic Acid
gm/ml	gram per milliliter
U	international units
IU/kg	international unit per kilogram
kcal	kilocalorie
Kg	kilogram
Mg	milligram
mg/dl	milligrams per deciliter
mM	millimole

MW	Molecular weight
Na	Sodium
ng/ml	nanograms per milliliter
PTH	Parathyroid Hormone
μg	microgram
μΜ	micromole
VDBP	Vitamin D binding protein
VITD	vitamin D <sub>3</sub>
Vitamin D <sub>2</sub>	Ergocalciferol
Vitamin D <sub>3</sub>	Cholecalciferol
WBS	Warner-Bratzler shear force

#### CHAPTER I

#### INTRODUCTION

The United States beef industry has continually lost its market share to poultry and pork over the last twenty years. According to the 1995 National Beef Quality Audit (Smith et al., 1995) low overall uniformity, inadequate tenderness and excessive fatness were ranked in the top ten quality concerns the cattle industry should be addressing in order to prevent further market share loss to other food protein sources.

Boleman et al. (1997) demonstrated consumers are willing to pay more for beef if they perceive it to have improved tenderness. This demonstrates an economic incentive for the cattle industry to produce a higher quality, more palatable product. One means of improving beef tenderness is supplementing cattle with vitamin D<sub>3</sub>. Swanek et al. (1999) demonstrated that feeding supplemental vitamin D<sub>3</sub> in finishing rations of feedlot steers for the final 5 to 10 days immediately prior to harvest aids in improving the tenderness and consistency of beef.

Although vitamin  $D_3$  may increase beef tenderness, there is a concern that dietary supplementation of vitamin  $D_3$  may have a detrimental effect on live animal and carcass performance of feedlot cattle. Enright et al. (1998) reported that feeding pigs moderate (55,031 IU/kg) and high (176,000 IU/kg) levels of vitamin  $D_3$  10 d prior to harvest decreased daily feed intake from 3.82 to 3.63 and 2.90 kg, respectively. Swanek et al. (1999) reported that cattle supplemented with 5 million IU of vitamin  $D_3$  for 5 d immediately prior to harvest

exhibited lighter (336.2 kg vs 326.4kg) carcass weights than non-supplemented cattle. The objective of this study was t measure the effects of dietary supplementation of vitamin D3 on feed consumption and carcass characteristics of feedlot cattle.

# CHAPTER II REVIEW OF LITERATURE

# Introduction

There are two major sources of dietary vitamin D: ergocalciferol (vitamin D<sub>2</sub>) is derived from the plant steroid ergosterol and is the usual form employed for vitamin D fortification of foods; and Cholecaliferol (vitamin D<sub>3</sub>), the primary source of supplemental vitamin D for domestic animals. Vitamin D<sub>3</sub> is commonly referred to as the "sunshine vitamin" because the skin converts 7dehydrocholesterol into vitamin D<sub>3</sub> when exposed to ultra violet light (DeLuca, 1979). Since the body is capable of producing choecaliferol, vitamin D does not meet the classical definition of a vitamin. Vitamin D may be more accurately described as a prohormone; that is, vitamin D is metabolized to a biologically active form that functions as a steroid hormone. However, since vitamin D was first recognized as an essential nutrient, it has historically been classified among the lipid soluble vitamins. Vitamin D's primary functions are to maintain calcium and phosphorus homeostasis, promote intestinal absorption of calcium, and bone deposition and mobilization of calcium, and (DeLuca, 1967). Failure of bone mineralization results in rickets; vitamin D is recognized as a treatment for this disease.

#### Vitamin D Absorption

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Vitamin D is absorbed from the small intestine by passive diffusion that depends on micellar mobilization, and the presence of bile salt for absorption into the lymphatic system (Norman, 1980). Absorption is most active in the duodenum (Schachter et al., 1964). However, the greatest amount is absorbed in the ileum due to the longer transit time of the food in this region of the intestine (Norman and DeLuca, 1963). Norman and DeLuca (1936) demonstrated that up to 80% of orally administered radioactive vitamin D in oil is absorbed. Norman (1980) found 50% of orally administered vitamin D is absorbed and transported via the lymph duct to the blood stream and then to the liver. Neville and DeLuca (1966) demonstrated that vitamin D<sub>3</sub> in an alcoholic solution is rapidly taken up by the liver. Within 60 minutes, plasma vitamin D is removed from the blood into the liver (Ponchon and DeLuca, 1969). Vitamin D<sub>3</sub> is transported in the blood stream bound the vitamin D-binding protein (VDBP) (Haddad and Walgate, 1976). The VDBP circulates in the plasma at about 1-2 ng/ml. The protein has a binding capacity in excess of the amount of vitamin D and its metabolites in the plasma (DeLuca, 1979).

#### Vitamin D Storage

In the rat, stored vitamin D is primarily found in fat deposits. However, rat tissue can not store vitamin D or its metabolites against a concentration gradient (Rosenstreich et al., 1971). Blood has the highest concentration of vitamin D when compared to other tissues (Quaterman, 1964). Mawer et al. (1972) indicated that human adipose tissue is a predominant storage site for vitamin D<sub>3</sub>

and muscle serves as a storage site for 1, 25 dihydroxyvitamin D<sub>3</sub>. Depletion of vitamin D is likely to take years, mainly because it depends on turnover of lipid deposits (DeLuca, 1979). However, the half-life of Vitamin D in plasma is estimated at 22 hours (Avioli et al., 1976). In humans, the half-life of 1,25-dihydroxyvitamin D<sub>3</sub> (1,25-(OH)<sub>2</sub> D<sub>3</sub>) in the plasma is 2 to 4 h (Gaynor et al., 1989).

#### Vitamin D Activation

Before vitamin D can exhibit any biological activity, it must first be metabolized by the body to its active forms. The hepatic uptake of vitamin D is the first step toward its activation and subsequent C-25 hydroxylation by the liver (Gascon-Barre' et al., 1982). 25-hydroxvitamin D<sub>3</sub> does not accumulate in the liver (DeLuca, 1979); it is transported to the kidney on a Vitamin D transport protein (MW 52,000) where it undergoes 1,25-hydroxylation to form 1,25dihydroxyvitamin D<sub>3</sub> (1,25-(OH)<sub>2</sub> D<sub>3</sub>), a metabolically active form of vitamin D.

#### 25-hydroxylation of Vitamin D

Vitamin D<sub>3</sub> is hydroxylated primarily in the liver on the 25 position of the carbon side chain to yield 25-(OH) D<sub>3</sub> (Ponchon and DeLuca, 1969). Tucker et al. (1973) reported that the chick kidney and intestine also possess 25hyroxylating ability, although, no detection of 25-hydroxylation in the kidney was observed by DeLuca (1974). Bhattacharyya and DeLuca (1974) reported 25hydroxylation in the intestine of the chick. However, 25-hydroxylation could not

be found in the rat intestine (Holick et al., 1976). Olson et al (1976) concluded that the major site of 25-hydroxylation is the liver.

The production of 25-(OH)  $D_3$  is catalyzed by the vitamin D 25-hydroxylase enzyme that hydroxylates carbon 25 of the side chain. 25-Hydroxylase is found in the microsomal fraction of the liver (DeLuca, 1974). 25-hydroxylation requires NADPh, molecular oxygen, and magnesium ions (DeLuca, 1979).

