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HAIR COAT AND STEROIDAL IMPLANT EFFECTS ON STEERS GRAZING NDOPHYTE-INFECTED TALL FESCUE DURING THE SUMMER

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Sixty steers were grazed on toxic tall fescue for 104 days to determine the effects of hair coats and steroidal ear implants on physiological measurements. Steers were stratified by body weight and hair coat color for assignment to six, 3.0-ha pastures of ‘Kentucky 31’ tall fescue. Main plot treatments of either ten clipped or ten unclipped steers were randomly assigned to pastures. Five steers in each pasture were implanted with Synovex-S (200 mg progesterone-20 mg estradiol) and five were implanted with Compudose (25 mg estradiol) as sub-plot treatments. Hair growth rate averaged 0.29 mm/day. Sweating rate declined (P < 0.001) over the grazing period and was higher (P< 0.10) with the estradiol implant. Rectal temperatures were lower (P < 0.05) in clipped cattle (39.3 vs. 39.5°C) when the highest ambient temperature (33°C) of the study was recorded. At high environmental temperatures, percentage of steers actively grazing was negatively correlated (P < 0.10) with ambient temperature. Winter hair coat retention, continuous hair growth, and reduced sweating caused impaired thermoregulation and thus decreased grazing frequency resulting in poor animal performance.

KEYWORDS: Beef Cattle, Fescue Toxicosis, Heat Stress, Hair Coat, Steroidal Implant

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HAIR COAT AND STEROIDAL IMPLANT EFFECTS ON STEERS GRAZING ENDOPHYTE-INFECTED TALL FESCUE DURING THE SUMMER

THESIS

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science in the College of Agriculture at the University of Kentucky

By

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Lexington, Kentucky

2007

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Chapter One: Introduction

Tall fescue [Schedonorus arundinaceus (Schreb.) Dumort; Soreng et al., 2001] is the most abundant cool-season grass in the United States covering approximately 15 million hectares (Bacon and Siegel, 1988). It is widely adapted to the area referred to as the transition zone or the “Fescue Belt,” covering the southeastern US. Upon its release in 1943, the Kentucky 31 tall fescue cultivar became widespread due to the distinct agronomic advantages it possesses. In the 1970s, scientists discovered tall fescue was infected with an endophyte that is responsible for the agronomic advantages this forage offers but also was responsible for poor animal performance that had became associated with cattle grazing fescue (Lacefield et al., 2004). Alkaloids produced by the endophyte have been determined to be the cause of three syndromes observed in animals consuming toxic tall fescue: fescue foot, fat necrosis, and fescue toxicosis or summer syndrome. Fescue foot and fat necrosis are typically observed in isolated incidences and do not cause the widespread economic losses associated with fescue toxicosis. In 1993, Hoveland reported that economic losses to the cattle industry were greater than $600 million annually from fescue toxicosis.

Fescue toxicosis symptoms include poor weight gain, reduced conception rates, rough (winter) hair coat retention, low serum prolactin, constricted blood flow, and decreased heat tolerance (Schmidt and Osborn, 1993; Strickland et al., 1993; Thompson and Stuedemann, 1993). Cattle grazing endophyte-infected fescue often suffer from heat stress at high ambient temperatures and humidities and, consequently, spend less time grazing which lowers intake (Hemken et al., 1981; Parish et al., 2003). Various factors influence susceptibility to heat stress including hair coat characters, blood flow, and heat
loss mechanisms. Clipping rough hair coats of both European and tropical breeds of cattle has been found to alleviate heat stress (Turner, 1962; Pathmasingham and Abdul, 1981) and cause increased weight gains. However, no research has been conducted on hair coats of cattle grazing endophyte-infected tall fescue.

Various management strategies have been evaluated in an effort to develop ways to manage cattle grazing toxic tall fescue without incurring the production losses associated with fescue toxicosis. A lot of effort has been focused on diluting the alkaloids in the diet by either interseeding clovers in fescue pastures or supplemental feeding of concentrates while cattle graze endophyte-infected fescue. Another management approach is to use estrogenic ear implants that increase feed efficiency and rate of gain. However, some implants contain compounds that may cause vasodilation (Vacca et al., 1999; Edwards, 2005). The use of these implants in stocker cattle grazing toxic fescue may alleviate the vasoconstriction caused by the alkaloids and allow for improved heat dissipation and, consequently, less vulnerability to heat stress. There is a possibility, however, that implantation increases heat stress in cattle grazing E+ tall fescue as increased dry matter intake results in greater heat production (Aiken et al., 2006).

A grazing experiment was conducted to evaluate the effects of hair coat retention on heat stress in beef cattle exhibiting fescue toxicosis and to determine if estradiol implants will alleviate heat stress.
Chapter Two: Literature Review

Tall fescue is a cool-season perennial grass that occupies approximately 15 million hectares (Bacon and Siegel, 1988; Stuedemann and Hoveland, 1988; Lacefield et al., 2004) in the United States. Tall fescue is widely adapted to the environmental conditions of a region referred to as the “transition zone” or “Fescue Belt”, a region which extends from Virginia to Georgia and west to the Great Plains. Improvement of tall fescue began in 1931, when Dr. E. N. Fergus of the University of Kentucky collected a tall fescue landrace from a hillside on the W. M. Suiter farm in Menifee County, KY. Following evaluation, the cultivar ‘Kentucky-31’ was commercially released in 1943; and its distinct agronomic advantages and vigorous promotion by William C. Johnstone (Lacefield et al., 2004) resulted in widespread adoption of tall fescue throughout the region.

Almost immediately farmers and researchers began observing poor animal performance and health, but it was not until the 1970s that these problems were associated with a fungal endophyte. Scientists at Auburn University and the University of Georgia observed cattle in pastures with high levels of endophyte infection performed poorly, while acceptable animal performance was observed in pastures with low infection levels (Lacefield et al., 2004). Researchers discovered that tall fescue was infected with the fungal endophyte, Neotyphodium coenophialum, which produced ergot alkaloids that include clavine alkaloids, lysergic acid amides and ergopeptines (Roberts and Andrae, 2004). The endophyte and the tall fescue plant co-habitate in a mutualistic relationship that provides the endophyte with nutrients, protection, and dissemination through seed,
and imparts improved tolerances to drought, pests, and herbivory to the host plant (Ball et al., 2003; Hoveland, 2004). Tall fescue also offers the agronomic advantages of good dry matter yields with marginal soil fertility needs and a long extended growing season (Ball, 1984). It is estimated that 90-95% of tall fescue grown in the United States is endophyte-infected (Bacon and Siegel, 1988; Shelby and Dalrymple, 1987). Tall fescue occupies approximately 5.5 million acres in Kentucky, of which more than 85 percent of pastures are endophyte infected (Lacefield et al., 2003).

