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RELATIONSHIPS BETWEEN ENDOCRINE FACTORS AND RATE, EFFICIENCY AND COMPOSITION OF GAIN OF BEEF FROM FOUR BIOLOGICAL TYPES

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RELATIONSHIPS BETWEEN ENDOCRINE FACTORS AND RATE, EFFICIENCY AND COMPOSITION OF GAIN OF BEEF FROM FOUR BIOLOGICAL TYPES

By

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ABSTRACT

RELATIONSHIPS BETWEEN ENDOCRINE FACTORS AND RATE, EFFICIENCY AND COMPOSITION OF GAIN OF BEEF FROM FOUR BIOLOGICAL TYPES

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One hundred fifty nine steers from four breed groups were used in a two year study to evaluate the relationship among hormones and rate, efficiency, and composition of gain. Breed groups consisted of unselected Herefords, Herefords selected for growth, Shorthorn x Angus x Hereford and Gelbvieh x Simmental x Holstein. Cattle within a breed group were subdivided into three pens and slaughtered after 225, 247, and 260 days on feed. Ninety days prior to slaughter, blood was collected on each animal every .5 hours for an 8-hour period for hormone analysis. Routine carcass measurements were taken 24 hours post-slaughter. A 9-10-11 rib section from each animal was dissected to estimate carcass composition. Selection for growth resulted in larger framed, heavier, faster growing, leaner cattle that had significantly lower percentages of carcass fat and higher percentages of carcass protein ($P < .01$). Selection for growth did not increase growth hormone or insulin-like growth factor I concentrations. There were significant ($P < .01$) differences in concentrations of growth hormone, insulin-like growth factor I, and insulin between the breed groups. Hormone concentrations were correlated with carcass traits and carcass composition.

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TABLE OF CONTENTS

LIST OF TABLES

INTRODUCTION

In recent years, the beef industry has seen a decline in market share relative to other suppliers of protein in the American diet. Although some decline can be attributed to changing lifestyles and consumer perceptions, many of today's challenges may be resolved by animal scientists and beef producers. Results of the 1991 National Beef Quality Audit identified three major factors that contributed to the decline in market share of beef products: excessive fat, low overall uniformity, and price. These problems are the result of several factors, one of which is the diverse genetic pool of beef cattle in the United States. Although this diverse population has contributed to the current problems facing the beef industry, it also has great potential for improving its consistency and competitiveness. Although selection in the beef cattle industry has occurred since its inception, only recently have predictable genetic evaluations of breeding stock been available for wide-spread use throughout the industry.

For the beef industry to become more competitive, production of a lean product is imperative. This is further emphasized by industry movement towards value-based marketing. Production of a leaner product may be accomplished through

available genetic resources and management techniques or through new technologies.

One such technology is the administration of exogenous hormones. It is known that the complex process of growth is mediated by several factors, including environment, nutrition, and hormones. Hormones are the mediators between the environment and the biological systems involved with growth and metabolism. Although the environment places limits on growth in practical situations, the other factors previously mentioned are limited by the genetics of the animal. Little research has been conducted to study differences in hormone concentrations between cattle of known genetic variation and what relationship exists between those endocrine factors and rate, efficiency, and composition of gain.

The Lake City Experiment Station breeding project offers a unique opportunity to investigate the differences in endocrine mediators in groups of cattle with diverse genetic genomes. The relationships between live-weight gain, feed efficiency or carcass composition and the endocrine mediators may identify a new phenotypic trait to enhance selection for superior livestock. Secondly, establishment of the relationships between endocrine factors and rate, efficiency, and composition of gain will enhance our understanding of the underlying tenets of animal growth.

The results of this research project will assist the industry in production of a leaner, more consumer-appealing product. For the industry to compete with other suppliers of

protein in human diets, a lean beef product must be made available. This may be accomplished by genetic selection or by exogenous manipulation with hormones. Before either of these factors can be approached, the basic relationships between genetic selection, animal performance, carcass characteristics, and endocrine factors must be established.

LITERATURE REVIEW

Several tools are available to the beef industry to accomplish changes in cattle growth and development. The loss of market share to other protein sources in recent years has underlined the importance of utilizing these tools to make all segments of the industry more competitive and profitable. The shift toward value-based marketing by the feedlot and packer segments in response to consumer demands will feedback on cow-calf and seedstock producers.

Selection programs will become increasingly important as the beef industry attempts to produce a more consumer acceptable product. Technological development in the last twenty years has greatly enhanced the ability of producers to make fast and predictable genetic improvement. The initiation of National Sire Evaluation programs in the early 1970's led to increased selection intensity and accuracy and made expected progeny differences (EPD's) available. The shift to National Cattle Evaluation beginning in 1984 made across-herd prediction possible for all breeding animals and provided a performance link across all segments of the industry for traits of interest (Middleton and Gibb, 1991).

The importation of continental European beef breeds in the late 1960's and early 1970's has resulted in a large

genetic base of breeds and biological types from which to select. Studies have clearly indicated that there are significant differences between breeds and biological types for growth, efficiency, and carcass traits. These breed differences and different mating systems are resources available to change the growth and development of cattle to meet the needs of health-conscious consumers (Willham, 1982).

Effects of Selection

During the past 25 years, numerous studies have been conducted to quantify phenotypic response to selection in beef cattle. Hough et al. (1985) designed a study to determine the response to yearling weight selection in Hereford cattle using nationally evaluated sires. The six year study (1978-1983) utilized sires from the top 1% of the Hereford breed for yearling weight EPD. The genetic trend in yearling weight was +6.2 kg/year and resulted in an indirect increase in weaning weight of 5.0 kg/year when compared to controls. There was also an increase in yearling hip height (.75 cm/year), indicating that frame size in selected cattle increased as weight increased. There were no significant responses in post-weaning average daily gain or fat thickness, although the selected line tended to grow faster and possess more lean tissue. The authors concluded that selection for yearling weight preferentially increased lean tissue mass as compared to fat.

Australian workers studied the effects of long-term,

single-trait selection for yearling weight in Angus cattle. Closed-line selection (utilizing sires produced within the herd) was based on weight gain from birth to one year of age. Three lines were utilized: high yearling weight, low yearling weight, and control. Responses in yearling weight were +15% and -14% for the high and low lines compared to controls, respectively. Similar trends were reported for weaning weight. At a constant age endpoint, there were no significant differences in feed efficiency or carcass traits, except the high yearling weight line of cattle were heavier. At a constant weight endpoint, selection for heavier yearling weights resulted in 10-15% less feed consumption, 2 mm less backfat and required 20 days less time on feed to reach a specified body weight than controls (Parnell, 1992).

Newman et al. (1973) summarized ten years of selection for yearling weight in two replicate Shorthorn herds. Yearling weight increased by 4.8 and 4.1 kg/year in males and 3.3 and 2.3 kg/year in females due to selection in the two herds. Furthermore, in a Nebraska study, progeny sired by Angus and Hereford bulls born in the early 1980's had 13 to 15% heavier weaning weights than those born in the late 1960's (Cundiff et al., 1991). Since many traits are positively correlated, single-trait selection may result in increases in other traits. For example, Koch et al. (1974) used three lines of Hereford cattle and practiced selection for weaning weight, yearling weight, or an index of yearling weight and muscle thickness. Growth traits increased similarly in all

three selected lines.

Andersen et al. (1974) investigated the response of intensive selection for yearling weight on growth and carcass characteristics. Cumulative selection responses of 41.5 and 46.2 kg in yearling weight over the five year study were observed. Associated with this response were increases in weaning weight and daily weight gain from birth to 10 months of age. The indirect effects on carcass merit were a higher percentage of bone and a trend for a decreased amount of weight and age adjusted fat thickness. Koch (1978) found changes in composition associated with selection for growth rate and muscling score to be in a positive direction. Phenotypic trends indicated that at a constant weight endpoint, percentage of retail product increased while trimmable fat decreased as rate of weight gain increased. The author suggested that dual-trait selection for weaning or yearling weight combined with measures of fatness or muscling would lead to increased carcass weight at a given age and a higher proportion of edible product (Koch, 1978).

Ohio workers (Bishop et al., 1991) examined response to selection for post-weaning feed conversion and correlated effects on post-weaning growth and carcass traits. No differences were found between high and low feed conversion progeny for feed intake although the high feed conversion (lower feed/gain) progeny gained significantly faster during the post-weaning test. Consequently, feed conversion efficiency was increased slightly. Progeny from the more

efficient feed conversion group had greater subcutaneous fat at slaughter, indicating the advantage in average daily gain resulted in accretion of more fat rather than lean.

Differences Among Breeds and Biological Types

Identification of a breed or biological type that is optimum for specific nutritional or management systems has been studied for decades. With the introduction of continental European breeds, crossbreeding has become an accepted method to utilize heterosis to match breed characteristics to market specifications and environment. Due to the large number and diversity of breeds contributing to the available genetic pool, vast differences in performance and carcass traits exist both between and within breeds and biological types.

Advantages in rate of gain and feed efficiency common to large, late maturing breeds have been well documented (Byers and Rompala, 1979; Crouse et al., 1985; Schmidt et al., 1987). Thonney et al. (1981) found larger framed Holstein steers consumed more dry matter, required one unit less feed per unit of gain, and grew .2 kg/day faster than small-framed Angus steers when compared at similar weights. However, within a breed type, only 2 to 19% of the variation in daily dry matter intake was explained by weight. The authors concluded that among cattle with a similar mature size, increasing weight has a dramatic effect on growth rate and feed efficiency as both variables decrease with increasing weight and maturity.

In most studies, the variations in growth rate, feed efficiency, and carcass composition between various cattle types have been compared either at a constant weight or after a constant time on feed. Smith et al. (1976) reported Simmental and Charolais sired steers grew faster than Hereford and Angus crossbred steers. Faster growing cattle were more efficient on an age and weight constant basis. Evaluation of feed efficiency over an age constant interval gives an advantage to breed groups that gain rapidly relative to weight being maintained whereas feed efficiency measured over weight constant intervals is increased by rapid growth rate because fewer days of maintenance are required. Additionally, the authors suggest that weight constant evaluation of efficiency favors breeds that are less mature because of their leaner composition of gain (Smith et al., 1976).