#### 1,25-hydroxylation of Vitamin D

From the liver, 25-(OH)  $D_3$  is transported to the kidney via the vitamin D transport binding protein transcalciferin (MW 52,000) (DeLuca, 1979). Hydroxylation of 25-(OH)  $D_3$  is catalyzed by vitamin  $D_3$  1-hydroxylase in the mitochondria to yield 1,25-(OH)<sub>2</sub>  $D_3$  (Figure 1). 1,25-hydroxylation is controlled directly by the parathyroid hormone (PTH). The PTH action is mediated by cyclic AMP, plasma calcium levels and phosphorous concentrations (Suda et al., 1977).

The 1,25-hydroxylation in intact mitochondria is supported by succinate, malate, or other Krebs cycle substrates and requires the electron transport chain as well as oxidative phosphorylation. When the mitochondria are swollen with calcium ions, NADPh is the electron donor (DeLuca And Schnoes, 1976). Thus the hydroxylation is independent of oxidative phosphorylation.

In non-pregnant mammals, the kidney is the site of  $1,25-(OH)_2D_3$  production. However, pregnant mammals have the ability to synthesize  $1,25-(OH)_2D_3$  in the placenta (DeLuca, 1988).



Figure 1. Activation of Vitamin D. (Source: Vitamin D, 1997)

## 1,25-dihydroxyvitamin D<sub>3</sub>

Tanaka and DeLuca (1973) demonstrated that 1,25-dihydroxyvitamin D<sub>3</sub> is the physiologically active form of Vitamin D<sub>3</sub> responsible for intestinal calcium transport, bone calcium mobilization, and is able to increase serum phosphorous concentration. Large doses of 25-(OH) D<sub>3</sub> increases calcium transported across the intestinal membrane and causes mobilization of bone calcium (Reynolds et al., 1973; Olson and DeLuca, 1969). However, 25-dihydroxyvitamin D<sub>3</sub> does not bring about these responses at physiological levels.

# Other Metabolites of Vitamin D<sub>3</sub>

In addition to the production of 1,25-(OH)<sub>2</sub> D<sub>3</sub>, the kidney converts 25-(OH) D<sub>3</sub> hydroxyvitamin D<sub>3</sub> to 23,25-dihydroxyvitamin D<sub>3</sub> (23,25-(OH)<sub>2</sub> D<sub>3</sub>) (Figure 2); 24,26-dihydroxy Vitamin D<sub>3</sub> (24, 25-(OH)<sub>2</sub> D<sub>3</sub>); 25,26-Dihydroxy Vitamin D<sub>3</sub> (25,26-(OH)<sub>2</sub> D<sub>3</sub>); 1,24,25-trihydroxyvitamin D<sub>3</sub> (1,24,25-(OH)<sub>3</sub> D<sub>3</sub>), and 1,25,36-trihydroxyvitamin D<sub>3</sub> (1,25,26-(OH)<sub>3</sub> D<sub>3</sub>). The role of these compounds in the function of vitamin D has not been fully evaluated, and significant physiological

roles are yet to be discovered (McDowell, 1989). Henry and Norman (1978) suggested that 24,25-(OH)<sub>2</sub> D<sub>3</sub> is responsible for the production of cartilaginous compounds and for the development of chick embryos. The hatchability of eggs is severely depressed in vitamin D deficient hens, even though they are fed 1,25 (OH)<sub>2</sub> D<sub>3</sub>. Without vitamin D, the upper mandible of chicks fails to develop. Consequently the chick cannot crack the shell, resulting in mortality. Therefore, McDowell (1989) concluded that 24,25-(OH)<sub>2</sub> D<sub>3</sub> is required. However, Hart et al. (1984) were unable to discover a role for 24,25-(OH)<sub>2</sub> D<sub>3</sub> in hatchability. Bordier et al. (1978) found that in humans 24,25-(OH)<sub>2</sub> D<sub>3</sub> is required with 1,25-(OH)<sub>2</sub> D<sub>3</sub> for healing of osteomalacia.

The 24-hydroxylation of 25-(OH)  $D_3$  and 1,25-(OH)<sub>2</sub>  $D_3$  is the primary mechanism and the first step in a metabolic pathway to inactivate and degrade these vitamin D metabolites (Suda et al., 1977). The primary regulators of renal 24-hydroxylase activity are PTH and 1,25-(OH)<sub>2</sub>  $D_3$  (Horst et al., 1981).

The major site of 24-hydroxylation is the kidney, although, the existence of 24-hydroxylase has been observed in intestinal tissue (Kumar et al., 1978) and in cartilage tissue (Garabedian et al., 1977). The role of 24-hydroxylase as an enzyme responsible for the production for the biologically active compound  $24,25-(OH)_2 D_3$  remains controversial.



Figure 2. Vitamin D metabolites (Source: Vitamin D, 1997)

#### Vitamin D<sub>3</sub> Regulation of Calcium and Phosphorus Homeostasis

The vitamin D derivative 1,25-(OH)<sub>2</sub> D<sub>3</sub> can regulate both serum calcium and phosphorus by affecting the kidney and the PTH (Ribovich and DeLuca, 1971). In the rat, low calcium diets stimulated and high calcium diets suppressed, the production and appearance of vitamin D (Boyle et al., 1971). Low blood calcium stimulates secretion of PTH which in turn increases the production of 1,25-(OH)2 D<sub>3</sub> in the proximal convoluted tubule of the kidney. Parathyroid hormone and 1,25 dihydroxyvitamin D<sub>3</sub> mobilize bone calcium and increase renal reabsorption. Low blood phosphorus stimulates the secretion of 1,25-(OH)<sub>2</sub> D<sub>3</sub> which increases the level of ionized calcium and in turn suppresses PTH. Lack of PTH results in increased phosphate retention by the kidney. 1,25dihydroxyvitamin D<sub>3</sub> stimulates mobilization of phosphate from bone and phosphate absorption in the small intestine (DeLuca, 1979). Friedlander et al., (1977) demonstrated that a low phosphate diet fed to chicks increases the circulating concentration of 1,25-(OH)<sub>2</sub> D<sub>3</sub> and the uptake of 1,25-(OH)<sub>2</sub> D<sub>3</sub> by the mucosal cells of the small intestine. Mayer et al. (1966) reported that PTH decreases the daily excretion of fecal phosphorus by cows.

#### Vitamin D<sub>3</sub> Effects on Plasma Calcium Concentrations

Calcium concentrations in blood plasma are maintained with percision. Normal serum calcium for cattle remains between 8 and 12 mg/dl (McDowell, 1989). However, Swanek et al. (1999) reported that steers supplemented with 5 million IU of vitamin D<sub>3</sub> for 5 d demonstrated a significant increase in plasma calcium concentrations as compared to non-supplemented steers. Likewise,

Montgomery et al. (1998) reported that steers supplemented with 5 or 7.5 million IU of vitamin  $D_3$  for 10 d resulted in an increase of plasma calcium at d 5 through d 10. Enright et al. (1998) observed that pigs supplemented with 331, 55,031 and 176,000 IU of vitamin  $D_3$  per kilogram of feed for 10 d demonstrated significantly higher serum calcium levels than non-supplemented pigs.

Vitamin D<sub>3</sub> injected intramuscularly may have effects similar to oral administration. Horst and Littledike (1979) observed that four weekly injections of 15 million IU of vitamin D<sub>3</sub> began to elevate blood calcium levels at d 8 through d 10 following the first injection of vitamin D<sub>3</sub>. Blood calcium levels continued to increase after each injection as did 1,25-(OH)<sub>2</sub> D<sub>3</sub>. Blood calcium levels peaked after d 3 of the fourth injection.