**Fescue Toxicosis**

Consumption of endophyte-infected Kentucky-31 tall fescue results in a range of animal problems collectively termed “fescue toxicosis”. The fescue toxicosis malady is characterized by poor weight gain, impaired reproductive performance, depressed milk production, retention of rough (winter) hair coats into the summer months, low serum prolactin, restricted blood flow to peripheral tissues and heat intolerance (Schmidt and Osborn 1993; Strickland et al., 1993; Thompson and Stuedemann 1993). Other symptoms that accompany fescue toxicosis are decreased feed intake, increased respiration rates, elevated body temperatures, excessive salivation, and decreased time spent grazing with more time spent in shade or standing in ponds (Schmidt and Osborn 1993; Thompson and Stuedemann 1993). Consequently, cattle exhibiting symptoms of toxicosis are vulnerable to severe heat stress at the onset of high ambient temperature and humidity (Hemken et. al., 1981) and have difficulty maintaining their body below the thermoneutral (20-25°C) zone at which heat stress begins (Hahn et al., 1974, Hahn et al., 1992).
Strickland et al. (1993) reported that lower weight gains observed in cattle consuming toxic tall fescue are likely due to decreased feed intake, digestibility, and restricted blood flow to the digestive tract. Grazing behavior is altered in cattle consuming toxic tall fescue by overall decreased intake as well as shifts in grazing patterns to increased nighttime grazing (Howard et al., 1992; Schmidt and Osborn, 1993; Paterson et al., 1995). Cattle also have decreased grazing times when grazing toxic tall fescue resulting in decreased gains. During hot periods of the day cattle tended to remain under shade with excessive salivation, elevated respiration and heart rates and possessed rough hair coats (Bond et al., 1984; Howard et al., 1992). Grazing studies conducted by Parish et al. (2003) showed that cattle grazing endophyte-infected fescue as compared to those grazing endophyte-free fescue spent less time grazing and therefore had lowered DM intake and rumination while also exhibiting more time in idling behavior and standing, a mechanism used to maximize heat loss. Aldrich et al. (1993) found cattle grazing endophyte infected fescue at high ambient temperatures (32°C) had a 22% decrease in feed intake. Further results showed Angus cattle were not able to compensate for time spent in the shade during the day by increased grazing times at night (Aldrich et al., 1993).

Specific ergot alkaloids, such as ergovaline, are thought to be primarily responsible for the ‘fescue toxicosis’ malady even though others are likely to play a role as well (Roberts and Andrae, 2004). Gadberry et al. (2003) found that ergovaline produced only some of the toxicosis symptoms, such as reduced serum prolactin, reduced skin temperature and vasoconstriction. Additionally, cattle on an endophyte-infected diet had lower prolactin levels and body temperatures than those receiving ergovaline
injections demonstrating there are other alkaloids involved. Research has shown
(Browning and Leite-Browning, 1997; Browning et al., 1998) that ergotamine and
ergonovine alter plasma concentrations of various pituitary hormones and
thermoregulation, which indicated these alkaloids also are partially responsible for
toxicosis symptoms. Porter et al. (1990) showed that lowered average daily gain (ADG)
and prolactin in cattle grazing endophyte infected fescue were associated with altered
pituitary and pineal mechanisms and thus increased dopaminergic, serotonergic and
pineal metabolites.

In 1993, Hoveland reported that economic losses to the cattle industry were
greater than $600 million annually with the primary losses attributed to reproductive
problems, low weight gains, and unthrifty appearance causing discounted sale prices for
cattle grazing tall fescue pastures; however, current annual economic losses from fescue
toxicosis are likely near one billion dollars. Stuedemann and Hoveland (1988) estimated
ADG decreases approximately 0.1 lb for every 10% increase in pasture infection rate.
Similarly, Paterson et al. (1995) reported decreases in ADG of 30-100% as compared to
steers grazing endophyte free fescue. Numerous research efforts have focused on various
aspects of fescue toxicosis; however, very little research has been conducted on the
retention of rough hair coats into the summer months and the associated heat stress.

Toxicosis Related Heat Stress

Cattle suffering from fescue toxicosis can undergo severe heat stress at high
ambient temperature and humidity (Ball et al., 2003; Hemken et al., 1981). Classic
symptoms of toxicosis include restricted thermoregulatory ability, decreased feed intake,
increased body temperature, excessive salivation, increased respiration rate, and increased time spent in shade (Aldrich et al., 1993). Rough hair coats observed in the summer and constriction of blood flow to peripheral tissues could explain why cattle inflicted with toxicosis suffer from heat stress. Cattle exhibiting fescue toxicosis often stand in the shade or in ponds, creeks, or mud holes, and will pant and salivate in an attempt to alleviate heat stress and decrease body temperature. Vasodilation has been found to occur in several livestock species under heat stress conditions (Johnson and Proppe, 1996). Under normal conditions, blood flow to the peripheral tissues enables the animal to dissipate body heat, but this ability is impaired in fescue cattle due to constricted blood flow. Rhodes et al. (1991) found reduced peripheral and core tissue blood flows in sheep and cattle consuming high-endophyte diets. Steers on an endophyte-infected diet and subjected to a heat challenge had increased rectal temperatures due to restricted peripheral blood flow reducing ability to transfer heat from the core to the body surface. Browning and Leite-Browning (1997) injected ergot alkaloids intravenously into cattle in a hot environment (32°C) and they responded with higher respiration rates, lowered skin temperature, elevated blood pressure and lowered heart rate. These results provided further evidence that constriction of peripheral blood flow by ergot alkaloids have a major role in promoting heat stress (Browning and Leite-Browning, 1997; Browning et al., 1998).

Results from studies with rats, sheep, and cattle on non-toxic diets indicate serum prolactin concentrations can increase with increases in body temperatures (Francesconi, 1996; Hurley et al., 1981). However, these studies demonstrated this is not the case with fescue steers as ability of the animals to adapt to high body temperatures is limited.
Decreases in prolactin with ergot alkaloid consumption are well documented and it is speculated this is a result of dopaminergic and antiserotonergic activities of the alkaloids (Oliver et al., 1993; Strickland et al., 1993). There is evidence of impaired dopamine and serotonin metabolism due to increased turnover rates of pituitary dopamine and serotonin at increased body temperatures (Porter and Thompson, 1992). This implies that there must also be some other mechanisms inhibiting prolactin secretion in the pituitary.

Thyroid hormones also have a negative correlation with body temperatures (Hurley et al., 1981). From a review of literature, Francesconi (1996) concluded that thyroid activity in several animal species decreases under prolonged heat stress regardless of differences in body composition and nutrition.

Hair Growth/Shedding

Aldrich et al. (1993) indicated that toxicosis is more directly linked to an inability to dissipate heat from the body rather than decreased prolactin concentrations. Al-Haidary et al. (2001) found the primary reason for observed hyperthermia in cattle consuming endophyte-infected diets and placed under a heat challenge was reduced peripheral heat loss. Rough hair coats could also contribute to the reduced ability to dissipate body heat and regulate body temperature. It is well documented that beef cattle exhibiting fescue toxicosis have retention of rough hair coats into the summer months (Hoveland et al., 1983; Schmidt and Osborn, 1993; Thompson and Stuedemann, 1993; Aiken et al., 1998). However, rough hair coats have been regarded as a symptom of toxicosis rather than a major problem. Various factors, such as hair coat, which increase resistance to changes in ambient temperature, influence the animal’s ability to regulate
body temperature (Finch, 1986). Under heat stress, animals which shed their hair coat have normal thermoregulatory ability which allows them to maintain thermoneutrality at higher ambient temperatures (Dowling, 1959; Yeates, 1955). Dowling (1959) observed higher body temperatures in unclipped cattle with winter hair coats than with clipped cattle, which indicated that once the winter hair coat was removed cattle regained their ability to control body temperature. Wooly or rough coated animals fail to stabilize heat regulation (Pathmasingham and Abdul, 1981; Yeates 1955), which partially explains why cattle suffering from fescue toxicosis may be inflicted with severe heat stress. Berman (2005) determined that hair coat length is one of the factors that influence threshold temperatures at which animals begin suffering from heat stress. Hair coats affect heat dissipation and heat stress thus significantly influencing cattle growth rates (Turner and Schleger, 1960; Turner, 1962). Olson et al. (2003) reported that cattle (e.g., *Bos indicus*) with a greater tolerance of high heat and humidity have longer grazing times under these conditions and, consequently, increased growth rate. Body temperatures and respiration rates rise with increases in coat scores (Turner and Schleger, 1960). Cattle with winter coats under high ambient temperature will exhibit greater heat stress than sleek or clipped cattle (Olson et al., 2003; Pathmasingham and Abdul, 1981).