One would expect larger, later maturing cattle to have an advantage as they would be younger physiologically; thus, would be depositing a lower proportion of fat in gain (Ferrell and Crouse, 1978). In an effort to address this problem, Ferrell and Crouse (1978) compared growth rate, feed efficiency and carcass characteristics of various types of steers at a constant carcass fat end-point. Larger framed Gelbvieh and Chianina steers had higher average daily gains than Red Poll steers. Gelbvieh crossbred steers consumed more dry matter which was not attributable to a difference in metabolic body size. The authors suggested a difference in net efficiency of energy utilization for maintenance of gain

due to steer type, with Gelbvieh sired steers being the least efficient.

The effects of larger framed, faster growing breeds can be complemented by crossbreeding. Long (1980) has summarized several studies utilizing straightbred and crossbred breed groups to estimate breed effects and heterosis across varying sexes and management systems. Post-weaning daily gains had an average heterosis effect of 6% in Shorthorn, Angus, and Hereford cattle. Similarly, yearling weight exhibited an average heterosis effect of 4%. The importance of sire breed within a crossbreeding system was made evident by Marshall et al. (1990) who found post-weaning average daily gain to decline for generations within rotations for which Hereford was the sire breed.

Reported effects of breed or biological type on carcass characteristics are variable because many are confounded with slaughter endpoints and feeding systems. Dikeman et al. (1985) reported larger, faster growing Simmental-Charolais steers were heavier, leaner, and more muscular with less marbling than conventional Hereford-Angus steers. Similarly, Marshall et al. (1990) found Simmental-Hereford calves produced heavier carcasses with less backfat, larger ribeyes, and a higher cutability than Angus-Hereford steers. However, the Angus-Hereford steers excelled in marbling and quality grade. Similar data have been reported by Crouse et al. (1985).

Martin et al. (1980) found that at the same amount of

marbling, carcasses from Simmental and Chianina-sired crossbreds were much leaner than carcasses from Angus sired steers. However, these effects were confounded with heavier carcass weights. In support of this observation, Smith et al. (1976) reported Charolais and Simmental sired steers have heavier weights and required more days on feed to reach 5% longissimus fat than Angus, Hereford, or Angus-Hereford steers. At a constant percentage fat in carcass soft tissues; larger framed Chianina and Gelbvieh cattle have heavier carcass weights, larger ribeye areas, and less internal fat with a correspondingly lower yield grade than Red Poll cattle. Conversely, the larger breeds had less marbling and more external fat. Simmental steers have been reported to have increased weight, higher percent lean and less fat trim in the hindquarter and flank compared to Polled Hereford steers. Polled Hereford steers had increased flank weights which contain a large fat component thereby making the flank more reflective of total fat rather than lean (Arnold et al., 1990).

Koch et al. (1976) found a positive association between growth rate of breed groups and percentage of retail product or bone. A negative association was observed between growth rate of breed groups and percentage of fat trim. Because of this negative association, breed groups attaining the same percentage of fat in the longissimus may have significantly different carcass weights. Crouse et al. (1985) suggested that increasing the rate of fattening through breed selection

reduces carcass weights and muscling.

Endocrine Influence on Growth

The complex process of growth includes increased cell number, size and the deposition of substances within these cells. Many factors are involved in these processes including hormones, diet, environment, age, and sex. The hyperplasia and hypertrophy of skeletal muscle, adipose tissue, and bone are of primary concern in meat-producing animals. The homeostatic and homeorhetic control of these tissues is regulated by hormones and hormone receptors. The process of tissue growth and metabolism may not be attributed to a single endocrine influence as one hormone may have multiple actions and multiple hormones may perform one function. The relationships between hormones and their receptors regulate growth and nutrient deposition within tissues.

Although the endocrine system regulates short and long term growth, it is the genetic ability of the animal that sets the upper limits to animal growth. The maximum growth potential of meat animals is not clear, nor are the rate limiting steps which cause individual animals to gain at varied rates, utilize nutrients more efficiently, or partition nutrients into specific tissues. It is not clear if genetic selection for growth and efficiency has altered the endocrine status of meat animals. Current research in animal growth includes the use of exogenous hormones to alter growth rate

and composition. Insight as to influences of physiological hormone concentrations on growth may result from the administration of these substances. Future research will also clarify tissue sensitivity, receptors, and clearance rates of hormones.

Growth Hormone

Growth hormone (GH; somatotropin) is a peptide hormone which is stored and secreted from the somatotropic cells of the anterior pituitary. In humans, several forms of GH differing in molecular weight are secreted (Lewis et al., 1978). These epitopes vary in immunoreactivity. Differing forms of GH have not been confirmed in the bovine species. However, results from research in primates suggest the possibility that different epitopes are produced and may have different biological activities (Baumann et al., 1985).

Control of GH secretion from the anterior pituitary is controlled primarily by two hypothalamic hormones (Martin and Millard, 1986; Buonomo and Baile, 1990; Frohman, 1991), growth hormone releasing factor (GRF; also called growth hormone releasing hormone, GHRH) and somatostatin (somatotroph release-inhibiting factor, SRIF). Growth hormone secretion in the ruminant is pulsatile and variable among animals (McAtee and Trenkle, 1971a; Breier et al., 1986; Anderson, 1987; Laurentie et al., 1989). Fluctuations in GRF and somatostatin are thought to cause GH pulses (Davis, 1988). In male rats, GH is secreted in peaks with higher amplitude and baseline

values than females. Similarly, bulls have higher peak amplitudes than steers (Afinson et al., 1975) and young bulls have higher GH concentrations than heifers (Keller et al., 1979). Neonatally secreted androgens imprint the high amplitude pulses in males and sexually dimorphic patterns in GH secretion may explain growth rate and body size differences between males and females (Gluckman et al., 1987).

Control of GH secretion also involves negative feedback. Growth hormone and insulin-like growth factor-I (IGF-I; somatomedin-C, SM-C) stimulate somatostatin release from the hypothalamus (Berelowitz et al., 1981), thereby inhibiting pituitary release of GH. Somatostatin inhibits GH response to GRF. Berelowitz et al. (1981) reported that IGF-I participates in the negative feedback loop with an immediate effect on hypothalamic somatostatin and a delayed effect on the anterior pituitary.

Nutritional status plays a role in determining the circulating GH concentration in cattle. Growth hormone concentrations are elevated during nutritional deficit in sheep and cattle (Ellenberger et al., 1989). Breier et al. (1986) observed a three-fold increase in GH pulse amplitude of Angus steers fed 1% versus 3% of live weight on a dry matter basis. There was no change in GH pulse frequency or baseline concentration. Fasting increases the half-life, and reduces the turnover and metabolic clearance rates of GH in calves (Trenkle, 1976). In lactating dairy cows, energy balance is negatively associated with concentrations of GH (Villa-Godoy,

1987). It is postulated that increased levels of GH at lower planes of nutrition are an adaptation to mobilize energy from adipose tissue to maintain basal metabolism (Bauman et al., 1982; Gluckman et al., 1987). However, under optimum planes of nutrition fed for maximum growth, there is little evidence to suggest that GH concentrations are significantly affected by nutritional status.

A decline in circulating GH with advancing age has been observed by several workers (Stern et al., 1971; Trenkle, 1971; Trenkle and Topel, 1978; Keller et al., 1979; Anderson, 1987). Early investigators attributed the decline in growth rate from birth to market weight to lowered serum GH concentrations (Baird et al., 1952; Baker et al., 1956). Purchas et al. (1970) reported a decrease in pituitary GH content and a decline in the ratio of pituitary weight to body weight with increasing age. Declines in rate of gain exhibited by cattle have coincided with decreases in circulating GH and a dilution of GH concentration on a per unit of body weight basis (Trenkle and Topel, 1978; Anderson, 1987).

Although the primary effects of GH on bone growth are mediated by the somatomedins (Spencer, 1985), it has been demonstrated that GH can directly stimulate bone growth. Isaksson et al. (1982) demonstrated a direct effect of GH on epiphyseal cartilage growth by injecting human GH locally into the growth plate of hypophysectomized rats. Width of the cartilage growth plate was increased after 4 days of GH

treatment in a similar study (Isaksson et al., as cited in Isaksson et al., 1986). Growth hormone binds to receptors on chondrocytes isolated from rabbit ear cartilage (Eden et al., 1983) and stimulates DNA synthesis in the same tissue (Madsen et al., 1983). Isaksson et al. (1986) suggests that GH directly stimulates chondrocyte differentiation in the growth plate. Local growth factors (IGF-I), produced in the growth plate, are responsible for subsequent clonal expansion. The finding that GH specifically binds to cells in the proximal part of the growth plate (Isaksson et al., 1986) would support this "dual effector" theory.

Growth hormone does not appear to have direct effects on growth of muscle cells in culture (Florini, 1985). Growth hormone had little effect on proliferation or amino acid uptake of rat myoblasts or myotubes in vitro (Florini et al., 1977; Ewton and Florini, 1980). Allen et al. (1983) found no effect of GH on actin synthesis in cultured satellite cell myotubes. Similarly, exogenous GH at physiological concentrations showed no effect on rat muscle satellite cell proliferation in vivo (Beermann et al., 1983). Harper et al. (1987) reported no effect of GH on protein synthesis and degradation in cultured ovine muscle cells.

In contrast to studies on individual muscle cells, GH has been found to be anabolic in isolated muscles. Growth hormone stimulates amino acid incorporation in diaphragm muscle from hypophysectomized (Kostyo and Engel, 1960; Kostyo and Schmidt, 1961) and normal (Albertsson-Wikland et al., 1980) rats.

Increased activity of the ribosomes was also found in the same tissues (Kostyo and Rillema, 1971). The effect of GH on proliferation of non-muscle cells may explain the results found when GH is added to isolated muscles as compared to cells in culture (Florini, 1985). It has been suggested that the actions of GH on skeletal muscle are mediated by IGF-I (Florini, 1985; Davis, 1988).

The effects of GH on adipose tissue metabolism are thought to be diabetogenic and lypolytic. Eisemann et al. (1986) showed fatty acid (FA) turnover rates are increased in dairy and beef cattle treated with highly purified bovine GH, coupled with an irreversible loss of FA from the plasma pool. The authors attributed the results to an enhanced release of FA from adipose tissue (lipolysis). In vitro studies with ruminant adipose tissue have not shown conclusive evidence that GH is lipolytic (Duquette et al., 1984; Etherton and Walton, 1986). Positive lipolytic responses to exogenous GH in vivo but not in vitro may suggest that the GH molecule needs to undergo in vivo modification or may activate a lipolytic intermediate (Hart, 1984a; Hart et al., 1984b; Etherton and Walton, 1986; Gluckman, 1987). In support of this theory, Hart et al. (1984b) found that recombinant GH increased FA concentrations in vivo but did not stimulate glycerol release in vitro.