Boling and Evans (1979) reported that intravenous administration of 2 million IU of vitamin  $D_3$  increased serum calcium 0.9 mg/dl on d 2 and 3. However, the same dose of vitamin  $D_3$  injected intramuscularly resulted in a smaller, more prolonged increase of serum calcium from d 2 through 25.

For the prevention of parturient paresis in lactating dairy cows, intramuscular injections of 700 µg of 1 $\alpha$ -hydroxyvitamin D<sub>3</sub> in propylene glycol have been used; this increased plasma calcium within 24 h by 2.4 mg/dl. Three to 5 d later a plateau was reached, and then amounts slowly declined until d 15 after the injection (Bar et al., 1988). Plasma concentration of 1 $\alpha$ -hydroxyvitamin D<sub>3</sub> peaked 48 h following the injection and then declined, reaching the initial concentration 6 d later. The biological half life of 1  $\alpha$ -dihydroxyvitamin D<sub>3</sub> was estimated at 2.1 d (Bar et al., 1988).

A combination of 0.5mg  $1\alpha$ -hydroxyvitamin D<sub>3</sub> plus 4 mg of 25-hydroxyvitamin D<sub>3</sub> injected intramuscularly in 5 d intervals prepartum maintained an increase in serum calcium levels through parturition at d 15 following the first injection (Hodnett et al., 1992). Serum calcium levels were significantly higher at d 2 postinjection by 0.6 mg/dl; by d 5 up to 7, serum calcium increased to 1.8 mg/dl higher than preinjection levels.

Srivastav et al. (1995) reported that Intraperitoneal injections of  $25-(OH)_2 D_3$ and 1,25-(OH)\_2 D\_3 at 65 or 650 pmol for 15 d increased serum calcium levels. At d 1, those animals treated with 65 pmol of 1,25-(OH)\_2 D\_3 were unaffected; however, after d 3 all treatments experienced an increase in serum calcium. At d 15, serum calcium levels increased further in animals treated with 1,25-(OH)\_2 D\_3, unlike those treated with 25-(OH)\_2 D\_3 which experienced a decline in serum calcium. This indicates that the elevation of serum calcium is dose dependent for both 25-(OH)\_2 D\_3 and 1,25-(OH)\_2 D\_3.

#### Calcium Concentration Mechanisms

Vitamin D increases serum calcium concentrations by stimulating intestinal calcium absorption (Nicolaysen, 1937), mobilizing calcium from bone (Bauer et al., 1955) and increasing renal reabsorption of calcium (Figure 3) (Yamamoto et al., 1984). Mobilization of calcium from bone and renal absorption of calcium require both PTH and 1,25-(OH)<sub>2</sub> D<sub>3</sub> (Yamamoto et al., 1984). Intestinal calcium absorption is increased indirectly by PTH, which in turn increases the production of 1,25-(OH)<sub>2</sub> D<sub>3</sub>. 1,25-dihydroxyvitamin D<sub>3</sub> with PTH and calcitonin constitute

the claciotropic hormones that maintain serum calcium either through direct or indirect effects on the intestine, kidney, and skeleton (Walters, 1992).



Figure 3. Vitamin D metabolites and mode of action. (Source: Vitamin D, 1997)

## Intestinal Calcium Absorption

Calcium absorption from the intestine involves two independent processes. One is a saturable transcellular process that is regulated by vitamin D, via the calcium-binding protein (CaBP, MW=8800). The transcellular movement is largely confined to the duodenum and upper jejunum portion of the intestine (Behar and Kerstein 1976). When calcium is in demand, most intestinal calcium absorption occurs via this process. The second process is a nonsaturable paracellular process that occurs throughout the length of the intestine (Nemere and Norman, 1991). This process becomes the prominent form of intestinal calcium absorption when intralumenal calcium concentrations are relatively high (Marcus and Lengemann, 1962).

Dietary calcium is absorbed according to requirements; on a low calcium diet the efficiency of calcium absorption is increased (Braithwaite, 1975). Klooster (1976) demonstrated that the percentage of calcium absorbed from the small intestine of dairy cows increased in response to a reduced dietary calcium intake.

## Transcellular Intestinal Calcium Transport

The three steps in the transcellular calcium transport path include the transfer of calcium across the brush border membrane into the cell interior, movement through the cellular compartment, and finally its extrusion across the basolateral membrane.

## A. Calcium entry

The entry of calcium from the intestinal lumen across the brush border membrane into the cell is a downward electrochemical gradient. According to Rasmussen et al. (1982), the rapid effect of 1,25-dihydroxyvitamin D<sub>3</sub> on increasing calcium uptake is attributed to alterations in the membrane phospholipid content and membrane fluidity. However, a relationship between vitamin D, membrane fluidity, and calcium transport has not been reported by others (Bikel et al., 1984; Schedl et al., 1994). Other factors have been implicated in calcium entry, including calbindin D<sub>28k</sub>, a calcium binding protein associated with the brush border membrane and alkaline phosphatase (Fullmer, 1992). Because of the impermeability of lipid membranes to calcium, Wasserman and Fullmer (1995) believed that an integral membrane transporter resides in the brush border membrane.

#### B. Intracellular calcium transfer

Calbindin acts as an intracellular ferry to transfer calcium through the cell to the basolateral membrane (Feher, 1983). The intracellular calcium gradient between the brush border and the basolateral membrane is increased by the presence of calbindin, provinding the force needed for calcium diffusion.

The key to calcium diffusion to the basolateral membrane involves: 1) the calcium binding sites associated with the brush border membrane of intestinal microvilli (Wilson and Lawson, 1980); 2) calbindin present within the cytoplasm (Glenney and Glenney, 1985); and 3) the basolateral calcium pump and sodium/calcium antiporter (Wasserman et al., 1982).

## C. Calcium extrusion

The extrusion of calcium from the cell occurs againist an electrochemical gradient. Two systems are available to extrude calcium against the gradient, the ATP-dependent plasma membrane calcium pump and the sodium/calcium exchanger.

The plasma membrane calcium pump (PMCP) transports 1 Ca<sup>2+</sup> per ATP. The transport is ignited by the ATP dependent phosporylation of aspartyl residue, and the activity of the pump is augmented by serine and threonine residues by protein kinase (Penniston and Enyedi, 1994; Carafoli, 1991).

The PMCP is the product of a multigene family; an increase in PMCP gene expression by 1,25-(OH)<sub>2</sub> D<sub>3</sub> is partial due to a transcriptional event (Pannabecker et al., 1995). Administration of 1,25-(OH)<sub>2</sub> D<sub>3</sub> to vitamin D deficient chicks increases the synthesis of the PMCP of the cell (Wasserman et al., 1992b). Wasserman et al. (1992a) reported that calbindin can stimulate the activity of the PMCP. In addition, Timmerman et al. (1995) reported that parvalbumin, another high affinity binding protein, also sitmulates PMCP activity. The exact mechanism of how these proteins affect PMCP is not exactly known.