Hair growth and shedding are tightly controlled and regulated by complex mechanisms under hormonal control. Rose et al. (2003) studying mink (*Mustela vison*), Paus and Cotsarelis (1999) studying humans and rats, and Chase (1954) using the rat model determined that hair growth cycles in mammals consist of anagen, catagen and telogen phases. During anagen, hair follicles actively grow hair. The catagen phase is marked by reductions in follicle activity as follicles transition to a period of rest, or
telogen (Ebling et al., 1991; Kobayashi et al., 2005). Stenn and Paus (2001) also characterized a fourth stage of the hair cycle: exogen, in which follicles shed the previously grown hair. Original suggestions were that cattle only showed one period of shedding each year with the summer coat elongating to form the winter hair coat. However, Dowling and Nay (1960) showed that cattle actually have two hair growth cycles per year with each follicle producing at least two hairs per year. Other animals, such as the sheep and mink, have clusters of follicles and secondary follicles not associated with sweat glands. In cattle, Carter and Dowling (1954) only observed primary hair follicles with an associated sweat gland, sebaceous gland, and arrector pili muscle.

Seasonal coat changes occur with great regularity in cattle as well as the fox, mink, horse, sheep and ferret, and are related to photoperiod. Studies with mink (Rose et al., 1984) showed that initiation of shedding is coupled with the growth of the winter coat as daylength decreases, and increasing daylength initiates shedding of the winter hair coat, which is followed by growth of the sleek, dense summer coat. Yeates (1955) showed with Shorthorn cattle that reversing the photoperiod resulted in a reversal in the hair growth cycle. Daylength directly influences cyclic patterns associated with hair growth and shedding while air temperatures may modify the cycle (Berman and Volcani, 1961).

Knowledge is lacking of the hormonal controls of hair coat cycles and changes in cattle. Thompson and Stuedemann (1993) suggested that decreased serum prolactin may be related to hair coat changes in cattle suffering from fescue toxicosis. However, Ebling et al. (1991) concluded prolactin inhibits hair growth and influences shedding. Seasonal decreases in prolactin in the fall induce growth of the winter coat while increases in
prolactin are associated with shedding and development of the summer coat (Porter and Thompson, 1992). Similarly, Thompson et al. (1997) found prolactin administered to mares during the winter induced hair shedding. As cattle suffering from fescue toxicosis have suppressed serum prolactin, levels of the hormone would not be sufficient to inhibit growth or stimulate shedding. Therefore, rough (winter) hair coats and growth is observed during the summer period. However, research with mink has shown that prolactin may not be essential for onset of summer or winter hair growth, but may affect density of hair (Rose et al., 1998).

Seasonal changes in intake, reproduction and coat growth may be under the control of the pineal gland through melatonin secretion (Thompson and Stuedemann, 1993). In a study with cattle, Wetteman et al. (1990) reported that melatonin secretion is increased as daylength decreases, and they suggested that this could stimulate hair growth. Similar results have been found in mink and this indicates the inhibition of prolactin synthesis and secretion by melatonin is responsible for initiating winter fur growth (Rose et al., 1987; Rose et al., 1998). Porter et al. (1993) reported decreased serum melatonin levels with consumption of endophyte-infected tall fescue and suggested that altered serotinergic mechanisms may be responsible for the melatonin decreases.

Berman and Volcani (1961) suggested that interactions between ambient temperature and light could influence thyroid secretion rate and that thermal- and photoperiodic-induced changes in cattle hair coats could be caused by their effects on secretion of the thyroid hormones. Thyroxine has been shown to have direct effects on hair growth and color (Berman, 1960). Estrogen may also lengthen the anagen or growing phase of the hair coat cycle (Paus and Cotsarelis, 1999).
Berman and Volcani (1961) reported that the shedding process occurs gradually throughout the year. However, later research found periods of shedding actually occur in the spring and fall each year (Hayman and Nay, 1961). Research by Dowling and Nay (1960) found a close relationship between hair shedding and initiation of a new hair coat, which suggested there are distinct periods of shedding in the hair coat cycle. The summer hair coat usually consists of short, thick hairs while the winter coat consists of long, thin hairs (Dowling and Nay, 1960; Hayman and Nay, 1961). Hayman and Nay (1961) found a bimodal distribution with hair length-diameter relationship in the winter coat of cattle, indicating that an undercoat is present. However, data did not support presence of an undercoat of hair in the summer. Although hair coat cycles and shedding are primarily influenced by photoperiod, restricted nutrition may delay shedding of the winter coat (Pathmasingham and Abdul, 1981; Wetteman et al., 1990; Gilbert and Bailey, 1991). Delayed shedding of the winter coat in cattle grazing endophyte-infected fescue may be explained through restricted nutrition because of limited dry matter intake. Yeates (1957) supported this idea by showing that cattle of European origin transferred to the equatorial region with increased daylength failed to completely shed their hair coat, and consequently, heat stress of these cattle increased and dry matter intake decreased.

Turner (1962) showed that clipping calves with rough hair coats increased weight gain in the summer and decreased respiration rate, and rectal and skin temperatures, while clipping cattle with sleek hair coats had no effect on weight gain or rectal and skin temperatures. Similar results were found in clipped Hereford cattle in which they maintained a lower rectal temperature (Hammond and Olson, 1994). Turner (1962) further reported that clipping rough hair coats also raised sweating rates. Regrowth of
hair after clipping was more rapid in late summer and fall than in late winter and spring. Similarly, Pathmasingham and Abdul (1981) reported that clipped calves had increased ADG and sweating rates with decreased rectal temperature and respiration rates. Improved temperature regulation from clipping had complex influences on grazing behavior, appetite, endocrine function and metabolism, making it difficult to explain the increases in growth rates (Turner, 1962). O’Bannon et al. (1955) concluded in Shorthorn cattle that clipping hair coats reduced the magnitude of hyperthermia in response to heat stress. Clipping of rough hair coats apparently allows for effective heat dissipation and the ability to maintain thermoneutrality at ambient temperatures that would cause them to experience heat stress if a rough hair coat was present. However, none of the cattle in these studies were grazing endophyte-infected fescue.

**Hair Density**

Conflicting research has been reported on hair density and differences in density between the summer and winter hair coat. Kaszowski et al. (1970) reported in mink the summer coat is flat due to lack of density and that mink grow a dense winter coat. Similarly, Bertipaglia et al. (2005) reported cattle with thick, denser coats to have increased vulnerability to heat stress and lowered fertility as compared to those with sleek, thin coats to promote greater evaporation through the coat layer. Finch (1986) also reported cattle with sleek, shiny coats reflect greater proportions of solar radiation than dense, wooly coated cattle. Thick, dense hair coats like those of the *Bos taurus* cattle exacerbate effects of heat stress by reducing heat flow (Finch et al., 1984). However, Turner and Schleger (1960) reported cattle with sleek, dense coats have lower body
temperature and higher growth rates than deep, wooly coated animals. In a review on heat stress in cattle, Blackshaw and Blackshaw (1994) contrasted the coats of *Bos taurus* and *Bos indicus* and explained the dense, smooth coat of *Bos indicus* cattle was able to reflect solar radiation better and had little insulating capacity. They further concluded dense, light colored coats provide the most protection against solar radiation and allows the most heat loss through the skin.