In hypophysectomized rats, glucose transport occurs at maximum rate and cannot be stimulated by insulin. Administration of GH to the same rats decreased glucose

transport and increased sensitivity to insulin (Schoenle et al., 1979). The ability of GH to alter tissue response to insulin has also been demonstrated in bovine adipose tissue (Etherton et al., 1987). No effects of GH on insulin-stimulated lipogenesis were observed with short-term incubations of swine adipose tissue (Etherton and Walton, 1986). However, chronic exposure of the tissue to physiological concentrations of GH showed a strong antagonisitic effect of insulin action on lipogenesis, suggesting that GH is acting to divert nutrients away from lipid synthesis.

Adipocyte differentiation has been shown to be affected by GH in vitro. Nixon and Green (1984) and Green et al. (1985) showed that GH stimulates the differentiation of 3T3 preadipose cells to adipocytes, and that IGF-I was not involved in differentiation. The "dual effector" theory states that GH directly stimulates cells to differentiate, and IGF-I acts on the differentiated cells to promote clonal expansion (Green et al., 1985). These results are in conflict with in vivo data, as increases in cell number would lead to an increase in lipid accretion (Boyd and Bauman, 1989).

Several workers have attempted to relate GH status of animals to growth rate. Larger breeds of beef cattle have higher mean GH serum concentrations than smaller breeds (Ohlson et al., 1981; Verde and Trenkle, 1982; Grigsby and Trenkle, 1986). Grigsby and Trenkle (1986) found Simmental steers to have higher GH concentrations, less frequent release

of GH, and secretory spikes of greater magnitude than Angus steers. Higher GH concentrations have been reported in rams selected for increased rate and efficiency of gain (Dodson et al., 1983) and in Hereford bulls selected for heavier body weight and muscling (Davis et al., 1983). Contrastingly, elevated GH concentrations have been reported in slow growth strains of chickens (Goodard et al., 1988), dwarf chickens (Hoshino et al., 1982), and swine (Norton et al., 1989). Dev and Lasley (1969) reported that dwarf Hereford cattle possessed a normal amount of GH. Purchas et al. (1970), Trenkle (1970), Irvin and Trenkle (1971), Keller et al. (1979) and Klindt et al. (1985) all found GH was not related to measurements of growth rate in ruminants while Hafs et al. (1971), Purchas et al. (1971), Trenkle and Topel (1978), Wheaton et al. (1986), and Verde and Trenkle (1987) obtained negative correlations. The contradictory reports of the correlation between GH and growth in the literature suggests other molecules or levels of regulatory control are involved.

Growth hormone has been found to be negatively related to carcass fatness (Purchas et al., 1970; Trenkle, 1970; Purchas et al., 1971; Trenkle and Topel, 1978; Keller et al., 1979; Klindt et al., 1985). Wangsness et al. (1977) reported lower GH levels in obese versus lean pigs. Trenkle and Topel (1978) found positive correlations between percent carcass muscle and GH status. Eversole et al. (1981) reported both average daily protein and fat gain to be negatively related to serum GH.

The complexity of factors involved in the development of

the various tissues involved in body growth and the possibility that many of the actions of GH are mediated by IGF-I, does not make it surprising to find inconsistent relationships between GH, growth and carcass traits. Measurement of circulating concentrations of GH does not provide insight into other factors involved in growth such as hormone receptors, tissue refractoriness or other steps involved in the secretion and metabolism of the hormone. Infrequent sampling technique to accurately assess GH status was also a problem in many early studies. Further research is needed to define the biological significance and the parameters involved in the episodic secretion of GH. Thus, correlations between endogenous GH secretion and growth or carcass composition as a selection tool in the animal industry have yet to be demonstrated.

Insulin-Like Growth Factor I

The insulin-like growth factors (IGF; somatomedins) are a family of circulating polypeptides derived from several tissues. The early study of Salmon and Daughaday (1957) described a factor in normal serum that stimulated the incorporation of labeled sulfate into cartilage explants. Serum from hypophysectomized rats failed to stimulate sulfate incorporation. However, serum from hypophysectomized rats treated with GH stimulated sulfate uptake. Direct addition of GH to the explant media failed to stimulate sulfate incorporation either in the presence or absence of

hypophysectomized rat serum. The "sulfation factor" found in serum that mediated the growth promoting actions of GH was later termed somatomedin (Daughaday et al., 1972).

Somatomedins are one of a variety of growth promoting factors found in serum that originate from different sources. Somatomedin-C is homologous to IGF-I and has structural similarity to proinsulin. Somatomedin-A and IGF-II are the same peptide. Insulin-like growth factor I is a basic, 70 amino acid single chain peptide and IGF-II is a neutral peptide consisting of 67 amino acids (Gluckman et al., 1987). Insulin-like growth factor II is primarily involved in fetal growth, while IGF-I is associated with postnatal growth and development.

Insulin-like growth factors are bound to large molecular weight proteins in blood (Spencer, 1987). Half-life of IGF is increased when bound to the transport protein. Transport proteins render IGF inactive, preventing insulin-like effects. Activity is restored upon release from the transport protein. The transport protein provides short term storage and transports IGF to target tissues (Spencer, 1987).

Liver is the major source of circulating IGF (D'Ercole et al., 1984). Schwander et al. (1983) showed that IGF is produced and secreted by the perfused rat liver. Many other tissues also synthesize IGF (D'Ercole et al., 1984), suggesting that IGF may exert its biological influence in an autocrine, paracrine, or endocrine manner. However, it has been estimated that over 90% of the total IGF is secreted by

the liver (Froesch et al., 1986).

Concentrations of IGF in serum are related to GH. Concentrations are lower in hypopituitary states and elevated in GH excess (Clemmons et al., 1987; Gluckman et al., 1987). Administration of GH to humans resulted in an increase in plasma IGF-I concentration (Copeland et al., 1980 cited in Clemmons et al., 1987). Underwood et al. (1982) reported a 5-fold increase in IGF concentration in intact compared to hypophysectomized ewes.

In chickens (Hoshino et al., 1982) and sheep (Roberts et al., 1990), IGF concentrations are greater in males than females. Bishop et al. (1989) showed a similar trend in beef cattle. Insulin-like growth factor I concentrations rise after birth and then remain constant from 6 to 18 weeks of age in rams (Olsen et al., 1981). Lund-Larsen et al. (1977) reported an increase in IGF-I concentration from 6 to 10 months of age in Red Danish bulls. Hoshino et al. (1982) showed a decline in IGF-I concentrations over time in chickens. Limited data are available on the effects of time or age on IGF-I concentrations; however, a decline in GH over time should result in a corresponding decrease in IGF-I. Indeed, Davis and Bishop (1991) reported circulating IGF-I concentrations to decline with age in heifers; and Hammond et al. (1990) reported a negative correlation between IGF-I concentration and days on feed.

Nutritional status plays a dominant role in regulating IGF-I concentrations. Gluckman et al. (1987) showed a 50

percent decrease in plasma IGF-I in Angus steers fed below maintenance compared with steers at higher intakes. Upon realimentation, IGF-I concentrations returned to normal (Ellenberger et al., 1989). Similarly, low energy diets have been associated with reduced IGF-I concentrations in steers (Elsasser et al., 1987; Houseknecht et al., 1988; Ellenberger et al., 1989; Elsasser et al., 1989; Hammond et al., 1990). Elsasser et al. (1989) reported lower IGF-I concentrations in a state of low or negative nitrogen balance and diminished response of IGF-I to exogenous GH. Concentrations of IGF-I increased with added protein in isocaloric diets. The authors speculate that protein may be the primary nutritional determinant of basal IGF-I in cattle and that undernutrition can uncouple the regulation of IGF-I by GH (Elsasser et al., 1989). Similar trends have been reported in humans (Clemmons et al., 1987).

Insulin-like growth factors have been identified in all tissues; including adipocytes, skeletal muscle and cartilage (Gluckman et al., 1987). The stimulatory effects of IGF-I on cartilage growth was first demonstrated by Simon and Daughaday (1957). More recent evidence suggests the growth promoting effects of GH may be attributed to IGF-I (Schoenle et al., 1982). These workers infused IGF-I into hypophysectomized rats and showed that tibial cartilage growth was restored to rates similar to GH treatment. In an effort to demonstrate paracrine function of locally produced IGF-I, Schlecter et al. (1986, cited in Davis, 1988) demonstrated inhibited tibia

growth in rats infused with anti-IGF-I antibody. In similar studies, exogenous IGF-I administration to hypophysectomized and normal rats has resulted in increases in tibial width, but not to the same degree as with GH treatment (Guler et al., 1986; Hizuka et al., 1986 cited in Clemmons et al., 1987).

The primary functions associated with IGF are stimulation of mitosis in cell culture, stimulation of growth in hypophysectomized animals and insulin-like effects (Gluckman et al., 1987). Rate of growth in both normal and hypophysectomized rats has been shown to increase with IGF-I administration (Froesch et al., 1986; Davis, 1988). Insulin-like growth factor I is active in stimulating anabolic processes in muscle (Florini,1985). Insulin-like growth factor I has been shown to stimulate proliferation, amino acid uptake and differentiation in cultured myogenic cells (Ewton et al., 1987). Harper et al. (1987) demonstrated the ability of IGF-I to stimulate muscle protein synthesis and decrease protein degradation in ovine myotubes. Dodson et al. (1987) reported IGF-I increased proliferation of satellite cells. However, Greene and Allen (1991) found IGF-I to have no effect on proliferation but rather to stimulate differentiation of bovine satellite cells in vitro.

The effects of IGF-I on adipose tissue are less clear than with muscle and bone. Insulin-like growth factor I elicits classical insulin-like effects on the target tissues of insulin. Increased glucose metabolism and lipid synthesis (Froesch et al., 1986); and decreased lipolysis in adipose

tissue (Gluckman et al., 1987) are associated with higher concentrations of IGF-I. Compared with adipose tissue, the rat heart muscle is 20 times more sensitive to IGF than adipose tissue. It is likely that IGF affects glucose metabolism in muscle through an IGF receptor (Froesch et al., 1986), whereas it has been postulated that IGF exerts insulin-like function in adipose tissue through the insulin receptor (Gluckman et al., 1987). Incorporation of labeled glucose into diaphragm muscle is stimulated at IGF concentrations lower than those necessary to produce insulin-like effects on adipose tissue. From these results, Froesch et al. (1986) have suggested IGF infusion would lead to an insulin-like effect on muscle before lipolysis is inhibited and glucose metabolism of adipose tissue is stimulated. Further studies combining in vivo and in vitro approaches are necessary to understand how IGF-I affects adipose tissue.