The extrusion of Ca<sup>2+</sup> by the sodium/calcium exchanger is linked to the downward gradient of sodium into the cell. The sodium/calcium exchanger transfers 3 Na<sup>+</sup> per 1 Ca<sup>2+</sup>. The estimated thermodynamic potential is, slightly less than the energy to extrude calcium (Baker, 1986). However, the Ca<sup>2+</sup> binding affinity of the sodium/calcium exchanger was reported to vary from 0.1 to 8  $\mu$ M (Kikuchi et al., 1988; Schoenmaker and Flik, 1992), a range appropriate for

the extrusion of Ca<sup>2+</sup> across the basolateral membrane. Ghijsen et al. (1983) reported that the sodium/calcium exchanger is not vitamin D dependent.

## Calpain Proteases

Proteolysis of myofibrillar proteins by calpain proteases is the major contributor to tenderization of beef during postmortem aging (Goll et al., 1983a; Tarrant, 1987). Two forms of calpains,  $\mu$ -calpain and m-calpain exist in the cytosol of mammalian muscle tissue (Goll et al., 1983b; Murachi, 1985). Activation of  $\mu$ -calpain requires 50 to 70  $\mu$ M calcium while m-calpain requires 1 to 5 mM calcuim (Dayton et al., 1981; Szpacenko et al., 1981). The myofibular proteins troponin t and desmin are degraded by calcium dependent proteases (Koohmaraie et al., 1986), as are Z-disks (Takahashi et al., 1987. The rapid loss of  $\mu$ -calpain during normal postmortem storage indicates that  $\mu$ -calpain, unlike mcalpain, is activated under postmortem conditions (Vidalence et al., 1983; Ducasting et al., 1985; Koohmaraie et al., 1987). Vidalence et al. (1983) demonstrated that both calpains are activated in the presence of 3 mM Ca<sup>2+</sup> where as only  $\mu$ -calpain is activated at 50  $\mu$ M Ca<sup>2+</sup>, a concentration that can be reached in muscle cells during postmortem storage.

#### Vitamin D<sub>3</sub> Effects on Meat Quality

Feeding increased levels of supplemental vitamin D<sub>3</sub> can improve meat tenderness. By elevating calcium levels to the extent of activating the calpain proteolytic system. Swanek et al. (1999) reported that steers supplemented with 5 million IU of vitamin D<sub>3</sub> for 5 d and 7.5 million IU of vitamin D<sub>3</sub> for 10 d prior to harvest produced steaks with a reduced Warner-Bratzler shear force values (WBS) by 6.6% and 18.0% respectively, at 7 d postmortem storage. However, Montgomery et al. (1998) reported that the maximal improvement of steaks from steers supplemented with 5 and 7.5 million IU of vitamin D<sub>3</sub> 9 d prior to harvest occurred 14 d postmortem storage. Moreover, Montgomery et al. (1998) concluded that the 5 and 7.5 million IU doses were equally effective in improving meat tenderness. In contrast to these findings, Enright et al. (1998) found that pigs supplemented with moderate (55,031 IU/Kg feed) and high (176,000 IU/Kg feed) levels of vitamin D<sub>3</sub> did not effect WBS values.

## Vitamin D Toxicity

Excessive amounts of vitamin D in the diet can produce a variety of effects, all associated with an abnormal elevation of blood calcium. Factors such as potency of chemical form, species, stage of production, route and duration of administration may influence the degree of vitamin D intoxication.

Vitamin D potency is species dependent. Vitamin D<sub>3</sub> has a greater biological activity than vitamin D<sub>2</sub> in rats and pigs (Horst et al., 1982) and in New World Monkeys (Hunt et al., 1969). When given in excessive amounts, vitamin D<sub>3</sub> is 10 to 20 times more toxic to ruminants than vitamin D<sub>2</sub> (NRC, 1987).

Route of administration, (intravenous > intramuscular > oral) influences vitamin D toxicity. A dose of 15 million IU of vitamin D<sub>3</sub> administrated Parenteral resulted in toxicity and death in pregnant dairy cows (Littledike and Horst , 1982). However, Hibbs and Pounden (1955) reported that oral administration of 30 million IU of vitamin D<sub>2</sub> daily for 21 to 30 d resulted in little or no toxicity in pregnant dairy cows.

Pregnant cows are more likely to develop vitamin D toxicity than non pregnant cows (Littledike and Horst , 1980). In the cow, the potential of toxic effects resulting from vitamin D<sub>3</sub> treatment is greater preceding parturition due to an increase in  $1\alpha$ , 25-dihydroxyvitamin D<sub>3</sub> receptors during pregnancy and lactation (Goof et al., 1991). The number of 1,25-dihydroxyvitamin D<sub>3</sub> receptors decrease with age in the cow (Horst et al., 1990).

Duration of vitamin D supplementation may affect the severity of vitamin D toxicosis. For most species, the maximal level of vitamin D<sub>3</sub> for long term supplementation (greater than 60 d) is 4 to 10 times their dietary requirement. During short term supplementation (less than 60 d), most species can tolerate up to 100 times their dietary requirement.

The primary sign of vitamin D toxicity in cattle is inappetence, followed by extensive weight loss, reduced rumination, depression, rough dry coat, delayed shedding, reduced milk production, inflammation and stiffness of joints and muscles, thinning of bones, premature ventricular systoles and bradycardia, and decreased concentration of blood magnesium (Littledike and Horst; 1982; NRC, 1987; McDowell, 1989).

The main pathological effect of ingestion of excessive amounts of vitamin D is widespread calcification of soft tissue, with the collecting tubules of the medulla in the kidney being the primary sight. Cardiovascular lesions as a result of extensive calcification are located primarily in the aorta, aorta valves, aortic arch, arteriole wall and small vessels (NRC, 1987). Glandular lesions of the parotid salivary gland and cartilage calcification may occur (Kent et al., 1958; McDowell, 1989).

#### CHAPTER III

#### MATERIALS AND METHODS

# Trials 1 and 2

In trials 1 and 2, 80 crossbred steers (590 kg mean live weight) were fed a conventional finishing diet fortified with 400 IU/hd/d of vitamin E for the last 100 d of the feeding period; supplemented with either 0 (CON) or 6 million IU/hd/d of vitamin D<sub>3</sub> (VITD) for four d immediately prior to harvest. Steers were randomly assigned to 2 pens in each trial, with 20 steers per pen An adjustment period of 18 d for trial 1 and 16 d for trial 2 was allocated prior to feeding the vitamin D<sub>3</sub> supplement. Feed consumption (kg fed/pen) was recorded daily. Individual steer weights were recorded d 1 of the adjustment period and d of harvest.

#### Animal Harvesting

Animals were harvested at a commercial packing plant in Booker, Texas. During exsagination, blood samples were collected utilizing EDTA tubes and centrifuged to obtain plasma. Plasma samples were placed on ice and transported to Oklahoma State University for quantification of ionized calcium using a blood gas analyzer (Ciba Corning, model 288).

Following a 24 h postmortem chilling period (2° C), quality and yeild grade factors were collected by a USDA grader.

# Subprimal Processing Trials

Carcasses were transported via refrigerated (3° C) truck to a commercial processing facility in Oklahoma City, Oklahoma where designated strip loin (IMPS # 180) and top sirloin (IMPS # 184) subprimals were individually identifed and collected from the left side of each carcass. Followng collection, suprimals were vacuum packaged and transported to the Food and Agriculture Products Research Center on the Oklahoma State University campus. Following 7 d of postmortem aging, strip loin and top sirloin subprimals were each fabricated into three 8.8 cm sections. Each section was further divided into three 2.54 cm longissimus muscle or gluteus medius muscle steaks.