**Coat Color**

Hair coat color has a significant effect on heat stress. Finch (1986) found that cattle with light colored coats or those with sleek, shiny coats reflected greater amounts of solar radiation than cattle with either dark coats or dense, wooly coats. *Bos taurus* breeds with dark coats have higher body temperature and less weight gain than do those breeds with white coats (Finch 1986). Results from Mader et al. (2002) showed cattle with dark coats had tympanic temperatures 0.2-0.6°C greater than cattle with lighter hair coats while dark coated cattle also had the greatest percentage of animals showing moderate to excessive panting in a thermoneutral climate. These dark coated cattle also tended to bunch together in a group whereas those cattle with light hair coats did not exhibit that behavior. Bertipaglia et al. (2005) observed increased coat surface and rectal temperatures indicating a decreased ability to dissipate heat in black cattle. However, Walsberg (1983) suggested that animals with dark coats may or may not have increased heat gain as a result of solar radiation. After reviewing studies using several mammals, including cattle, and birds, Walsberg (1983) conducted an additional experiment with birds and concluded the heat load on black birds over grey birds was not significant with
the difference in heat load being equivalent to a 1-2°C rise in air temperature. Further conclusions were drawn that explained how varying coat structures and optical properties of hair influenced heat gain as much or more than coat color.

Sweating Rates

Heat dissipation through the respiratory tract by panting and through the skin by sweating are the routes by which heat loss may occur when environmental temperatures exceed body temperatures (Brook and Short, 1960). The most efficient route of evaporative cooling in sheep, cattle, and goats is sweating (Finch, 1986). The ability to dissipate excess heat through sweating allows the animal to maintain and control body temperature at ambient temperatures above the thermoneutral zone or under other stressful conditions. Early research presented conflicting evidence on whether or not cattle can sweat; however, Dowling (1958) reported that cattle sweat and their sweating mechanism was largely responsible for body temperature control. Brook and Short (1960) presented similar evidence in sheep sweating and further determined there are two types of epidermal glands in sheep, a sebaceous gland associated with every hair follicle and a sweat gland associated with each primary hair follicle. Mink have a more complex system of follicles, but are similar to sheep which also have secondary follicles (Rose et al., 2003). Conversely, cattle only possess primary hair follicles. Carter and Dowling (1954) observed in cattle that each hair follicle was associated with an apocrine, or sweat, gland. Active sweat glands have a distribution similar to hair follicles and research shows each hair follicle has a sweat gland with a duct opening onto skin at the base of the hair follicle (Ferguson and Dowling, 1955). Pan et al. (1969) studied sweating rates at
various regions in the body and found lower sweating with the shoulder region in Jersey, and crossbred cattle had the highest sweating rate.

Various factors affect sweat glands and influence gland activity. Finch (1986) described the association that exists between sweat glands and capillary beds as blood supply to the skin is essential for adequate sweat production. In cattle and other species, the rate of sweat production is greatly influenced by the rate of blood flow. Cattle usually have increased blood flow in the skin when under heat stress to allow for increased heat dissipation through sweating. Finch (1986) further explained that the thick, wooly coat of *Bos taurus* breeds prevents heat loss from the skin and that after an initial increase the sweating rate reaches a plateau. Yet another significant difference between the *Bos taurus* and *indicus* breeds is that high humidity apparently negatively affects sweating rate in the *B. taurus* cattle.

Activity of sweat glands is a major source of variation in thermoregulation. Differences between species of cattle in thermoregulatory ability are attributed to differences in sweating due to variation in numbers and activity of sweat glands (Dowling, 1958; Ferguson and Dowling, 1955). Crossbred cattle with *Bos indicus* breeding have been found to have lower sweating rates in the morning and increased rates at midday while the *Bos taurus* breeds have lower sweating rates at midday (Schleger and Turner, 1965). Brook and Short (1960) estimated that sweating in sheep accounted for approximately a third of the heat dissipation, but found that sweat glands of sheep were not as efficient as those of cattle and humans. Results of several studies with different species were used to calculate a ratio of sweat/gland/hour to gland volume to yield units of mg/hr/mm$^3$. Ratios were 2.5, 6.0 and 433 mg/hr/mm$^3$ for sheep, cattle, and humans,
respectively. Allen (1962) suggested reasons for greater heat tolerance in *Bos indicus* cattle breeds and ability to withstand temperatures 10-15°F higher without utilizing heat loss mechanisms is due to greater surface area, lower productivity, and lower heat production. Results from Allen (1962) found Jersey heifers had 60 g/m²/hr greater sweating rate than Zebu at skin temp of 34.4°C.

Differences in thermoregulatory ability among groups of cattle have been attributed to differences in coat character (Dowling, 1956; Turner and Schleger, 1960). Animals which fail to shed their winter coat had 60% less sweat volume than animals that shed their coat (Dowling, 1956). Sweating rates are negatively correlated with coat score as the seasonal variation in coat score accounts for the seasonal variation in maximum sweating rate (Yeates, 1955). Similarly, Pathmasingham and Abdul (1981) found hair coat clipping improved heat dissipation and increased sweating rates and heat loss efficiency, which may decrease respiration rates. Nay (1959) determined differences in morphology of sweat glands between breeds of cattle are also responsible for differences in sweating rate. Brahman and other heat tolerant breeds were found to have large club shaped sweat glands that are coiled at one end while Shorthorns have tubular, coiled glands, which have lower capacity than the club like glands.

Heat tolerant animals show a positive correlation for sweating rate versus body temperature and respiration rates, while heat susceptible animals show a negative correlation (Turner, 1962; Schleger and Turner, 1965; Yeates, 1955). Al-Haidary et al. (2001) found that heifers subjected to a heat challenge by increasing air temperature from 21°C to 31°C were able to double their respiratory and skin vaporization. Consumption of endophyte-infected diets during a constant heat challenge at 32°C resulted in a 50%
reduction in skin vaporization (Aldrich et al., 1993). However, Al-Haidary et al. (2001) did not observe differences between calves on endophyte-infected or non-infected diets in skin or respiratory vaporization during a heat challenge, but they did report higher body temperatures in animals on an endophyte-infected fescue diet.

**Estrogenic Growth Promoters**

Various management strategies have been evaluated to determine the potential of alleviating fescue toxicosis. Estrogenic growth promoters are used widely throughout the cattle industry for growing and finishing cattle. Known as anabolic agents due to their estrogenic activity, research has shown increased growth rate and efficiency with possible endocrine effects from using implants (Brandt, 1993). Estrogen, progesterone and their interaction have been shown to affect thermoregulation in humans and livestock. Estrogen supplementation had no effect on esophageal (core body) temperature of exercising women (Chang et al., 1998); however, other experiments with women have shown supplemental estrogen will cause vasodilation in vascular beds of peripheral tissues (Gudmundsson et al., 1999; Chan et al., 2001). In pigs, intravenous infusion of 17-β estradiol resulted in widespread dilation in vascular beds, with more vasodilation in coronary than mesenteric tissues (Vacca et al., 1999). Progesterone appears to elevate body temperature (Katovich and O’Meara, 1987; Stachenfield et al., 2000). Stachenfield et al. (2000) reported that progestin administration in women elevated esophageal temperature above thresholds for sweating, while estrogen reduced temperature below the threshold. Ford et al. (1977) found similar results in ewes when progesterone administration increased vasoconstriction induced by periarterial adrenergic nerve
stimulation while estrogen administration decreased vasoconstriction. No difference was observed from the control ewes when progesterone and estrogen were administered together. Evidence from Edwards (2005) indicated estrogen can induce vasodilation and progesterone and estrogen can act synergistically to induce vasodilation, while progesterone alone can alleviate agonist-induced vasoconstriction of arteries. Similarly, Samaan and Crawford (1995) showed adding progesterone to estrogen does not modify the effects of adding estrogen alone. In experiments with pigs, Molinari et al. (2001) found progesterone infusion resulted in vasodilation in vascular beds of coronary, mesenteric, renal and iliac tissues and mechanisms responsible involved the release of nitric oxide.