Correlations between IGF-I concentration and animal performance have been variable. Eigenmann et al. (1984) studied IGF-I concentrations in lines of Poodles bred for different mature body sizes. Larger breeds of Poodles have significantly higher concentrations of IGF-I, whereas normal growth hormone concentrations were found in all groups. Selection for high lean tissue in mice resulted in increased body weight and higher basal IGF-I concentrations. Selection for fatness had no effect on IGF-I status in the same study (McKnight and Goddard, 1989). Similarly, Blair et al. (1988) reported increases in 6-week and mature body weights after 7

generations of selection for elevated IGF-I in mice. In cattle, Davis et al. (1992) reported a low IGF-I selection line tended to have higher weaning weights, daily gains, and yearling weights than the high IGF-I line. Limited data are available in cattle using IGF-I concentrations as a selection tool.

Lund-Larsen et al. (1977) found circulating IGF-I to be positively related with rate of gain and growth, and negatively related to feed conversion efficiency in Red Danish bulls. Insulin-like growth factor I concentrations were also found to be positively correlated with body weight and hip height in sets of identical twin heifers (Davis and Bishop, 1991). Goddard et al. (1988) reported IGF-I was not related to growth rate between lines of chickens, although higher IGF-I was positively correlated with an increase in body weight.

Olsen et al. (1981) measured IGF-I concentrations in Dorset lambs from 2 to 18 weeks of age. Insulin-like growth factor I was positively correlated with relative weight gain (gain as a percentage of body weight) but not absolute body weight gain over the period. Faster growing Suffolk sired lambs were found to have higher IGF-I than Finnsheep by the same workers (Wangsness et al., 1981). Hammond et al. (1990) found IGF-I concentration to be positively related to estimated percentage of Brahman breeding and inversely related to estimated percentage of English breeding. However, the specific design of the study was to evaluate the effects of nutritional levels on IGF-I concentration and not breed of

cattle.

These same workers found IGF-I to be significantly correlated with empty body weight (r= -.60), empty body water (r= -.59) and empty body protein (r= -.60). Davis et al. (1992) reported a positive relationship between ribeye area, carcass weight, marbling, and quality grade with IGF-I. In the same study, IGF-I was positively related to backfat, percentage kidney, pelvic and heart fat, and yield grade; however, the authors attribute these findings due to a corresponding increase in carcass weight. In contrast, Anderson (1987) reported negative correlations ($P < .05$) between IGF-I concentrations and percentage carcass fat (r= -.60), carcass fat accretion rate (r= -.57), total carcass fat (r= -.52), fat thickness (r= -.73) and percentage carcass protein (r= .60).

Insulin

Insulin is a peptide hormone secreted from the beta cells of the pancreatic Islets of Langerhans. In coordination with other anabolic and catabolic hormones, insulin controls partitioning of available nutrients during growth. Insulin has pronounced effects on carbohydrate and protein metabolism by regulating entry of glucose and amino acids into tissues. Due to differences in metabolism between ruminant and nonruminant species, insulin may exert dissimilar functions in different species. As a result of microbial fermentation in the rumen, ruminants utilize acetate instead of glucose as a

major substrate for energy storage and oxidation and are almost totally dependent on gluconeogenic pathways for the supply of needed glucose in both the fed and fasted state (Prior and Smith, 1982).

McAtee and Trenkle (1971b) found a biphasic secretory pattern of insulin after a meal in growing cattle. There is a rapid increase of circulating insulin followed by a second rise of insulin which lasts between 2 and 6 hours, coinciding with absorption of the products of digestion and peripheral tissue anabolism (Weekes, 1986). Because carbohydrates are fermented in the rumen, concentration of insulin in the blood is not correlated with blood glucose (Trenkle, 1981).

Products of digestion that induce release of insulin from the pancreas are not clearly defined in the ruminant. Intravenous injection of propionate or butyrate stimulate release of insulin. Amino acid infusion also causes a release of insulin (McAtee and Trenkle, 1971b). However, the authors point out that there is not a marked increase in concentrations of propionate, butyrate, or free amino acids in the blood of ruminants after feeding. Contrastingly, Stern et al. (1971) found intravenous glucose administration to elevate insulin concentrations in suckling, weanling, and mature ruminants.

Heifers fasted for intervals of two to eight days had lower concentrations of circulating insulin than during the fed state (McAtee and Trenkle, 1971b). An increased proportion of concentrate in diets enhanced the magnitude of

post-feeding rise of insulin in sheep (Weekes, 1986) and cattle (Trenkle, 1970). Growing lambs fed a fixed amount of feed per unit of metabolic weight had an increased secretion of insulin as age and body weight increased (Weekes, 1986). Trenkle (1970), Trenkle and Topel (1978), and Verde and Trenkle (1987) reported insulin concentrations were lowest in young cattle and gradually increased with age and weight. The increased insulin response to feeding with age may be associated with the increase in deposition of body fat (Weekes, 1986).

Insulin is generally thought to stimulate lipogenesis (Prior and Smith, 1982). The effects of insulin on fat metabolism in the man and rat are well established. Insulin increases adipocyte uptake of fatty acids by stimulation of lipoprotein lipase activity. Lipogenesis is stimulated by increased glucose uptake and increased activities of pyruvate dehydrogenase, acetyl-CoA carboxylase and fatty acid synthesis (Weekes, 1986). Insulin is also thought to decrease the mobilization of stored triglyceride (Martin et al., 1984; Weekes, 1986).

Insulin receptors have been found on adipocytes from cattle (Vernon et al., 1985). Incubation with physiological concentrations of insulin for 24 hours stimulated glucose and acetate utilization by sheep adipose tissue (Vernon et al., 1985). Yang and Baldwin (1973) found a combination of insulin and glucose increased acetate utilization by isolated bovine adipocytes. Insulin treatment of diabetic steers

significantly decreased plasma glucose, lactate, free fatty acid and triglyceride concentrations. Further results from these studies suggested that insulin was necessary to reestablish rates of acetate and lactate incorporation into fatty acids in adipose tissue in vitro (Prior and Smith, 1982). Prior and Smith (1982) have suggested that the primary effects of insulin on ruminant adipose tissue are to increase the uptake of glucose and to stimulate lipoprotein lipase with an overall effect of increasing triglyceride deposition.

Insulin is thought to be one of the major regulators of muscle protein metabolism (Etherton, 1982). Cattle hind-limb studies have been used to study the effect of insulin to increase uptake of amino acids. The work of Brockman et al. (1975) showed insulin had no effect on hepatic removal of amino acids, suggesting skeletal muscle would account for a major portion of these effects. Indeed, Prior and Smith (1983) reported that insulin treatment of diabetic steers reversed an increase in plasma amino acid concentrations. Similar results have been obtained in sheep (Prior and Smith, 1982). Airhart et al. (as cited in Florini, 1985) demonstrated stimulation of protein synthesis in chick myoblasts with physiological concentrations of insulin. Muscle cell DNA, RNA, and protein synthesis are decreased in insulin deficient rats and these effects are reversed by insulin administration (Martin et al., 1984).

Florini (1985) suggested that insulin plays an essential role in maintaining cells in a viable condition, thus allowing

cells to grow rather than a direct stimulatory effect. The basis for this theory originates from the crossreactivity of insulin and IGF-I receptors. The IGF type 1 receptor binds IGF-I and has a weak crossreactivity with insulin. Both IGF-I and IGF-II have a weak affinity to the insulin receptor (Gluckman et al., 1987). The close homology of the IGF type 1 and insulin receptors and the corresponding crossreactivity of the two hormones may explain the anabolic effects of insulin on muscle when added at high concentrations (Florini, 1985). The mode of action of IGF and insulin in any tissue may depend on the distribution of insulin and IGF receptors in that tissue (Gluckman et al., 1987). Direct action of insulin on cell growth remains inconclusive and further research is needed to define the effects of insulin in the ruminant and its synergism with other hormones controlling tissue metabolism. Etherton and Kensinger (1984) propose that measurements of insulin receptor sensitivity, secretion and metabolic clearance rate may provide a better understanding of the physiological role of insulin on growth.

The importance of insulin in the regulation of growth is made apparent by the effects of diabetes. Romsos et al. (1971) was able to reverse chronic tissue wasting and weight loss in diabetic pigs with insulin administration. However, circulating insulin concentrations appear to be unrelated to growth rate (Irvin and Trenkle, 1971; Trenkle and Topel, 1978; Etherton, 1982). Wangsness et al. (1977) reported a line of pigs selected for slow growth and obesity had higher insulin

concentrations than the faster growing, lean control line. Contrastingly, Norton et al. (1989) found elevated insulin concentrations in gilts selected for rapid versus slow growth. Iowa workers have also reported conflicting evidence with respect to cattle breed and insulin concentration. Irvin and Trenkle (1971) originally reported no differences in circulating concentrations among breeds. Grigsby and Trenkle (1986) found earlier maturing Angus steers have significantly higher insulin concentrations than Simmental steers. In a later study, large frame steers had higher blood insulin concentrations compared to medium or small frame steers (Verde and Trenkle, 1987). The authors attribute the latter finding to an increased level of feed intake in the large frame steers. Similarly, it has been suggested that a positive relationship between growth rate and insulin could not be demonstrated due to the variation in insulin concentration throughout the day in response to feeding (Etherton and Kensinger, 1984). However, Eversole et al. (1981) reported insulin concentration to be positively related to average daily gain.

Despite the inability of workers to relate insulin with growth, insulin has been shown to be strongly correlated with carcass fatness. In growing cattle, Trenkle and Topel (1978) found that circulating insulin concentrations were positively correlated with percentage of carcass fat and negatively related with carcass muscle. These correlations are opposite those reported for GH (Purchas et al., 1970; Keller et al.,

1979; Klindt et al., 1985). Elevated GH and low insulin concentrations in larger, leaner breeds of cattle favor increased and more prolonged growth of skeletal muscle rather than shifting energy to adipose tissue. Smaller breeds of cattle have more insulin and is associated with increased fat deposition at an earlier age (Trenkle, 1981). Although this hypothesis has yet to be confirmed, it would support the theory that a number of hormones and their interactions are involved in the complex process of growth and ultimately carcass composition.