Following a 7, 14 or 21 d storage period steaks were individually vacuum packaged and stored at 4°C until analysis. The two remaining strip loin and top butt sections were aged for 14 and 21 d at 4°C until fabrication.

#### a-Tocopherol Tissue Concentrations of Longissimus Muscle Steaks

Tissue samples (5 g) were excised from the strip loin to determine tissue  $\alpha$ tocopherol concentration. Concentrations of  $\alpha$ -tocopherol in muscle tissues were determined following sponification and extraction, by reverse-phase high pressure liquid chromatography and fluorescence detection (Liu et al., 1996).

## Shear Force Evaluation

Steaks were randomlized and thawed at 4°C for 24 hours, then cooked to an an internal temperature of 70°C using an impingement convection oven (Lincoln Impinger, model 1132-000-A). Cooked steaks were then allowed to cool

to 25° C before removing six cores parallel to the orientation of the muscle fibers. Shear force values were determined for each core using a Universal Instron testing machine (Model 4502, Instron, Canton, MS) with a Warner-Bratzler attachment. Shear force values for individual steaks were calculated by averaging the shear force values generated by each steaks respective core.

## Shelf Life Analysis

Steaks (Longissimus and Gluteus Medius muscles) were placed individually in styrofoam trays, overwrapped with polyethylene film and displayed in a retail case under fluorescence lamps (sylvania, 75 watts) for 7 d at a temperature ranging from 2° to 3° C. Steaks were randomly arranged daily prior to 8:00 a.m. A three member trained visual panel evaluated the steaks twice daily at 8:00 a.m. and at 5:00p.m. for lean color, fat color, percent discoloration and overall appearance (Tables I and II). Immediately following visual appraisal, color measurements using the L\*, a\*, and b\* color space setting of a Minolta colorimeter (Minolta Chroma Meter CR-300, Minolta Corp). were obtained at three locations per steak.

# Sensory Evaluation

Steaks (Longissimus and Gluteus Medius muscles) were randomly selected and thawed for 24 hours at 2°C, then broiled on an impingement convection oven to an internal temperature of 70°C. Once reaching 70°C steaks were individually placed in an aluminum container and transported to the sensory evaluation laboratory. Four to six trained sensory panelists evaluated steaks for overall tenderness, juiciness, connective tissue amount, and flavor intensity (Tables III and IV).

## Trial 3

In trial 3, 39 steers (573 kg mean live weight) were fed a finishing diet fortified with 400 IU/hd/d of vitamin E for the last 100 d of the feeding period; supplemented with either 0 (CON) or 6 million IU/hd/d of vitamin D<sub>3</sub> (VITD) for 6 d immediately prior to harvest. An adjustment period of 7 d was allocated prior to feeding the vitamin D<sub>3</sub> supplement. Individual steer weights were recorded d 1 of the adjustment period and d of harvest.

# Suprimal Processing

An additional Longissimus muscle steak was removed from anterior end of the strip loin of designated carcasses for shear force evaluation following a 7 d aging period

#### Statistical Analysis

Each dependent variable associated with the shear force, sensory evaluation, minolta, and shelf life data was analyzed as a split split plot with the main units in a Completely Randomized Design. Sensory evaluation data was blocked by panel member. In each case, treatment was the main unit treatment factor, cut was the sub unit treatment factor and storage time was the sub sub unit treatment factor. Proc GLM and Proc MIXED in SAS were used for these analyses. A 2x3 factorial in a Completely Randomized Design was used to analyze the plasma calcium data using proc GLM. Chi-square test in Proc FREQ were used to analyze distributions of quality grade, marbling score, KPH, preliminary yield grade and yield grade among treatments.
Lean Color (oxygenated)	Fat Color
8 - Bright Cherry-Red	8 - Creamy white
7 - Moderately Bright-Cherry-Red	7 - Mostly creamy white
6 - Cherry-Red	6 - Slightly tan
5 - Slightly Dark-Red	5 - Tan
4 - Moderately Dark-Red or Brown	4 - Light brown
3 - Dark-Red or Brown	3 - Moderately brown
2 - Very Dark-Brown	2 -Brown or slightly green
1 - Extremely Dark Brown or Green	1 - Dark brown or green

Table I. Visual evaluation scales for lean color and fat color.

Table II.	Visual evaluation	scales	for per	rcent d	liscoloration	and	overall
	appearance.						

Percent Discoloration	Overall Appearance
7 - None	7 - Extremely desirable
6 - 1-10	6 - Desirable
5 - 11-25	5 - Slightly desirable
4 - 26-50	4 - Acceptable
3 - 51-75	3 - Slightly undesirable
2 - 76-99	2 - Undesirable
1 - Complete	1 - Extremely undesirable

Juiciness	Cooked Beef Fat	Overall Tenderness
8 Extremely Juicy 7 Very Juicy 6 Moderately Juicy 5 Slightly Juicy 4 Slightly Dry 3 Moderately Dry 2 Very Dry 1 Extremely Dry	2 Very Strong 1 Slightly detectable 0 Non Detectable	8 Extremely Tender 7 Very Tender 6 Moderately Tender 5 Slightly Tender 4 Slightly Tough 3 Moderately Tough 2 Very Tough 1 Extremely Tough

TABLE III. Sensory Evaluation Scale for Juiciness, Cooked Beef Fat and Overall Tenderness

### Table IV. Sensory Evaluation Scale for Connective Tissue Amount, Flavor Intensity, and Off Flavor

Connective Tissue Amount	Flavor Intensity	Off-Flavor
8 None	8 Extremely Intense	4 None
7 Practically None	7 Very Intense	3 Slightly
6 Traces	6 Moderately Intense	2 Moderate
5 Slight	5 Slightly Intense	1 Intense
4 Moderate	4 Slightly Bland	
3 Slightly Abundant	3 Moderately Bland	
2 Moderately Abundant	2 Very Bland	
1 Abundant	1 Extremely Bland	

### CHAPTER IV

### RESULTS AND DISSCUSON

Trials 1 and 2

### Live Performance

Simple statistics for live performance are represented in Table V. Dry matter intake was not statistically different (P>.05) for steers included in trials 1 and 2. The VITD steers exhibited a gradual decrease of DMI through d 3 of the vitamin  $D_3$  supplementation period. This may be due to the on set of vitamin D induced hypercalcemia. However, on d 4 DMI increased .99 kg/hd presumably due to chance.

### Carcass Traits

In trials 1 or 2, no significant differences (P>.05) were detected for carcass weight, dressing percentage, Longissimus muscle area, yield grade and quality grade. However, carcass weight, dressing percentage and percentage of cattle grading premium and low choice tended to be greater for VITD cattle compared to CON cattle (Tables VI and VII). Although, VITD cattle exhibited lighter final live weights than CON, results indicate these cattle possessed heavier, leaner carcasses with a higher degree of marbling.

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### a-Tocopherol Tissue Anaylsis

Tissue from carcasses originating from cattle fed VITD had  $\alpha$ -tocopherol levels that were not different from their CON counterparts (Figure 4).

### Shear Force Anaylsis

In trials 1 and 2, no significant differences were detected, for Longissimus or Gluteus medius muscle steaks within an aging period (Tables VIII and IX). However, following a 7 d postmortem aging period Longissimus steaks for VITD carcasses tended to have a lower shear force values when compared to CON steaks (4.47 kg vs 4.32 kg). However, similar values were observed between longissimus steaks for VITD and CON after aging exceeded 7 days.