Estrogen implants are used for enhanced growth because of their ability to improve feed conversion efficiency and rate of weight gain (Enright et al. 1990). Brandt (1993) estimated that a 15.1% increase in ADG could be achieved in implanted stocker cattle over gains achieved by non-implanted cattle. Coffey et al. (1992) found increased gains in steers implanted with progesterone-estradiol implants while grazing endophyte-infected fescue as compared to non-implanted steers. Implanted steers grazing endophyte infected fescue at low stocking rates had improved ADG but also had increased rectal temperature, which could exacerbate heat stress experienced in cattle grazing tall fescue (Aiken et al., 2006). Implantation may increase body weight gains; however, further heat stress could be of concern in cattle suffering from toxicosis. Results from Aiken et al. (2006) also showed steroid hormones did not influence shedding of winter hair coats. Even though implantation allows for increased gains in cattle grazing endophyte-infected fescue, the gains while grazing low endophyte or endophyte free fescue were still greater
(Beconi et al., 1995). In the feedlot, steers ear implanted with Synovex-S® (200 mg progesterone-20 mg estradiol benzoate) had greater ADG than non-implanted steers, while also retaining more energy and 25% more protein (Rumsey, 1982). Similarly, Rumsey and Hammond (1990) reported steers implanted with estrogentic growth promoters had greater ADG and increased nitrogen retention, but also had elevated maintenance energy requirements. Rumsey et al. (1997) suggested a possible explanation for enhanced growth with estrogentic implants is through enhanced synthesis of thyroid stimulating hormone which, in turn, stimulates the synthesis of triidothyrodine (T₃) and thyroxine (T₄). This explanation assumes that enhancement in thyroid function is indeed needed for faster growth rates. Similarly, research by Kahl et al. (1978) indicated that increased plasma and free thyroxine levels and an increase in T₄:T₃ ratios in steers implanted with Synovex-S resulted in greater thyroid hormone concentrations and increased growth rates. Rumsey et al. (1992) reported similar results for shifts in the thyroid hormones with the progesterone-estradiol implant, but with no increases in protein deposition.
Chapter Three: Materials and Methods

Experimental Site

The grazing experiment was conducted at the University of Kentucky Animal Research Center (UK-ARC) in Woodford County. Protocol for the experiment was approved by the Institutional Animal Care and Use Committee at UK (00996A2006). The 104-day grazing trial was conducted from May 3 – August 15, 2006 with 62 predominately Angus steers obtained from a collaborator. Steers grazed six, 3.0-ha pastures of endophyte-infected Kentucky-31 tall fescue on Maury (fine, mixed, semiactive, mesic Typic Paleudalfs) and McAfee (fine, mixed, active, mesic Mollic Hapludalfs) silt loam soils. Pastures were stocked at a low stocking rate of 3.3 steers/ha. Pastures were fertilized in April with 56 kg N ha\(^{-1}\) using liquid nitrogen. Pastures were mowed in early May and late June at a high mowing height to clip seed heads and decrease risk of pinkeye infections (*infectious bovine keratoconjunctivitis*).

Animal Management

Water and minerals (Burkmann Mills, Danville, KY) were provided ad libitum. Cattle were treated with moxidectin dewormer (1mL/10 kg BW pour-on) (Cydectin®, Fort Dodge Animal Health, Fort Dodge, IA) for parasites and vaccinated for respiratory disease with DurGuard 6HS® (Durvet, Inc. Blue Springs, MO) at the initiation of the study. Steers with rectal temperatures above 40.6°C at any time during the study were treated with florfenicol (Nuflor® Schering-Plough Animal Health, Summitt, NJ, 40 mg/kg body weight, subcutaneous) to maintain animal health. Calves exhibiting pinkeye were treated with oxytetracycline (LA-200® Pfizer Animal Health, Exton, PA, 4.5 mL/100 lbs
body weight). Shut-Eye (American Animal Health, Inc., Wisner, NE) patches were used for those calves with severe pinkeye. On May 31 calves received a zetacypermethrin, piperonyl butoxide fly tag (PYthon MagnuM™ Y-Tex®, Cody, WY). Calves were treated with Cydectin pour-on dewormer again on July 19.

Animal Responses and Experimental Design

Cattle were received at the UK-ARC on April 18, 2006, and upon arrival were placed in a 6.0-ha endophyte-infected fescue holding pasture. A split-plot design was used for this study with clipping treatment as the main plot and implant treatment as the sub plot with three replications. Steers were stratified by body weight and hair coat color before being randomly assigned to treatments. Main plot treatments of either ten clipped or ten unclipped steers were randomly assigned to pastures. Calves assigned to the clipped treatment were clipped completely from behind the front shoulder to the rump, excluding the underline and legs. Approximately thirty to forty percent of the body surface was clipped (Figs. 1, 2). The whole body could not be clipped due to limitations with the head chute. Estimates of percentage of body surface clipped were made using an ArcMap program, ArcSketch (RockWare, Inc., Golden, CO) (Figures 1, 2). This was done by dividing the outlined area (body surface) in Fig. 1 by the outlined area in Fig. 2 and multiplying by 100. Calves were also randomly allotted to ear implant treatments of Synovex-S (200 mg progesterone, 20 mg estradiol; Fort Dodge Animal Health, Fort Dodge, IA) or Compudose (25 mg estradiol; VetLife, Inc., Des Moines, IA), such that there were five calves with each implant for each pasture. Calves received numbered ear tags with one of two colors, depending on implant assignment. Coat scores were
Figure 1. Measurement of clipped body surface.

Figure 2. Measurement of entire body surface.
recorded; rectal temperatures and unshrunk body weights were measured. Animals were assigned coat score ratings of S (sleek), R1 (less than 25% coverage of rough hair), R2 (25 to 75% coverage of rough hair), or R3 (75 to 100% coverage of rough hair) to designate the type of coat and degree of shedding.

On April 27 steers assigned to the clipped treatment were clipped. Oster ClipMaster (Boca Raton, FL) clippers with top and bottom blades of 18 and 23 teeth were used to clip the hair. All calves with orange tags received Synovex-S ear implants and calves with white ear tags were implanted with Compudose. On the first day of grazing, April 28, an area approximately $41 \text{ cm}^2$ was clipped over the front shoulder of each steer using Oster Power Pro Cordless clippers with #40 blades. Clipped hair was collected from each steer for determination of hair mass per cm$^2$. At the conclusion of grazing, hair in an area adjacent to the area previously clipped was measured for length, collected after clipping for determination of hair mass per cm$^2$, and clipped area was measured (approximately $88 \text{ cm}^2$).

Cattle were weighed unshrunk on May 3 and at 28-d intervals until the termination of grazing. On these dates, hair length was measured on the clipped areas over the shoulder using a metric ruler and the hair was re-clipped prior to collecting sweat measurements using a Delfin Vapometer (Stamford, CT). Rectal temperatures also were recorded on these dates using a TM99A digital temperature instrument (Cooper-Atkins Corporation, Middlefield, CT). On May 31, the area of hair clipped over the front shoulder was measured so hair weight per unit area could be determined. Calves initially implanted with Synovex-S were re-implanted on June 28 as activity period is only 70 days compared to 150 days with Compudose.
Grazing frequency was monitored every half hour on May 25 from 5 am to 8 pm, July 7 from 5 am to 10 pm, and July 18, July 25, August 1, August 14 from 6 am to 10 pm. Numbers of calves according to tag color were classified as either actively grazing, passively grazing or camping. Passive grazing is defined as standing in field, lying in field or walking. Camping is defined as lying near water, standing around water or mineral feeder or congregating in one area of the field. Weather conditions were recorded as sunny, partly cloudy, cloudy, rain, lightning, or sprinkling. Average number of steers and percentage of steers grazing were then calculated for each period of time throughout the day so comparisons could be made between treatments. Ambient temperatures and humidity for each day of measurement was obtained from the weather station located on the UK-ARC. A temperature-humidity index was also calculated as described by Tarazon-Herrera et al. (1999) with the following formula; THI = 0.45 T + 0.55 TH – 31.9 H + 31.9 where T = dry bulb temperature expressed in °F and H = relative humidity/100.