Thyroid Hormones

Triiodothyronine (T3) and thyroxine (T4) are amine hormones produced, stored, and secreted by the thyroid gland. Thyroid hormones are iodinated derivatives of the amino acid tyrosine, with the subscripts denoting the number of iodine atoms in the molecule. Of the two iodinated thyronines, thyroxine is predominant; accounting for approximately one-third of the total iodine in the thyroid, with less than ten percent in the form of T3. Thyroid hormones are found in the bloodstream primarily bound to thyroxine binding globulin. A very low percentage of hormone circulates unbound. The concentration of T4 in plasma is much greater than T3 due to its greater affinity for the binding protein. Conversion of T4 to T3 by peripheral deiodination of the T4 molecule suggests that T4 may serve as a storage form of the more biologically active T3.

Secretion of thyroid hormone is under control of the hypothalamic-pituitary axis. The hypothalamic releasing hormone, thyrotropin releasing hormone (TRH), stimulates secretion of thyroid stimulating hormone (TSH, thyrotropin) from the anterior pituitary. Thyroid stimulating hormone stimulates release of T3 and T4 (thyroid hormone) from the thyroid gland. Thyroid hormone exerts a negative feedback on the anterior pituitary to decrease the sensitivity of TSH secreting cells to the stimulatory effects of TRH.

Hammond et al. (1984) used Hereford steers to investigate the rhythmicity of circulating T3 and T4. Time series analysis suggested 12 and 24 hour cyclical trends for T3, which may have been related to feeding period. Thyroxine demonstrated a 24 hour cyclical pattern and relatively larger values were found in the early afternoon and decreasing values through the morning hours. However, day and time had no significant effect on T3 or T4 as concentrations over a 48 hour period varied only 8 and 0.3 ng/ml for T4 and T3, respectively. There was a tendency to increase concentrations of both hormones at or shortly after feeding.

Thyroid hormones do not seem to be strongly influenced by cattle age. Irvin and Trenkle (1971) studied the effects of age, breed, and sex on the concentration of protein-bound iodine (PBI, thyroid hormone index) in cattle from 18 to 371 days of age. No differences were found although 18 day old cattle tended to have higher average concentrations of PBI. Similar findings were reported by Trenkle (1970) who found no

variation in PBI concentration over a 142 day feeding period with older cattle. Blood samples taken for 12 months in cattle from 5 to 17 months of age revealed concentrations of T3 increased during the first 4 months of the experiment and T4 concentrations decreased slightly during the same period (Verde and Trenkle, 1987). For the remainder of the study, T3 remained steady while T4 increased. Patterns of the concentrations of thyroid hormones were similar for all groups of cattle studied. Work in Belgium would support the findings of a slight increase in T4 concentrations with age (Fabry, 1983). Advancing age has no effect on the secretory pattern of TSH or the clearance and secretion rates of TRH in rams (Morrison et al., 1981).

Little research has been conducted to study the relationship between sex and thyroid hormones. Kahl and Bitman (1983) found bulls to have higher T3 and T4 concentrations than heifers between 1 and 4 months of age. Over a longer time period, Irvin and Trenkle (1971) saw no differences in PBI related to sex. Similarly, Anderson et al. (1973) found no differences in growing Jersey heifers and bulls.

Ellenberger et al. (1989) investigated thyroid hormone status in steers during compensatory and normal growth and dietary restriction. During restricted growth, mean serum concentrations of T4 were lower and T3 concentrations remained unchanged. Upon realimentation T4 concentrations increased. Reductions in T3 and T4 concentrations have been associated

with calorie-restricted diets in the rat (Schalch and Cree, 1985). In periparturient cows, elevated T3 and T4 concentrations were associated with diets exceeding NRC energy requirements versus those fed at NRC recommendations (Pethes et al., 1985). The authors also noted that T3 paralleled T4 throughout the experiment. Hammond et al. (1984) reported plasma T4, but not T3, concentration increased with increasing nitrogen level in the diet. However, this increase could have been related to a trend toward higher digestible energy intake with the higher nitrogen diets. The same workers failed to show differences in thyroid hormone concentrations in steers fed on two winter nutritional levels or during grazing (Hammond et al., 1990).

Thyroid hormones are important in bone growth as hypothyroidism results in decreased bone growth. Hyperthyroidism increases bone resorption but has no effect on net bone growth (Spencer, 1989). Mundy et al. (1976) showed thyroid hormone can directly stimulate bone resorption. Receptors for T3 have been found on chondrocytes in the growth plate and thyroid hormone administration to hypothyroid animals increases the size of the growth plate (Spencer, 1989). The finding that dwarf chickens have lower circulating T3 concentrations would support the theory that normal growth is dependent on a euthyroid state (Bowen et al., 1987).

Skeletal muscle protein synthesis and degradation are affected by thyroid hormone status. Reduced growth is associated with hypothyroidism and hyperthyroidism. A minimal

amount of T3 is essential for normal muscle growth and suboptimal concentrations lead to dwarfism (Goldberg et al., 1980) as seen in the chicken (Lilburn et al., 1986; Bowen et al., 1987). Thyrotoxicosis is accompanied by weight loss and severe muscle wasting. Goldberg et al. (1980) attempted to clarify the effects of high and low doses of thyroid hormones on muscle. The authors compared the effects of catabolic (high) and anabolic (low) doses of T4 on muscle protein synthesis and breakdown in hypophysectomized rats. Rates of protein synthesis did not differ in the two groups. However, rates of protein degradation were 50 to 75 percent greater in the high dose group suggesting increased protein catabolism was responsible for severe muscle wasting associated with hyperthyroidism. Thyroidectomized animals have reductions in both protein synthesis and degradation causing growth to cease (Goldberg, 1980). In a sex-linked abnormality causing dwarfism in chickens, Bowen et al. (1987) observed that T3 supplementation could increase growth. The same treatment decreased growth in normal strains (normal T3 concentrations) which agrees with the adverse effects of excess thyroid hormone.

Triiodothyronine may influence GH and IGF production and activities in tissue. Thyroidectomized rats have depressed hypothalamic GRF and rats treated with an antithyroid drug have reduced pituitary and plasma GH concentrations (Cabello and Wrutniak, 1989). In the dwarf mouse or hypophysectomized rat, administration of thyroid hormone and GH increased or

restored concentrations of IGF (Cabello and Wrutniak, 1989). Froesch et al. (1976) indicated that T3 is needed for maximum stimulation of chick cartilage by IGF. Thyroid hormones have been found to be positively related to IGF concentrations in backgrounded but not feedlot steers (Hammond et al., 1990). Hoshino et al. (1982) reported reduced T3 and IGF-I concentrations in dwarf chickens. Thyroid hormones may be positively related to insulin (Weekes, 1986; Verde and Trenkle, 1987). Relationships between thyroid hormones and other classical hormones (GH, IGF-I, insulin) need further clarification.

Efforts to relate circulating concentrations of thyroid hormones to different cattle types and weight gains have generally been unsuccessful and difficult to interpret. Irvin and Trenkle (1971) found PBI to be similar between various purebred and crossbred British breeds. Similar results have been obtained in three frame sizes of cattle with differing propensities to deposit fat (Grigsby and Trenkle, 1986) and in strains of chickens selected for growth (Goddard et al., 1988). In contrast, Verde and Trenkle (1987) reported large framed, fast growing steers (Simmental cross) had higher mean T4 concentration than small framed, slower growing steers (Angus-Hereford cross). No difference was observed in T3 concentration. Thyrotropin secretion was similar between breeds (Ohlson et al., 1981) and did not change with selection for growth (Davis et al., 1983) in cattle. However, Dodson et al. (1983) indicated higher overall means and baseline values

for TSH in Targhee rams selected for rate and efficiency of gain.

Trenkle (1970) found no relationship between PBI and weight gain in steers while Kahl and Bitman (1983) indicated a positive correlation between thyroid hormones and weight gain in Holstein calves. Variability in the relationships between thyroid hormones and daily gain in cattle are best demonstrated by the results of Fabry (1983). A significant positive correlation existed between daily gains over a 12 month period and T4 concentrations measured at 8 to 10 and 15 to 20 days of age. However, in a separate experiment, significant negative correlations existed between daily gains during a 1 year period and T4 sampled at the end of the first month of life.

Verde and Trenkle (1987) reported positive correlations between T4 and dry matter intake or body weight of both small and large frame steers during a 12 month period. Standal et al. (1987) indicated correlations between thyroid hormones and production traits (feed intake, growth rate, feed conversion efficiency) in growing heifers were low. Measures of thyroid hormones have not been found to be related to carcass traits (Purchas et al., 1971).

It is not surprising that attempts by several workers to relate thyroid status to growth have been unsuccessful. Growth of various tissues may be dependent on the euthyroid state as growth is slowed above or below an optimal concentration. A number of clinical or experimental

observations underline the importance of thyroid hormones in the regulation of growth. However, further research is needed to allow a more complete understanding of the relationships between T3 and T4 with other hormones and growth.

OBJECTIVES

The changes in cattle type that have occurred in the last two decades have been well documented. These changes have resulted in larger framed, later maturing animals that are able to attain heavier weights while maintaining carcass traits that are acceptable to consumers. Most of these changes have been the result of genetics through the introduction of new breeds and through the advancement of selection practices and technologies.

The process of growth and development is generally thought to be primarily under the control of the endocrine system. Several groups of workers have conducted studies to relate differences in serum hormone concentrations with differences in growth rate and carcass composition of cattle. The primary objective of this study was to evaluate differences of critical hormones among four distinct biological types of cattle. These populations of cattle offer a unique opportunity to evaluate changes in hormone parameters that have occurred as a result of selection. Assessment of the relationship of these hormones to various measures of rate and composition of gain was also intended.

Recent research with administration of exogenous hormone, in conjunction with in vitro techniques, has greatly enhanced

our understanding of how hormones affect growth and development. However, specific roles of individual hormones, and how hormones interact to influence biological systems have yet to be determined.

With these thoughts in mind, this experiment was designed with the following null hypotheses:

1. Circulating hormone concentrations of growing beef steers will be unaffected by breed, biological type, or selection for growth.

2. Circulating hormone concentrations of growing beef steers will be unrelated to growth rate, carcass traits, and measures of carcass composition.