### Objective Color Measurements

Results from the Minolta colorimeter readings, for trials 1 and 2, indicated no significant differences for L\*, a\* or b\* values for Longissimus or Gluteus Medius muscle steaks between CON or VITD groups (Figures 5-10). Although, the CON steaks tended to have consistently higher L\*, a\* and b\* values.

### Visual Panel Evaluation

Panelist scores for lean color, fat color, percentage of lean discoloration and overall appearance were significantly higher (P<.05) for Longissimus muscle steaks at 7 d of aging compared to their Gluteus medius counterparts (Figures 12-15). Visual color measurements suggested that Gluteus medius muscle steaks were less desirable in their lean color, displayed browner fat, more discolored and had less desirable overall appearance in all categories across aging periods than Longissimus muscle steaks. Results from trials 1 and 2 suggest that Longissimus muscle steaks aged 7 d possessed more desirable lean color descriptive scores for all visual categories (Figures 12-15).

### Sensory Evaluation

No significant differences were detected by panelist between VITD or CON Longissimus muscle steaks, regardless of aging period, for juiciness, beef fat flavor, overall tenderness, connective tissue amount, flavor intensity or off flavor (Tables X, XII and XIV). However, Gluteus medius muscle steaks from CON carcasses aged 7 d exhibited a significantly greater (P<.05) overall tenderness score (Table XI), although this significance was not detected in the WBS value. Gluteus medius muscle steaks aged 14 and 21 d did not display statistically significant shear force values to those of VITD steaks (Tables XIII and XV).

	Tre	eatment*	
	0	6*10 <sup>6</sup> VIT D <sub>3</sub>	
Supplementation, d	DMI/hd	DMI/hd	D₃/IU/hd
1	9.30	9.81	6,707,847
2	9313	9.38	6,208,760
3	9.28	6.95	4,601,641
4	7.83	7.95	5,260,022

TABLE V.	Mean dry matter intake (DMI/hd) and vitamin D <sub>3</sub> consumption
	(IU/hd) for steers supplemented with either 0 or 6 million IU of
	vitamin D <sub>3</sub> for 4 days prior to harvest (Trial 1 and 2).

\*No significant difference between dry matter intake treatment means (P>.05).

	Trea			
	Vitamir	n D <sub>3</sub> , IU/d	<del>.</del>	
Item	0	6 * 10 <sup>6</sup>	SEM	Р
Steers, N	40	40		
Initial Live Weight, kg	588.27	593.68	37.64	.65
Final live weight, kg	609.21	608.73	38.35	.95
Carcass weight, kg	378.51	380.10	24.39	.83
Dressing Percentage	64.71	65.10	.51	.28
Ribeye area, cm <sup>2</sup>	89.74	88.12	.51	.45

Table VI. Least squares means for carcass traits of steers supplemented with either 0 or 6 million I.U. of vitamin D<sub>3</sub> per day for 4 days prior to harvest (Trials 1 and 2).

	Treatment			
-	Vitamin D <sub>3</sub> , IU/d			
Characteristic	0	6 * 10 <sup>6</sup>	Distribution of Characteristic Value	P>X <sup>2</sup>
Preliminary Yield Grade			1.5 (0.1) 4.4	.68
Internal (KPH) Fat, %			1.5 (0.5) 4.0	.80
Final Yield Grade			1.0 (1.0) 4.0	.82
Yield Grade 1 %	15.0	12.5		
Yield Grade 2 %	47.50	40.0		
Yield Grade 3 %	30.0	40.0		
Yield Grade 4 %	7.5	7.5		
Marbling			320 (10) 570	.31
Quality Grade				.10
Premium Choice %	7.5	10.0		
Low Choice %	20.0	40.0		
Select %	72.5	50.0		
Standard %	0.0	0.0		

Table VII. Chi square distributions for carcass traits of steers supplemented with either 0 or 6 million IU of vitamin D<sub>3</sub> per day for 4 days prior to harvest (Trials 1 and 2).

	Treatment			
	Vitamin D <sub>3</sub> , IU/d		<b>—</b> 1	
Characteristic	0	6 * 10 <sup>6</sup>	Distribution of Characteristic Value	P>X <sup>2</sup>
Preliminary Yield Grade			1.5 (0.1) 4.4	.68
Internal (KPH) Fat, %			1.5 (0.5) 4.0	.80
Final Yield Grade			1.0 (1.0) 4.0	.82
Yield Grade 1 %	15.0	12.5		
Yield Grade 2 %	47.50	40.0		
Yield Grade 3 %	30.0	40.0		
Yield Grade 4 %	7.5	7.5		
Marbling			320 (10) 570	.31
Quality Grade				.10
Premium Choice %	7.5	10.0		
Low Choice %	20.0	40.0		
Select %	72.5	50.0		
Standard %	0.0	0.0		

Table VII. Chi square distributions for Carcass traits of steers supplemented with either 0 or 6 million IU of vitamin D<sub>3</sub> per day for 4 days prior to harvest (Trials 1 and 2).



Figure 4. Treatment means\* for alpha-tocopherol tissue concentrations of Longissimus muscle steaks

\*No significant difference between treatment means.

<sup>1</sup>Control = Steaks from cattle supplemented with vitamin E for 100 days prior to harvest; Vitamin D = Steaks from cattle supplemented with 400 IU of vitamin E for 100 days prior to harvest and  $6*10^6$  IU of vitamin D for either 4 or 6 days prior to harvest.

35

	Treatment			
Aging Period	CON	VITD	SEM*	P Value =
7 d	4.47	4.32	.35	.66
14 d	3.64	3.70	.35	.88
21 d	3.68	3.55	.35	.71

Table VIII .	Treatment <sup>1</sup>	Warner-Bratzler shear force means for Longissimus
5	steaks strati	fied by aging period (Trials 1 and 2).

<sup>1</sup>Treatment = steers supplemented with either 0 or 6 million IU of vitamin  $D_3$  for 4 days prior to harvest. \*Standard error of the mean represents standard error for difference between treatment means.

	Treat	tment		
Aging Period	CON	VITD	SEM*	P Value
7 d	4.33	4.61	.37	.45
14 d	3.85	3.60	.36	.67
21 d	3.62	4.18	.36	.12

Table IX .	Treatment	Warner-Bratzler shear force means for Gluteus medius	5
	steaks st	ratified by aging period (Trials 1 and 2).	

<sup>1</sup>Treatment = steers supplemented with either 0 or 6 million IU of vitamin  $D_3$  for 4 days prior to harvest. \*Standard error of the mean represents standard error for difference between treatment means.



Figure 5. Effect of treatment<sup>a</sup> within an aging period on mean L\* values of Longissimus muscle steaks (Trials 1 and 2)

"No significant difference due to treatment within an aging period.

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Figure 6. Effect of treatment<sup>a</sup> within an aging period on mean L\* values of Gluteus Medius muscle steaks (Trials 1 and 2)

<sup>a</sup>No significant difference due to treatment within an aging period.

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Figure 7. Effect of treatment<sup>a</sup> within an aging period on mean a\* values of Longissimus muscle steaks (Trials 1 and 2)

<sup>a</sup>No significant difference due to treatment within an aging period.



Figure 8. Effect of treatment<sup>a</sup> within an aging period on mean a\* values of Gluteus Medius muscle steaks (Trials 1 and 2)

<sup>a</sup>No significant difference due to treatment within an aging period.