**Post-Graze Monitoring Phase**

A two week monitoring phase was conducted from August 15 – 30 during which steers were maintained on a concrete pad. Steers received ad libitum hay containing only a small percentage of tall fescue along with ad libitum water and minerals. Soybean hull pellets were fed at a rate of approximately 4.4 kg/steer/day. Blood samples and rectal temperatures were collected weekly following procedures previously described. Weights and sweat meter readings were taken for the first week of the monitoring phase.
Pasture Measures

Forage availability was monitored using a disk meter similar to Bransby et al. (1977) with the exception that the falling plate was ? cm in diameter and weighed ? kg. Disk meter height was recorded for 50 random locations within each pasture at 14-d intervals by taking 50 disk meter readings per pasture. Once during the grazing season when disk meter heights were collected, forage beneath the disk meter was clipped to ground level at five random locations in each pasture. These samples were dried at 60°C in a forced-air oven for 48 h and weighed. Dry matter (DM) per unit of land area was subsequently regressed on disk meter height. Tiller samples were taken on June 6 and August 9 to estimate percent endophyte infection level by tissue immunoblot and alkaloid content by HPLC. Fifty random samples of tillers from 50 individual plants were taken per pasture for infection level and fifty for ergovaline concentration. Tiller samples were collected by clipping individual tillers to ground level using Stanley™ (New Britain, CT) retractable utility knives.

Lab Analyses

Tiller samples were frozen until lab analysis could be completed. Tiller samples were assayed using a blot test for infection levels (Gwinn et al., 1991). Tiller samples collected for alkaloid content were freeze dried and then analyzed for ergovaline concentration by HPLC florescence using a modification of procedures developed by Yates and Powell (1983). Hair samples were washed with water using vacuum filtration, dried in an oven at 60°C for 48 hrs, and weighed to determine hair dry weight/unit area.
**Statistical Analyses**

All responses in the pasture and post graze phases were analyzed as repeated measures using PROC MIXED of SAS (Littell et al., 1996; SAS Inst. Inc., 2002) to evaluate effects of clipping hair coats, ear implants, date and all interactions. All data were analyzed using the first order-autoregressive covariance structure. Data were analyzed as a split-plot design using group within pasture as the experimental unit. Means separations were performed on least square means using the PDFF option in SAS for grazing frequencies and all responses, except hair growth and hair density. PROC CORR of SAS was used to determine correlation coefficients for ambient temperature and percentage of steers actively grazing at dates throughout the study and periods throughout the day. Hair density differences were analyzed using PROC TTEST to determine treatment effects on differences in hair density over the grazing season. Subsamples of animals from each group (n = 3) were used for the post graze phase; therefore, data for these measures were also analyzed using a split plot design using group within pasture as the experimental unit.
Chapter Four: Results and Discussion

Forage Data

Forage availability declined over the grazing season (P < 0.0001). However, there was ample forage available for consumption at all times as forage availability stayed above 3500 kg ha\(^{-1}\). Ergovaline measures were (0.33 ± 0.05 µg/g DM) on June 6 and increased steadily (1.24 ± 0.55 µg/g DM) by August 9. Hill et al. (1994) was able to elicit toxicosis symptoms with ergovaline concentrations as low as 0.65 µg/g DM in steers grazing toxic tall fescue for 60 days. After analyzing tiller samples taken at the beginning of the grazing experiment average pasture endophyte infection rates were 63.6% and ranged from 54 to 77.4%.

Animal Performance

ADG gradually declined over the grazing season as ambient temperature and humidity increased (Figure 3). Cumulative ADG after the first 28 days was 0.76 ± 0.07 kg and declined to 0.51 ± 0.07 kg at d 104. Average daily gain in this experiment is higher than gain (0.46 kg/d) reported by Aiken et al. (2001) in steers grazing endophyte-infected fescue and implanted with Synovex-S. This could be partially explained by the lower ergovaline concentration (1.24 vs. 2.35 µg/g DM) at the conclusion of the pasture phase in the present experiment. Likewise, pasture infection rates in this study were lower than the 75% infection rates reported in the grazing experiment by Aiken et al. (2006). Stuedemann and Hoveland (1988) reported a 0.1 lb decrease for every 10%
<table>
<thead>
<tr>
<th>Date</th>
<th>Kg/ha</th>
</tr>
</thead>
<tbody>
<tr>
<td>May 22</td>
<td>4123.33 a</td>
</tr>
<tr>
<td>June 6</td>
<td>4190.00 a</td>
</tr>
<tr>
<td>June 20</td>
<td>4025.83 ab</td>
</tr>
<tr>
<td>July 10</td>
<td>3957.17 ab</td>
</tr>
<tr>
<td>July 24</td>
<td>3676.83 c</td>
</tr>
<tr>
<td>August 9</td>
<td>3556.00 c</td>
</tr>
</tbody>
</table>

**Table 1.** Forage availabilities for the 2006 grazing season at the University of Kentucky Animal Research Center.
Figure 3. Mean cumulative average daily gain for steers grazing on toxic tall fescue in 2006 at the University of Kentucky Animal Research Center shown with standard error bars. Letters represent statistical differences (P<0.05) among dates.
increase in pasture infection rate, which supports the ADG indicated in this study. Paterson et al. (1995) reported a range in ADG of 0.21 to 0.62 kg/d for steers grazing highly infected (> 61%) tall fescue pastures. Hoveland et al. (1983) reported 0.45 kg/d ADG in cattle grazing highly endophyte-infected tall fescue.

Although ADG was unaffected by implants (P > 0.10) used in this study, Duckett and Andrae (2001) reported that Synovex-S resulted in a 12% increase in ADG while Compudose caused a 14% increase in ADG. Davenport et al. (1993) also showed Compudose implants improved ADG of steers grazing high endophyte-infected fescue (0.43 kg/d vs. 0.62 kg/d; shrunk weights) less than for nonimplanted steers grazing low endophyte-infected fescue. Aiken et al. (2001) observed a trend towards increased ADG in implanted steers over nonimplanted steers grazing endophyte-infected tall fescue. However, Aiken et al. (2006) reported a weak response of ADG to stocking rate without implantation.

Clipping did not affect ADG in this study (P > 0.10). However, several experiments have found clipping rough hair coats improved weight gain in the summer (Turner, 1962; Pathmasingham and Abdul, 1981). In these studies the entire body was clipped with the exception of the head, ears, tail and legs below the knee or hock. The differences in amount of hair clipped may explain why clipping did not affect ADG in this study. Hair coats have been found to have a significant effect on heat dissipation and heat stress (Yeates, 1955; Turner and Schleger, 1960; Turner, 1962). Cattle possessing rough, dense hair coats typical of Bos taurus breeds have lowered feed intake due to decreased grazing times and, consequently decreased growth rates as compared to cattle with short, sleek coats (Bond et al., 1984; Howard et al., 1992; Olson et al., 2003).
Grazing frequency was unaffected by treatment; however, period interacted with date (P < 0.001) in affecting percent of steers actively grazing (Table 2). At high environmental temperatures (≥ 24.5°C) and THI (≥ 74) (Figure 4) the percentage of steers actively grazing was negatively correlated (P < 0.10) with ambient temperature (Tables 3, 4). Percentage of calves actively grazing throughout the day declined to 30% when ambient temperature > 24.5°C. At lower environmental temperatures (< 24.5°C), the percentage of steers grazing averaged 44%. Results from grazing studies conducted by Parish et al. (2003) indicated cattle grazing endophyte-infected fescue spend less time grazing than cattle grazing endophyte-free fescue. In support of correlations found in the current study, Aldrich et al. (1993) reported a 22% decrease in feed intake in calves grazing at 32°C and a negative correlation between grazing times and heat index. Angus steers grazing toxic tall fescue limited themselves to morning and evening grazing with longer grazing times in the evening and results indicated loss of grazing time to standing under shade was not compensated by increased nighttime grazing (Aldrich et al., 1993). However, results from the present study did not indicate increased nighttime grazing (P > 0.05; Table 2).