MATERIALS AND METHODS

Cattle and Management

One hundred fifty nine steers from four breed groups were utilized in a two year experiment to evaluate the relationship among various hormones and rate, efficiency, and composition of gain. The steers utilized were obtained from the herds assigned to a breeding project at the Lake City Experiment Station, Lake City, Michigan. Group 1, an unselected Hereford (UH) herd, had no selection practiced since the initiation of the project in 1966. Group 2 (selected Hereford) steers came from the same original parentage as group 1. Cows in group 2 were artificially inseminated to superior growth (yearling weight) sires within the Hereford breed. Groups 3 and 4 were rotational crossbreeding herds. Moderate sized, moderate milk production breeds (Shorthorn, Angus, Hereford; SAH) comprised group 3. Group 4 consisted of three large sized, high milk production breeds (Gelbvieh, Simmental, Holstein; GSH). Selection in both crossbred groups was for yearling weight. In both years, Shorthorn and Gelbvieh served as sire breeds for groups 3 and 4, respectively. A summary of breed groups and estimated frame scores is given in Table 1.

After weaning, cattle were weighed and transported 220 km to the test facility. Initial weight was determined by the

Table 1. Description of different breed groups

Group	Selection criteria	Frame score	SEM
1 Unselected Herefords (UH)	None	1.6[a]	.13
2 Selected Herefords (SH)	Growth	5.3[b]	.10
3 Shorthorn x Angus x Hereford (SAH)	Growth	6.0[c]	.10
4 Gelbvieh x Simmental x Holstein (GSH)	Growth	6.3[d]	.11

[a,b,c,d] Means within a column lacking a common superscript differ ($P < .01$).

average of weights taken on two consecutive days upon arrival. At weaning, all calves were vaccinated for clostridial and respiratory diseases, treated for internal and external parasites and given a growth-promotant implant containing estradiol and progesterone[1]. Cattle within a breed group were allotted to pens to equalize age and randomly assigned into three slaughter groups (Table 2). Each slaughter group consisted of one pen of steers from each breed group. Cattle were housed on the south side of a covered, open sided slatted floor facility. Cattle were allowed a minimum of 1.86 square meters per animal.

Steers were adjusted to an 80% concentrate diet (Table 3) within 21 d after arrival at the test facility. Steers were given ad libitum access to diets and fresh feed added once daily. Pen feed refusals were collected and weighed weekly. Cattle were weighed prior to feeding at 28 d intervals. Feedstuffs were collected at two week intervals and analyzed for dry matter and protein content (AOAC, 1984).

Carcass Composition

Cattle were weighed on two consecutive days immediately prior to slaughter and the average recorded as final weight. Hip heights were taken on all steers approximately one week prior to the first slaughter. Cattle were transported 114 km to a commercial slaughter facility and slaughtered within one

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[1]Synovex-S. Syntex Animal Health Inc., West Des Moines, IA.

Table 2. Effects of breed group and year on initial age (d) of cattle in slaughter groups

Slaughter group	Year	Breed group UH	SH	SAH	GSH
1	1	195 ± 8.9	198 ± 7.5	201 ± 7.5	194 ± 7.0
2	1	200 ± 8.9	198 ± 7.0	207 ± 7.5	191 ± 7.5
3	1	200 ± 8.9	200 ± 7.0	202 ± 7.5	195 ± 7.0
1	2	192 ± 10.0	167 ± 7.0	191 ± 7.0	184 ± 7.5
2	2	196 ± 8.9	168 ± 8.1	191 ± 7.5	194 ± 8.9
3	2	195 ± 8.9	169 ± 7.5	189 ± 7.0	185 ± 7.5

Table 3. Diet composition[a]

Component	Percentage of dry matter
High moisture corn	85.0
Corn silage	10.0
Supplement[b]	5.0

[a] Diet was formulated to provide 11.0% crude protein and contained 2.4 Mcal NEm/kg and 1.48 Mcal NEg/kg of dry matter.

[b] Supplement ingredients (as-fed basis): soybean meal, 50.1%; calcium carbonate, 20.9%; trace mineral salt, 9.5%; urea, 7.1%; potassium chloride, 5.3%; dicalcium phosphate 2.1%; ground corn, 3.5%; Selenium 200, 1.0%; vitamin A, .15%, Rumensin 60, .35%. The total diet was formulated to contain: Ca, .5%; P, .35%; K, .6%; Se, .02 mg/kg; vitamin A, 454 IU/kg; Monensin, 4.54 mg/kg.

hour.

Approximately 24 h postmortem, carcasses were ribbed and carcass characteristics measured. Fat thickness, ribeye area, maturity, marbling score, adjusted fat thickness, and percentage kidney, pelvic, and heart fat were determined by trained university personnel. Yield grades were calculated and quality grades assessed.

A five rib section (ribs 9 to 13) was removed from each carcass and transported to the Michigan State University Meats Laboratory. The 9-10-11 rib section was prepared according to procedures described by Hankins and Howe (1946). The 9-10-11 rib section was deboned, bone and soft tissue weights recorded, and the soft tissue ground three times. Soft tissue was mixed by hand between grindings to assure a representative sample. Approximately 450 g of sample was collected and stored in a Whirlpack bag at -30 degrees C until further preparation. Samples were thoroughly homogenized with liquid nitrogen in a large, industrial strength Waring blender prior to dry matter, protein, and ether extract analysis.

Triplicate samples were dried in aluminum pans for 48 h at 60 degrees C to determine dry matter content (AOAC, 1984). Crude protein content of duplicate samples was calculated from total nitrogen as determined by the Kjeldahl procedure using a Technicon auto-analyzer system (AOAC, 1984). Fat content of each sample was determined in triplicate by ether extraction for 12 h in a Soxhlet apparatus. Percentage carcass moisture, fat and protein were estimated from rib fat and protein using

the equations of Crouse and Dikeman (1974). Estimations of percentage carcass bone were made using the equation developed by Hankins and Howe (1946).

Blood Collection and Hormone Assays

One pen of cattle from each breed group was assigned a bleeding date corresponding with slaughter group (Table 4). Approximately 21 d prior to blood collection, cattle were placed in individual stalls in the metabolism room at the MSU Beef Cattle Research Center. Diets and feeding regimen remained consistent with cattle in pens. Dry matter intake was measured on individual animals while in the metabolism stalls. Over the 21 d adaption period, steers were adapted to halters to facilitate blood collection. Under veterinary supervision, steers were fitted with a polyvinyl cannula in the jugular vein the day prior to blood collection. The next day, beginning at 0900 h, blood samples were taken from each steer every 30 min for 8 h. Blood samples were stored at room temperature for 2 to 4 h and stored overnight at 4 degrees C. Serum was obtained by centrifugation at 2000 x g for 25 min. Serum was decanted and stored at -20 degrees C until further analysis. Steers were returned to original pens the day following blood collection.

Serum bovine growth hormone was quantified using a double antibody radioimmunoassay (Zinn et al., 1989). Analysis of pulsatile GH secretion was performed using PULSAR (Merriam and Wachter, 1982). Binding proteins for IGF-I were removed by

Table 4. Blood collection and slaughter schedule

Slaughter group	Year	Days on feed: Blood collection	Days on feed: Slaughter
1	1	137	230
2	1	165	251
3	1	200	265
1	2	120	220
2	2	148	243
3	2	176	255

formic acid/ethanol extraction as reported by Bruce et al. (1991). The international IGF-I reference standard (Bristow et al., 1990) was used as the standard. Insulin-like growth factor I concentration of serum extracts was measured by radioimmunoassay using rabbit anti-hIGF-I (L. Underwood, University of North Carolina-Chapel Hill, personal communication). The antisera is specific for IGF-I and had less than .5% cross-reactivity with IGF-II. After overnight incubation of samples and standards with first antibody, labeled IGF-I was added and samples were incubated for an additional 48 h. Bound IGF-I was precipitated with Staphloccocus aureus protein (Sigma Chemical Company, St. Louis, MO) and the resulting pellet was counted. Commercially prepared radioimmunoassay kits were used to quantitate serum insulin, T4 (Corning Medical, Medfield, MA), and T3 (Refsal et al., 1984).

Statistical Analysis

Data were analyzed by analysis of variance with breed group, slaughter group, and year as the main effects. All interactions were included in the model. Analysis was performed using the General Linear Models Subroutine of SAS (SAS, 1987). Initial age was included as a covariate due to the young age of SH·steers in year two (Table 2). Least square means with standard errors are presented in the tables.

RESULTS

Feedlot Performance

Feedlot performance reported on an animal basis for breed groups (BG) and slaughter groups (SG) is shown in Tables 5 and 6, respectively. Unselected Hereford steers were lightest initially and at slaughter ($P < .01$). Initial and final weights increased ($P < .01$) as frame score increased among BG. Final weights increased with time on feed ($P < .01$). Selected Hereford steers gained the fastest and UH steers the slowest across all SG ($P < .01$). Crossbred steers (SAH and GSH) were intermediate to UH and SH, but not different from each other for ADG. Average daily gains were similar across SG.

Feed intakes and feed conversion efficiencies are reported on a pen basis over the entire feeding period. Daily feed intake paralleled live weight for UH, SH, and GSH steers. Daily feed intake was highest for SAH steers ($P < .01$). Steers in SG 3 consumed more feed on a daily basis than steers in SG 1 and SG 2 ($P < .01$). Unselected and selected Hereford steers required less feed per unit of gain than SAH or GSH steers ($P < .01$) over the entire trial, with UH steers having the most desirable feed conversion numerically. Feed conversion efficiency tended to decrease with time on feed (Table 6).

Table 5. Feedlot performance of breed groups

Item	Breed group			
	UH	SH	SAH	GSH
Initial wt, kg	152.5[a] ± 4.8	214.0[b] ± 3.9	256.5[c] ± 3.9	277.3[d] ± 4.0
Final wt, kg	403.1[a] ± 8.1	539.3[b] ± 6.8	557.6[c] ± 6.6	584.6[d] ± 6.8
ADG, kg	1.03[a] ± .02	1.33[c] ± .02	1.24[b] ± .02	1.26[b] ± .02
DMI, kg/steer/d	5.69[a] ± .09	7.54[b] ± .09	8.20[d] ± .09	7.98[c] ± .09
Feed conversion efficiency, feed/gain	5.53[a] ± .14	5.66[a] ± .14	6.60[b] ± .14	6.34[b] ± .14

[a,b,c,d] Means within a row lacking a common superscript differ ($P < .01$).