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Figure 9. Effects of treatment<sup>a</sup> within an aging period on mean b\* values of Longissimus muscle steaks (Trials 1 and 2)

<sup>a</sup>No significant difference due to treatment within an aging period.

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Figure 10. Effect of treatment<sup>a</sup> within and aging period on mean b\* values of Gluteus Medius muscle steaks (Trials 1and 2)

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<sup>a</sup>No significant difference due to treatment within an aging period.

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Figure 11. Mean lean color scores for steaks stratified by aging period (Trials 1 and 2)

<sup>abcd</sup>Means with same superscript are not different (P<.01).

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<sup>abcde</sup>Mean with same superscript are not different (P<.01).

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<sup>abcde</sup>Means with same superscript are not different (P<.05).



Figure 14. Mean overall appearance scores for steaks statified by aging period (Trials 1and 2)

<sup>&</sup>lt;sup>abcdef</sup>Means with same superscripts are not different (P<.05).

	Treat		
Characteristic <sup>*a</sup>	CON	VITD	SEM*
Juiciness	5.09	5.24	.21
Beef fat flavor	.38	.48	.21
Overall tenderness	4.65	4.56	.21
Connective tissue amount	5.23	5.02	.29
Flavor intensity	5.35	5.43	.21
Off flavor	3.94	3.88	.08

# Table X . Sensory least squares means for Longissimus muscle steaks aged 7 days.

Juiciness: 1=extremely dry, 8=extremely juicy; Beef fat flavor: 0=none, 2=very strong; Overall tenderness: 1=extremely tough, 8=extremely tender; Connective tissue amount: 1=abundant, 8=none; Flavor intensity: 1=extremely bland, 8=extremely intense; Off-flavor: 1=intense, 4=none. \*No significant difference between treatment means for any characteristic.

	Treat		
Characteristic	CON	VITD	SEM*
Juiciness	4.84	4.46	.38
Beef fat flavor	.68	.97	.20
Overall tenderness	4.06 <sup>a</sup>	3.38 <sup>b</sup>	.31
Connective tissue amount	4.63	4.64	.27
Flavor intensity	5.09	5.07	.20
Off flavor	3.86	4.05	.08

## Table XI. Sensory least squares means for Gluteus medius muscle steaks aged 7 days.

Juiciness: 1=extremely dry, 8=extremely juicy; Beef fat flavor: 0=none, 2=very strong; Overall tenderness: 1=extremely tough, 8=extremely tender; Connective tissue amount: 1=abundant, 8=none; Flavor intensity: 1=extremely bland, 8=extremely intense; Off-flavor: 1=intense, 4=none. <sup>ab</sup>Significant difference for overall tenderness (P=.03).

	Treat		
Characteristic <sup>ab</sup>	CON	VITD	SEM*
Juiciness	4.60	4.50	.21
Beef fat flavor	.35	.42	.21
Overall tenderness	4.81	5.21	.32
Connective tissue amount	5.19	5.42	.29
Flavor intensity	4.98	5.19	.21
Off flavor	3.88	3.90	.08

# Table XII. Sensory least squares means for Longissimus muscle steaks aged 14 days.

.

<sup>a</sup>Juiciness: 1=extremely dry, 8=extremely juicy; Beef fat flavor: 0=none, 2=very strong; Overall tenderness: 1=extremely tough, 8=extremely tender; Connective tissue amount: 1=abundant, 8=none; Flavor intensity: 1=extremely bland, 8=extremely intense; Off-flavor: 1=intense, 4=none. <sup>b</sup>No significant difference between treatment means for any characteristic.

	Treat		
Characteristic <sup>ab</sup>	CON	VITD	SEM*
Juiciness	4.32	4.72	.22
Beef fat flavor	.49	.79	.21
Overall tenderness	4.34	4.60	.34
Connective tissue amount	4.80	5.04	.31
Flavor intensity	5.17	5.32	.22
Off flavor	3.82	3.96	.09

## Table XIII. Sensory least squares means for Gluteus Medius muscle steaks aged 14 days.

<sup>a</sup>Juiciness: 1=extremely dry, 8=extremely juicy; Beef fat flavor: 0=none, 2=very strong; Overall tenderness: 1=extremely tough, 8=extremely tender; Connective tissue amount: 1=abundant, 8=none; Flavor intensity: 1=extremely bland, 8=extremely intense; Off-flavor: 1=intense, 4=none. <sup>b</sup>No significant difference between treatment means for any characteristic.

	Treatment		
Characteristicab	CON	VITD	SEM*
Juiciness	4.56	4.49	.21
Beef fat flavor	.55	.45	.21
Overall tenderness	5.07	5.15	.32
Connective tissue amount	5.29	5.38	.29
Flavor intensity	5.10	5.32	.21
Off flavor	3.91	3.97	.08

# Table XIV. Sensory least squares means for Longissimus muscle steaks aged 21 days.

<sup>a</sup>Juiciness: 1=extremely dry, 8=extremely juicy; Beef fat flavor: 0=none, 2=very strong; Overall tenderness: 1=extremely tough, 8=extremely tender; Connective tissue amount: 1=abundant, 8=none; Flavor intensity: 1=extremely bland, 8=extremely intense; Off-flavor: 1=intense, 4=none. <sup>b</sup>No significant difference between treatment means for any characteristic.

	Treat		
Characteristic <sup>ab</sup>	CON	VITD	SEM*
Juiciness	4.13	4.12	.20
Beef fat flavor	.43	.83	.20
Overall tenderness	4.38	4.10	.31
Connective tissue amount	5.22	5.29	.28
Flavor intensity	5.13	5.11	.21
Off flavor	4.05	4.08	.08

# Table XX. Sensory least squares means for Gluteus Medius muscle steaks aged 21 days.

<sup>a</sup>Juiciness: 1=extremely dry, 8=extremely juicy; Beef fat flavor: 0=none, 2=very strong; Overall tenderness: 1=extremely tough, 8=extremely tender; Connective tissue amount: 1=abundant, 8=none; Flavor intensity: 1=extremely bland, 8=extremely intense; Off-flavor: 1=intense, 4=none. <sup>b</sup>No significant difference between treatment means for any characteristic.

### CHAPTER V

### RESULTS AND DISSCUSION

Trial 3

#### Live Performance

In trial 3, DMI was not statistically different for CON and VITD cattle (Table XVI). However, VITD steers exhibited a gradual decrease of DMI from d 1 through d 6 of the vitamin  $D_3$  supplementation period. The decrease in DMI may be due to the onset of vitamin D induced hypercalcemia.

#### Carcass Traits

Although, no statistical differences were detected for the treatment groups, VITD cattle exhibited a smaller final live weight, a higher dressing percentage and a greater percentage of carcasses grading low choice than the CON group (Tables XVII and XVIII).

Thus in trials 1, 2 and 3 VITD cattle exhibited a smaller final live weight. However, VITD carcasses possessed a higher dressing percentage and a greater or equal number of premium or low choice quality grades. Suggesting that supplementing feedlot cattle with vitamin D<sub>3</sub> prior to harvest maybe advantageous for selling cattle on a formula basis.

### Ionized Calcium Plasma Concentrations

The VITD cattle exhibited a higher (P<.01) ionized calcium blood plasma concentration than CON group (Figure 15). However, these values are within the normal plasma calcium values of 3.5 to 6.5 mg/dl. This increase may be a result

of vitamin D induced hypercalcemia due to prolonged supplementation of vitamin D<sub>3.</sub>

### Shear Force Anaylsis

The CON Longissimus muscle steaks exhibited a significantly lower (P<.05) Warner-Bratzler shear force value than VITD Longissimus muscle steaks at 14 and 21 d of aging (Table XIX). No significant differences were detected between treatment groups within an aging period for Gluteus medius muscle steaks (Table XX).