Decreased intake and altered grazing behavior including shifts in grazing patterns to increased nighttime grazing in cattle consuming toxic tall fescue has been previously reported (Howard et al., 1992; Paterson et al., 1995; Schmidt and Osborn, 1993). Olson et al. (2003) reported calves with sleek coats have greater heat tolerance and had increased grazing times as a result. West (2003) reported the critical THI was 72 as compared to previous results that indicated the critical THI was 77. The threshold thermoneutral temperature at which onset of heat stress can be detected was found by
<table>
<thead>
<tr>
<th>Period</th>
<th>May 25</th>
<th>July 7</th>
<th>July 18</th>
<th>July 25</th>
<th>Aug. 1</th>
<th>Aug. 14</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early morning</td>
<td>35.4 a A</td>
<td>47.8 b AB</td>
<td>52.2 c B</td>
<td>57.6 b B</td>
<td>52.0 b B</td>
<td>63.2 c B</td>
</tr>
<tr>
<td>Late morning</td>
<td>56.4 b B</td>
<td>18.8 a A</td>
<td>27.2 b A</td>
<td>19.0 a A</td>
<td>24.4 a A</td>
<td>44.9 b B</td>
</tr>
<tr>
<td>Early afternoon</td>
<td>28.2 a B</td>
<td>55.5 bc C</td>
<td>11.9 ab AB</td>
<td>25.1 a AB</td>
<td>9.8 a A</td>
<td>26.3 a AB</td>
</tr>
<tr>
<td>Late afternoon</td>
<td>35.5 a B</td>
<td>30.0 a AB</td>
<td>10.7 a A</td>
<td>17.2 a A</td>
<td>14.0 a A</td>
<td>38.8 ab B</td>
</tr>
<tr>
<td>Early evening</td>
<td>66.2 b B</td>
<td>68.6 c B</td>
<td>45.5 c A</td>
<td>65.6 b B</td>
<td>53.7 b AB</td>
<td>51.6 bc AB</td>
</tr>
</tbody>
</table>

Table 2. Percentage of steers actively grazing toxic tall fescue over the 2006 grazing season calculated using least square means. Uppercase letters represent differences (P<0.05) within rows and lowercase letters represent differences within columns.

<table>
<thead>
<tr>
<th></th>
<th>May 25</th>
<th>July 7</th>
<th>July 18</th>
<th>July 25</th>
<th>Aug. 1</th>
<th>Aug. 14</th>
<th>Total over season</th>
</tr>
</thead>
<tbody>
<tr>
<td>Correlation</td>
<td>-0.47165</td>
<td>-0.38526</td>
<td>-0.93272</td>
<td>-0.78149</td>
<td>-0.82144</td>
<td>-0.95258</td>
<td>-0.67378</td>
</tr>
<tr>
<td>coefficient</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P &lt; F</td>
<td>0.4226</td>
<td>0.5219</td>
<td>&lt; 0.05</td>
<td>0.1185</td>
<td>&lt; 0.10</td>
<td>&lt; 0.05</td>
<td>&lt; 0.0001</td>
</tr>
</tbody>
</table>

Table 3. Correlation coefficients for percent of steers actively grazing toxic tall fescue at varying ambient temperatures throughout the day.
<table>
<thead>
<tr>
<th></th>
<th>Early morning</th>
<th>Late morning</th>
<th>Early afternoon</th>
<th>Late afternoon</th>
<th>Early evening</th>
</tr>
</thead>
<tbody>
<tr>
<td>Correlation</td>
<td>0.48400</td>
<td>-0.43326</td>
<td>-0.88717</td>
<td>-0.81645</td>
<td>-0.75917</td>
</tr>
<tr>
<td>coefficient</td>
<td>P &lt; F</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.3307</td>
<td>0.3908</td>
<td>&lt; 0.05</td>
<td>&lt; 0.05</td>
<td>&lt; 0.10</td>
</tr>
</tbody>
</table>

Table 4. Correlation coefficients for percent of steers actively grazing toxic tall fescue at varying ambient temperatures during periods of the day averaged over the 2006 grazing season.
Figure 4. Mean ambient temperature (°C) and Temperature-Humidity Index (THI) for days grazing frequencies were monitored at the University of Kentucky Animal Research Center in 2006.
Hahn et al. (1992) to be 20-25°C. Therefore, observations from grazing frequency in the current study indicated that grazing frequency declined (P < 0.10) as calves began experiencing heat stress at high ambient temperature and humidity. The decreased grazing times at high THI explains the decrease in ADG in the mid to late summer months (Figure 3). Similarly, Bond et al. (1984) reported decreased gains in cattle grazing high endophyte-infected fescue due to less time spent grazing and reduced intake.

**Physiological Measures**

A clipping x date interaction (P < 0.05) was observed for rectal temperature and clipped calves had lower rectal temperatures (Figure 5) when ambient temperatures were greater than 25° C on July 26 (Figure 6). Lack of a clipping treatment effect during the first two 28-d periods could be attributed to regrowth of the hair coat of clipped calves by the end of the 28 d period. On the last two dates, calves were brought in one week prior to collecting measurements and those assigned to the clipping treatment had their hair coats clipped again so subsequent measurements taken would be able to pick up the actual treatment effect on rectal temperature and other measures.

Similar results were found by Pathmasingham and Abdul (1981) and Turner (1962) when clipping rough hair coats of calves reduced rectal temperature, but clipping calves with sleek hair coats had no effect on rectal temperature. Research has shown that several factors, such as constricted blood flow and rough hair coats, may increase rectal temperature, and both are symptoms of fescue toxicosis (Dowling, 1959; Browning and Leite-Browning, 1997; Rhodes et al., 1991). Berman (2005) found hair coat length influences the threshold temperature at which heat stress occurs. Similarly, Olson et al.
Figure 5. Rectal temperatures for cattle grazing toxic tall fescue taken over the 2006 grazing season shown at the University of Kentucky Animal Research Center with the clipping*date interaction and standard error bars.
Figure 6. Mean ambient temperature (°C) and Temperature-Humidity Index (THI) for days physiological measures and weights were taken from cattle at the University of Kentucky Animal Research Center.
(2003) observed that cattle with winter coats and subjected to high temperatures had greater heat stress than sleek or clipped cattle.

Although no implant treatment effect occurred in this study, studies in women have found that progesterone treatment elevates body temperatures (Katovich and O’Meara, 1987; Stachenfield et al., 2000). Stachenfield et al. (2000) further reported that progesterone treatment raised body temperatures above the threshold for sweating while estrogen supplementation reduced core temperatures below the threshold. However, Samaan and Crawford (1995) did not observe differences when progesterone was added to estrogen as compared to effects of estrogen alone. Likewise, pigs in another study exhibited vasodilation as a result of progesterone infusion (Molinari et al., 2001). When compared to non-implanted steers, past research has shown that implanted steers have higher rectal temperatures while grazing toxic tall fescue (Aiken et al., 2006).