Table 6. Feedlot performance of slaughter groups

Item	Slaughter group 1	2	3
Initial wt, kg	219.5[a] ± 3.5	221.7[a] ± 3.6	233.9[b] ± 3.5
Final wt, kg	494.7[a] ± 6.1	522.6[b] ± 6.2	546.1[c] ± 6.0
ADG, kg	1.22 ± .02	1.22 ± .02	1.20 ± .02
DMI, kg/steer/d	7.21[a] ± .08	7.32[a] ± .08	7.52[b] ± .08
Feed conversion efficiency, feed/gain	5.86 ± .12	5.99 ± .12	6.24 ± .12

[a,b,c] Means within a row lacking a common superscript differ ($P < .01$).

Carcass Characteristics

Differences in carcass characteristics among BG reflect the diversity of cattle types used in this study. Carcass measurements of BG are shown in Table 7. Carcass weights paralleled live weights and there were no differences in dressing percentage due to BG. As frame size and slaughter weight increased among BG, fat thickness decreased while carcass weight and ribeye area (REA) increased ($P < .01$). Across all SG (Table 8), UH carcasses had the highest REA/carcass weight ($P < .01$), marbling score ($P < .05$), and corresponding quality grade ($P < .10$). Carcass weights and final weights increased as time on feed (SG) increased. Fat thickness ($P < .05$), REA and marbling score ($P < .01$) were lowest for SG 1. Slaughter group 3 carcasses had the smallest REA on a carcass weight basis ($P < .01$). Significant SG x year interactions existed for marbling score ($P < .01$) and quality grade ($P < .10$). Least squares means of the slaughter group x year interactions are listed in Table 12.

Carcass Composition

Proportions of carcass fat, protein, moisture, and bone for BG and SG are given in Tables 9 and 10, respectively. Unselected Hereford carcasses had the highest percentages of carcass fat, and GSH carcasses were the leanest across all BG ($P < .01$). Estimates of carcass protein and moisture were inversely related to carcass fat. Carcass fat increased with SG, while carcass protein and moisture decreased ($P < .01$).

Table 7. Carcass measurements of breed groups

Item	Breed group UH	SH	SAH	GSH
Carcass wt, kg	244.1[c] ±5.3	329.5[d] ±4.4	346.6[e] ±4.3	364.4[f] ±4.5
Fat thickness, mm	12.6[c] ±.62	10.4[d] ±.52	8.5[e] ±.51	7.2[f] ±.52
Adjusted fat thickness, mm	15.4[c] ±.66	12.3[d] ±.55	10.1[e] ±.54	8.3[f] ±.55
Ribeye area, cm^2	67.0[c] ±1.4	77.6[d] ±1.2	80.7[e] ±1.3	90.9[f] ±1.2
REA/carcass wt; cm^2/kg	.276[c] ±.004	.236[e] ±.003	.234[e] ±.003	.250[d] ±.003
Yield grade	3.29[c] ±.10	3.04[d] ±.09	2.91[d] ±.09	2.34[e] ±.09
Marbling score[a]	548[g] ±11.3	507[h] ±9.3	521[h] ±9.2	504[h] ±9.4
Quality grade[b]	12.1[i] ±.16	11.6[j] ±.14	11.7[j] ±.13	11.5[j] ±.14

a 400 = Slight 0; 500 = Small 0.
b 11.0 = high Select; 12.0 = low Choice.
c,d,e,f Means within a row lacking a common superscript differ ($P < .01$).
g,h Means within a row lacking a common superscript differ ($P < .05$).
i,j Means within a row lacking a common superscript differ ($P < .10$).

Table 8. Carcass measurements of slaughter groups

Item	Slaughter group 1	2	3
Carcass wt, kg	305.4[c] ±4.0	321.3[d] ±4.1	336.8[e] ±3.9
Fat thickness, mm	8.6[f] ±.46	10.0[g] ±.48	10.4[g] ±.45
Adjusted fat thickness, mm	10.8 ±.49	11.9 ±.50	11.9 ±.48
Ribeye area, cm^2	75.8[c] ±1.1	80.9[d] ±1.1	80.5[d] ±1.1
REA/carcass wt; cm^2/kg	.251[d] ±.003	.255[d] ±.003	.241[c] ±.003
Yield grade	2.86 ±.08	2.84 ±.08	3.00 ±.08
Marbling score[a]	498[c] ±8.4	531[d] ±8.6	532[d] ±8.2
Quality grade[b]	11.5 ±.12	11.8 ±.13	11.8 ±.12

a 400 = Slight 0; 500 = Small 0.
b 11.0 = high Select; 12.0 = low Choice.
c,d,e Means within a row lacking a common superscript differ ($P < .01$).
f,g Means within a row lacking a common superscript differ ($P < .05$).

Table 9. Carcass composition of breed groups

Item	Breed group			
	UH	SH	SAH	GSH
Carcass fat, %	$36.5^{c} \pm .53$	$34.2^{b} \pm .44$	$33.5^{b} \pm .43$	$30.0^{a} \pm .44$
Carcass protein, %	$13.5^{a} \pm .16$	$13.9^{b} \pm .13$	$14.3^{c} \pm .13$	$15.0^{d} \pm .13$
Carcass moisture, %	$49.2^{a} \pm .37$	$50.7^{b} \pm .31$	$51.2^{b} \pm .30$	$53.6^{c} \pm .31$
Carcass bone, %	$13.8^{a} \pm .16$	$14.5^{b} \pm .14$	$14.8^{c} \pm .13$	$14.8^{c} \pm .14$

^{a,b,c,d} Means within a row lacking a common superscript differ ($P < .01$).

Table 10. Carcass composition of slaughter groups

Item	Slaughter group 1	2	3
Carcass fat, %	$32.2^{a} \pm .40$	$33.3^{b} \pm .41$	$35.1^{c} \pm .39$
Carcass protein, %	$14.3^{b} \pm .12$	$14.5^{b} \pm .12$	$13.7^{a} \pm .12$
Carcass moisture, %	$52.0^{c} \pm .28$	$51.2^{b} \pm .28$	$50.3^{a} \pm .27$
Carcass bone, %	$14.5 \pm .12$	$14.5 \pm .13$	$14.5 \pm .12$

a,b,c Means within a row lacking a common superscript differ ($P < .01$).

Table 11. Influence of breed group and year on selected carcass characteristics

	Breed group			
	UH	SH	SAH	GSH
Year 1				
Quality grade[ab]	12.1 ± .23	11.1 ± .19	11.8 ± .20	11.3 ± .18
Carcass fat, %[b]	37.2 ± .73	35.4 ± .60	33.6 ± .63	29.5 ± .59
Carcass protein, %[b]	13.1 ± .22	13.6 ± .18	14.5 ± .19	15.0 ± .18
Carcass moisture, %[b]	48.6 ± .51	49.8 ± .42	51.1 ± .44	54.0 ± .41
Year 2				
Quality grade	12.1 ± .24	12.0 ± .21	11.7 ± .18	11.7 ± .20
Carcass fat, %	35.7 ± .76	32.9 ± .68	33.4 ± .59	30.5 ± .66
Carcass protein, %	13.8 ± .23	14.1 ± .20	14.2 ± .18	15.1 ± .20
Carcass moisture, %	49.8 ± .53	51.6 ± .48	51.2 ± .41	53.3 ± .46

[a] 11.0 = high Select; 12.0 = low Choice.
[b] Breed group x year interaction (P<.10).

Table 12. Influence of slaughter group and year on selected carcass characteristics

Item	Slaughter group 1	2	3
Year 1			
Marbling score[ac]	488 ± 11.8	494 ± 11.9	538 ± 11.7
Quality grade[bd]	11.4 ± .17	11.4 ± .17	11.8 ± .17
Carcass fat, %[d]	33.3 ± .55	33.4 ± .56	35.0 ± .55
Carcass moisture, %[d]	51.0 ± .39	51.1 ± .39	50.4 ± .38
Year 2			
Marbling score	507 ± 12.2	569 ± 12.7	526 ± 11.9
Quality grade	11.6 ± .18	12.2 ± .19	11.9 ± .17
Carcass fat, %	31.0 ± .57	33.1 ± .60	35.2 ± .56
Carcass moisture, %	52.9 ± .40	51.2 ± .42	50.3 ± .39

[a] 400 = Slight 0, 500 = Small 0.
[b] 11.0 = high Select, 12.0 = low Choice.
[c] Slaughter group x year interaction ($P < .01$).
[d] Slaughter group x year interaction ($P < .10$).

Year x BG interactions (P < .10) existed for carcass fat, protein, and moisture. Significant SG x year interactions (P < .10) existed for carcass fat and moisture. Least squares means for selected carcass characteristics are given in Tables 11 and 12.

Hormone Parameters

Serum hormone concentrations for each breed group are reported in Table 13. Each GH value shown represents the mean of 17 serum samples analyzed on each steer. Hourly serum samples from each steer were pooled for quantification of IGF-I, insulin, T3 and T4.

Across all bleed groups, UH and GSH steers had higher (P < .01) GH concentrations than SH and SAH steers. A bleed group x breed group interaction (P < .01) existed for GH. Least squares means are reported in Table 15. Insulin-like growth factor I concentrations paralleled GH. Unselected Hereford steers had higher IGF-I concentrations than other BG (P < .01), with SH and GSH steers not different from each other but higher than SAH steers. Purebred steers (UH and SH) had higher (P < .01) insulin concentrations than crossbred steers. Triiodothyronine (P < .10) and thyroxine (P < .01) concentrations were lower in SH steers than other BG. Thyroxine concentrations were found to be higher in UH steers than SH or SAH steers.

The effects of sampling date on serum hormone means are shown in Table 14. Growth hormone and IGF-I means declined

Table 13. Serum hormone concentrations for breed groups

Item	Breed group			
	UH	SH	SAH	GSH
Growth hormone, ng/ml	$3.70^{b} \pm .17$	$3.31^{a} \pm .14$	$3.23^{a} \pm .14$	$3.89^{b} \pm .14$
IGF-I, ng/ml	$880.5^{c} \pm 28.3$	$795.3^{b} \pm 23.5$	$724.1^{a} \pm 23.1$	$808.4^{b} \pm 23.6$
Insulin, µU/ml	$35.1^{b} \pm 1.9$	$33.9^{b} \pm 1.6$	$27.2^{a} \pm 1.6$	$27.4^{a} \pm 1.6$
T_3, ng/ml	$2.40^{e} \pm .06$	$2.21^{d} \pm .05$	$2.36^{e} \pm .05$	$2.35^{e} \pm .05$
T_4, ng/ml	$102.4^{c} \pm 2.8$	$87.3^{a} \pm 2.3$	$95.0^{b} \pm 2.3$	$98.9^{bc} \pm 2.4$

^{a,b,c} Means within a row lacking a common superscript differ ($P < .01$).
^{d,e} Means within a row lacking a common superscript differ ($P < .10$).