### **Objective Color Measurements**

In trial 3, VITD and CON Longissimus muscle steaks exhibited statistically similar mean L\* values (Figure 16). However, VITD Gluteus medius muscle steaks demonstrated higher (P < .01) mean L\* values than CON Gluteus medius muscle steaks at 14 and 21 days of age (Figure 17). Mean b\* values for VITD Longissimus muscle steaks were higher (P < .05) at 14 and 21 d of aging than their CON counterparts (Figure 20). Mean b\* values for CON and VITD Gluteus medius muscle steaks within an aging period were not statistically significant (Figure 21). Longissimus and Gluteus medius muscle steaks from CON and VITD carcasses displayed statistically similar mean a values (Figures 18 and 19).

### Visual Panel Evaluation

Longissimus muscle steaks exhibited a more desirable lean color, fat color, percent discoloration and overall appearance score than Gluteus medius muscle steaks (P<.05) across aging periods (Figures 22-25). Visual color measurements

suggested that Gluteus medius muscle steaks were less desirable in their lean color, displayed browner fat, more discolored and had less desirable overall appearance in all categories across aging periods than Longissimus muscle steaks. Results from trial 3 suggest that Longissimus muscle steaks aged 7 d possessed more desirable scores for all categories (Figures 22-25).

	Treatment*		
	0	6*10 <sup>6</sup> VITD	
Supplementation, d	DMI/hd	DMI/hd	D <sub>3</sub> /IU/hd
1	7.91	9.35	6,184,668
2	7.91	8.51	5,632,466
3	8.96	8.30	5,522,025
4	7.38	7.18	4,748,942
5	8.25	6.69	4,417,620
6	6.90	5.76	3,810,197

TABLE XVI. Mean dry matter intake (DMI/hd) and vitamin D<sub>3</sub> consumption (IU/hd) for steers supplemented with either 0 or 6 million IU vitamin D<sub>3</sub> for 6 days prior to harvest (Trial 3).

\*No significant difference between dry matter intake treatment means (P>.05).

	Treat	nent		
	Vitamin I	D <sub>3</sub> , IU/d		
Item	0	6 * 10 <sup>6</sup>	SEM	Р
Steers, N	19	20		
Initial Live Weight, kg	578.73	567.33	26.31	.57
Final live Weight, kg	584.27	566.99	25.35	.29
Carcass weight, kg	365.27	364.33	17.63	.95
Dressing Percentage	65.39	67.09	1.51	.43
Longissimus muscle area, cm <sup>2</sup>	90.83	90.83	.51	.99

Table XVII. Least squares means for carcass traits of steers supplemented with either 0 or 6 million IU of vitamin D<sub>3</sub> per day for 6 days prior to harvest (Trial 3).

	Treatment			
	Vitamin D <sub>3</sub> , IU/d		-	
Characteristic	0	6 * 10 <sup>6</sup>	Distribution of Characteristic Value	P>X <sup>2</sup>
Preliminary Yield Grade			2.3 (0.1) 4.4	1.00
Internal (KPH) Fat, %			1.5 (0.5) 3.0	.99
Final Yield Grade			1.0 (1.0) 4.0	1.00
Yield Grade 1 %	20.05	21.00		
Yield Grade 2 %	64.16	65.00		
Yield Grade 3 %	10.53	10.00		
Yield Grade 4 %	5.26	4.00		
Marbling			260 (10) 520	1.00
Quality Grade				.99
Premium Choice %	10.26	10.26		
Low Choice %	82.06	87.18		
Select %	4.56	2.56		
Standard %	3.12	0.00		

Table XVIII. Chi square distributions for carcass traits of steers supplemented with either 0 or 6 million IU of vitamin D<sub>3</sub> per day for 6 days prior to harvest (Trial 3).



<sup>t</sup>reatment least squares means differ (P<.01). <sup>1</sup>Trial 3 = Steers supplemented with either 0 or 6 million IU of vitamin  $D_3$  for 6 days prior to harvest

	Treat	ment		
Aging Period	CON	VITD	SEM*	P Value
7 d	4.72	4.85	.29	.67
14 d	3.10 <sup>a</sup>	4.18 <sup>b</sup>	.48	.02
21 d	3.01 <sup>a</sup>	4.15 <sup>b</sup>	.50	.02

Table XIX.	Treatment'	Warner-Bratzler	shear force	means for	Longissimus
steaks stratified by aging period (Trial 3).					

<sup>1</sup>Treatment = steers supplemented with either 0 or 6 million IU of vitamin  $D_3$  for 6 days prior to harvest. \*Standard error of the mean represents standard error for difference between treatment means. <sup>ab</sup>Means within a row with different superscripts differ (P<.05).




\*No significant differences due to treatment within an aging period.

Treatment				
Aging Period	CON	VITD	SEM*	P Value
7 d	3.83	3.94	.53	.82
14 d	3.75	4.58	.53	.12
21 d	4.04	4.17	.53	.81

Table XX.	Treatment' Warner-Bratzler shear force means for Gluteus Medius
	steaks stratified by aging period (Trial 3).

<sup>1</sup>Treatment = steers supplemented with either 0 or 6 million IU of vitamin D<sub>3</sub> for 6 days prior to harvest. \*Standard error of the mean represents standard error for difference between treatment means.



# Figure 17. Effect of treatment within an aging period on mean L\* values of Gluteus Medius muscle steaks (Trial 3)

<sup>abcde</sup>Means within an aging period with the same superscripts are not different (P<.01).



Figure 18. Effect of treatment<sup>a</sup> within an aging period on mean a\* values of Longissimus muscle steaks (Trial 3)

<sup>a</sup>No significant differences due to treatment within an aging period.



Figure 19. Effect of treatment<sup>a</sup> within an aging period on mean a\* values of Gluteus Medius muscle steaks (Trial 3)

\*No significant differences due to treatment within an aging period.



<sup>abcde</sup>Means within an aging period with the same superscript are not different (P<.05).



Figure 21. Effect of treatment<sup>a</sup> within an aging period on mean b\* values of Gluteus Medius muscle steaks (Trial 3)

<sup>a</sup>No significant difference due to treatment within an aging period.



<sup>&</sup>lt;sup>abcde</sup>Means with same superscript are not different (P<.05).



<sup>abcde</sup>Mean with same superscript are not different (P<.01).



<sup>abcd</sup>Means with same superscript are not different (P<.05).





<sup>abcdef</sup>Means with same superscripts are not different (P<.05).

#### CHAPTER VI

## SUMMARY AND CONCLUSIONS

Steers supplemented with VITD tended to exhibit a gradual decrease (P>.05) In DMI during the supplementation period. Final live weights were lighter (P>.05) for VITD cattle compared to their counterparts; however, their dressing percentages were greater than CON (P>.05). Carcasses from VITD cattle expressed a greater or equal percentage of premium and low choice quality grades compared to CON carcasses The results suggests that it may be advantageous to sell feedlot cattle supplemented with vitamin D<sub>3</sub> on a formula basis. However, the optimum dosage rate and duration of feeding supplemental vitamin D<sub>3</sub> to feedlot cattle to avoid a decrease in dry matter intake remains to be determined.

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