Sweat production declined (P < 0.001) over the grazing season (Figure 7). Compudose ear implants resulted in higher sweat production over Synovex-S implants (P<0.05). Sweating rate is associated with increased peripheral blood flow (Finch, 1986). Various research studies have shown fescue toxicosis results in constricted blood flow (Rhodes et al., 1991; Browning and Leite-Browning, 1997). Research in both humans and livestock has shown that estrogen induces vasodilation while progesterone can cause vasoconstriction (Ford et al., 1977; Gudmundsson et al., 1999; Chan et al., 2001; Edwards, 2005). However, in pigs, progesterone has been found to cause vasodilation or to not alter the effects of estrogen when administered together (Samaan and Crawford, 1995; Molinari et al., 2001). Under heat stress, cattle increase sweating rate and respiratory volume which allows for more heat dissipation (Al-Haidary
Figure 7. Mean sweat production for steers grazing toxic tall fescue in 2006 at the University of Kentucky Animal Research Center shown with standard error bars. Letters represent statistical differences (P<0.05) among dates.
et al., 2001). However, the results from the current study suggest that cattle suffering from fescue toxicosis do not have the ability to increase sweating rate.

Finch (1986) reported that 70-85% of heat loss in cattle is due to sweating with the remainder lost through panting. Allen (1962) found a linear increase in sweating rate with air temperature in Jersey cattle and they continued to increase sweating rates at air temperatures greater than 29.4°C. However, Pan et al. (1969) found that increased environmental temperature or heat stress does not necessarily cause linear increases in sweating rate and elevated sweating rates may not be maintained during prolonged periods of heat stress. Aldrich et al. (1993) reported that cattle grazing endophyte-infected fescue during a constant heat challenge had a 50% decrease in skin vaporization. Results from this experiment indicate that, under heat stress, fescue cattle do not have the ability to increase sweating rate to the level necessary to maintain a thermoneutral body temperature. These previous research studies indicate that the decreased sweating rate observed in cattle grazing high endophyte-infected fescue is likely due to vasoconstriction induced by the alkaloids.

Clipping the hair coat did not significantly affect sweating rate (P = 0.17) for this study. However, in other studies with cattle clipping the hair coat caused a highly significant increase in sweating rate and heat loss efficiency (Berman, 1957; Turner, 1962; Pathmasingham and Abdul, 1981).

Mean hair growth rate was 0.285 mm day\(^{-1}\) and did not differ over the grazing season (P > 0.10). The growth rate was unaffected by either the clipping or implant treatment (P > 0.10). Total hair growth over the season was 29.64 mm. Hair density on the first day of the grazing trial was 11.48 mg cm\(^{-2}\) and at the end of the trial averaged
13.73 mg cm\(^{-2}\) for an average increase (P < 0.05) of 2.8142 ± 1.0217 mg cm\(^{-2}\) over the grazing season. It is unclear whether the hair growth observed over the season was entirely due to growth of the summer coat or if the winter coat continued to grow; further experiments need to be conducted to clarify this. Since total hair growth measured in an area adjacent to where hair growth measurements were taken during the trial was similar (24.69 mm vs. 29.64 mm) it can be concluded that hair growth was not a result of clipping.

Some studies indicate the winter hair coat is denser than the summer coat (Kaszowski et al., 1970) and, in mink, growth of the summer hair coat is finished by mid-July at which point the hair follicles rest until August when fall hair shedding is initiated. Cattle in this study continued to grow hair until the end of the trial in mid-August. However, Rose et al. (1984) found mink to have a sleek, dense summer coat. Even if the winter hair coat is denser than the summer coat, it is likely that the increase in density over the course of this study was the result of continual hair growth. At initiation of grazing, all calves had rough hair coats with the majority having a coat score of R2 or R3 (>25% coverage of rough hair). Throughout the study, most steers had the appearance of having rough coats.

Ebling et al. (1991) found decreased prolactin was responsible for inhibiting growth and triggering shedding of the winter hair coat. However, Porter and Thompson (1992) found that decreased prolactin was responsible for winter hair growth while prolactin increases in the spring was responsible for shedding of the winter hair coat and growth of the summer coat. Decreased serum prolactin is a common result of fescue toxicosis (Schmidt and Osborn 1993; Strickland et al., 1993; Thompson and Stuedemann...
1993). The continual growth of hair and increase in hair density over the season suggests that prolactin was not the only control responsible for hair growth and shedding. Altered thyroid hormones could also have affected hair growth (Berman, 1960). Therefore, the likely explanation for these observations on hair coats is that steers retained most of their winter hair coat and the winter hair coat continued to grow due to decreased prolactin and other hormonal influences.

**Post-Graze Monitoring Phase**

During the two week monitoring phase following grazing animal performance and other measured responses indicated that toxicosis was alleviated. Initial rectal temperatures following termination of grazing tall fescue pastures were similar between implant and between clipping treatments (P > 0.10). Rectal temperature averaged 39.3°C upon removal from pasture and decreased to 39°C by d 15. Other studies have demonstrated alleviation of heat stress following removal from toxic tall fescue and placement on a non-toxic diet. Aiken et al. (2006) observed rectal temperatures above 40.6°C when steers were pulled off toxic fescue and declined to 39.8°C by d 10. Differences in rectal temperature between the studies were most likely due to differences in ambient temperature (Figures 6, 8; 23°C vs. 24°C) at the end of the grazing phase. Ambient temperatures at the conclusion of grazing were declining whereas with Aiken et al. (2006) the temperature was holding steady and observations were taken in late June. Data in the present study agrees with Aiken et al. (2006) as rectal temperatures coincided with declines in ambient temperatures. Cole (1993) found that ambient temperature affects rectal temperature in stressed calves. Aiken et al. (2001) found it took less than 6
Figure 8. Mean rectal temperature (°C) with standard error bars for the post-graze phase for steers after grazing toxic tall fescue at the University of Kentucky Animal Research Center. Letters represent statistical differences (P<0.05).
days for rectal temperature to be reduced to the threshold once steers were placed on a fescue free diet.

There was a large numerical increase in sweat production after one week of being off endophyte-infected pasture (28.4 g/m$^2$/hr to 32.3 g/m$^2$/hr), but this difference was not statistically significant ($P = 0.1897$). If sweat meter readings had been taken after two weeks post grazing and if all 60 animals had been included in the current study, it is more likely that sweat production differences would have been significant.
Chapter Five: Conclusion

The normal hair cycle is altered in cattle suffering from fescue toxicosis. Therefore, hair growth and shedding mechanisms could not function normally and resulted in winter hair coat retention and continuous hair growth over the grazing season. These disruptions in the normal cattle hair coat caused increased vulnerability to heat stress. Clipping the hair coat decreased rectal temperature during periods of high environmental temperature and humidity. Cattle usually deal with heat stress by increasing blood flow and sweating rate and consequently increasing heat loss to the environment. However, steers in this study experienced a dramatic decline in sweat production over the grazing season. Decreased sweating rates were most likely caused by vasoconstriction as previous research studies have shown a close relationship between blood flow and sweating rate. Various research studies have also shown that cattle consuming endophyte-infected fescue suffer from vasoconstriction.

Thermoregulatory ability was impaired at higher ambient temperatures and exacerbated by reduced sweating due to rough hair coat retention and continuous hair growth into the summer. As a result of these conditions, the steers were vulnerable to heat stress and performance of the yearling steers declined in the late summer with higher ambient temperatures. Under elevated environmental temperatures, grazing frequency was negatively correlated with ambient temperature at periods throughout the day and during days over the grazing season. This decline in grazing frequency was partially responsible for the decreased gains observed in late summer. Poor animal performance in yearling steers grazing toxic tall fescue was likely a result of rough hair coat retention,
continuous hair growth, decreased sweating rates and declines in grazing frequency during periods of high ambient temperature and humidity.
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Vita

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