Table 14. Serum hormone concentrations for bleed groups

Item	Bleed group 1	2	3
Growth hormone, ng/ml	4.06[c] ± .13	3.42[b] ± .13	3.12[a] ± .13
IGF-I, ng/ml	898.6[c] ± 21.1	797.3[b] ± 21.7	710.4[a] ± 20.7
Insulin, µU/ml	30.8 ± 1.4	32.6 ± 1.5	29.2 ± 1.4
T_3, ng/ml	2.36[b] ± .05	2.44[b] ± .05	2.20[a] ± .05
T_4, ng/ml	98.2 ± 2.1	95.2 ± 2.2	94.3 ± 2.1

[a,b,c] Means within a row lacking a common superscript differ ($P < .01$).

Table 15. Influence of breed group and bleed group or growth hormone concentration (ng/ml)[a]

	Breed group			
Bleed group	UH	SH	SAH	GSH
1	4.11 ± .31	4.21 ± .24	3.45 ± .24	4.46 ± .24
2	4.00 ± .29	2.85 ± .25	3.29 ± .25	3.53 ± .27
3	2.99 ± .29	2.87 ± .24	2.95 ± .24	3.68 ± .24

[a] Breed group x bleed group interaction ($P < .01$).

over time ($P < .01$). Concentrations of T3 were lowest for bleed group 3 ($P < .01$).

Analysis of growth hormone secretion for breed and bleed groups are shown in Tables 16 and 17, respectively. Baseline GH concentration was highest ($P < .05$) for Gelbvieh-sired crossbred steers. Ranking of breed groups by growth hormone concentration for the eight hour sampling period was similar whether measured by area under the curve or mean GH concentration. No differences were detected for peak number or time between peaks (inter-peak interval), although UH steers had numerically fewer peaks and a longer inter-peak interval than other BG. The lowest ($P < .01$) peak amplitude was calculated for SAH steers.

Growth hormone secretion patterns across all BG over time are reported in Table 17. Higher ($P < .01$) baseline GH concentrations were found in steers in bleed group 1. Growth hormone area under the curve declined over time ($P < .01$). Steers in bleed group 1 had a higher peak number and frequency ($P < .01$), along with a longer inter-peak interval ($P < .10$) than steers in bleed groups 2 and 3. Lower ($P < .01$) peak amplitudes were reported for bleed group 3.

Serum hormone relationships for breed groups are presented in Table 18. Purebred Hereford steers had higher ratios of IGF-I/GH and insulin/GH than GSH steers. Shorthorn-sired crossbred steers had less ($P < .01$) IGF-I and insulin per unit of GH than SH steers. GSH steers had the lowest ($P < .01$) insulin/GH ratio when compared to all BG. Both

Table 16. Growth hormone analysis of breed groups

Item	Breed group UH	SH	SAH	GSH
Baseline GH, ng/ml	2.77[a] ±.12	2.62[a] ±.10	2.77[a] ±.10	3.07[b] ±.10
GH area under curve, ng x min/ml	1774[d] ±81.1	1593[c] ±67.3	1556[c] ±66.1	1853[d] ±67.6
Peak no.	1.18 ±.16	1.69 ±.13	1.54 ±.13	1.66 ±.13
Peak amplitude, ng/ml	6.06[d] ±.73	5.72[d] ±.60	3.96[c] ±.59	5.90[d] ±.61
Inter-peak interval, min	158 ±23.9	109 ±19.8	109 ±19.5	82 ±19.9

[a,b] Means within a row lacking a common superscript differ ($P < .05$).
[c,d] Means within a row lacking a common superscript differ ($P < .01$).

Table 17. Growth hormone analysis of bleed groups

Item	Bleed group 1	2	3
Baseline GH, ng/ml	3.12[b] ± .08	2.67[a] ± .09	2.62[a] ± .09
GH area under curve, ng x min/ml	1952[c] ± 60.6	1651[b] ± 62.2	1479[a] ± 59.3
Peak no.	1.99[b] ± .12	1.50[a] ± .12	1.54[a] ± .12
Peak amplitude, ng/ml	6.57[b] ± .54	5.59b ± .56	4.06[a] ± .53
Peak frequency, peaks/min	.0042[b] ± .0002	.0031[a] ± .0003	.0032[a] ± .0002
Inter-peak interval, min	150.0[d] ± 17.9	97.1[c] ± 18.3	97.2[c] ± 17.5

[a,b] Means within a row lacking a common superscript differ (P < .01).
[c,d] Means within a row lacking a common superscript differ (P < .10).

Table 18. Serum hormone relationships for breed groups

	Breed group			
Item	UH	SH	SAH	GSH
IGF-I/GH	$253.4^{bc} \pm 12.2$	$264.0^{c} \pm 10.1$	$231.7^{ab} \pm 10.0$	$218.8^{a} \pm 10.2$
Insulin/GH	$10.2^{bc} \pm .76$	$11.3^{c} \pm .63$	$9.0^{b} \pm .62$	$7.7^{a} \pm .63$
Insulin/IGF-I	$.041^{e} \pm .003$	$.044^{e} \pm .002$	$.040d^{e} \pm .002$	$.035^{d} \pm .002$

a,b,c Means within a row lacking a common superscript differ ($P < .01$).
d,e Means within a row lacking a common superscript differ ($P < .10$).

Hereford breed groups had higher ($P < .10$) ratios of insulin/IGF-I than GSH steers.

Table 19 illustrates the effects of bleed group on relationships of serum hormones. Steers sampled in bleed group 1 had significantly ($P < .01$) lower ratios of insulin/GH than steers in later bleed groups. The same response was noted for the relationship of insulin/IGF-I ($P < .10$).

Correlations between GH, IGF-I and insulin and selected carcass traits and estimates of carcass composition are given in Table 20. Growth hormone was negatively correlated with measures of fatness and positively correlated with estimates of carcass muscle. Similar correlations existed for IGF-I and certain carcass characteristics. Insulin concentration was positively correlated with carcass fat measures while being negatively related to carcass protein and moisture.

Correlations between serum hormone relationships and carcass characteristics are listed in Table 21. Ratios of insulin to GH and IGF-I were positively correlated with measures of fat in the carcass. Negative relationships existed between estimated carcass protein and moisture and insulin:GH and insulin:IGF-I ratios.

Table 19. Serum hormone relationships for bleed groups

Item	Bleed group 1	2	3
Insulin/GH	8.1[a] ± .56	10.5[b] ± .58	10.1[b] ± .55
Insulin/IGF-I	.035[c] ± .002	.042[d] ± .002	.043[d] ± .002

a,b Means within a row lacking a common superscript differ (P < .01).
c,d Means within a row lacking a common superscript differ (P < .10).

Table 20. Correlations between GH, IGF-I and insulin and carcass characteristics

	GH	Probability	IGF-I	Probability	Insulin	Probability
Fat thickness	-.22	.006	.06	.45	.29	.001
REA/carcass wt	.20	.01	.20	.01	.04	.65
Yield grade	-.17	.03	.08	.31	.23	.003
Marbling score	-.27	.001	.06	.45	.20	.009
Carcass fat	-.28	.001	-.17	.03	.22	.003
Carcass protein	.25	.002	.11	.19	-.21	.009
Carcass moisture	.25	.001	.15	.06	-.22	.004
Carcass bone	.08	.32	-.08	.35	.30	.001

Table 21. Correlations between serum hormone relationships and carcass characteristics

	IGF-I/ GH	Probability	Insulin/ GH	Probability	Insulin/ IGF-I	Probability
Fat thickness	.17	.03	.33	.001	.25	.001
Yield grade	.08	.30	.25	.001	.25	.001
Marbling score	.17	.03	.28	.001	.22	.007
Carcass fat	.08	.32	.29	.001	.32	.001
Carcass protein	-.11	.17	-.26	.001	-.26	.001
Carcass moisture	-.07	.37	-.28	.001	-.30	.001
Carcass bone	-.09	.26	-.24	.003	.22	.004

DISCUSSION

Differences in feedlot performance and carcass characteristics of the four breed groups reflect the effects of selection and diversity among breeds and biological types, as all steers were raised and managed at the same location and under the same conditions throughout the entire trial.

The dramatically higher initial weight, final weight, frame score and ADG of SH, SAH, and GSH steers versus UH steers demonstrate the effects of long-term selection for yearling growth. These expected results are in agreement with similar growth performance reported by Newman et al. (1973), Cundiff et al. (1991) and Parnell (1992). The corresponding increase in frame size with selection for growth was also reported by Hough et al. (1985). Presumably because of heavier weights throughout the feeding period and higher maintenance requirements, the two crossbred genotypes were less efficient in the conversion of feed to live animal gain.

Carcass results further magnify the effects of selection for growth observed in this study. One would expect the larger, later maturing cattle (SH,SAH, and GSH) to have an advantage in carcass composition as they would be younger physiologically; and therefore, would be depositing a lower proportion of fat in carcass gain. The superior marbling

scores and quality grades attained by UH carcasses are reflective of higher percentages of carcass fat. The ability of smaller, earlier maturing cattle types to attain acceptable quality grades with fewer days on feed is well documented (Crouse et al., 1985; Dikeman et al., 1985; Marshall et al., 1990). Although UH carcasses had the smallest absolute REA, due to lighter carcass weights, UH steers had the largest REA per kg carcass weight. This muscling advantage on a carcass weight basis existed despite the increased subcutaneous fat and higher percentage of carcass fat associated with UH carcasses.

The observed differences in carcass traits and measures of carcass composition are expected when comparing straightbred English-type steers with continental European crossbred steers (Smith et al., 1976). As expected, SAH and GSH steers had heavier carcasses, less backfat, larger REA, and lower yield grades than SH steers when slaughtered at a similar age. Estimates of percentage carcass fat, protein, and moisture demonstrate the same trends. Across slaughter groups, estimated carcass fat did not account for differences observed in marbling score among the three selected breed groups. Although not statistically different, the highest marbling scores were observed in SAH carcasses which were intermediate to SH and GSH in carcass fat, indicating differences in carcass fat depots among breeds or biological types (Smith et al., 1976; Arnold et al., 1990). Carcass characteristics reported for the three selected breed groups

are reflective of traits associated with the breed of sire for each respective cattle type (Smith et al., 1976; Crouse et al., 1985; Dikeman et al., 1985; Arnold et al., 1990).

Decreases in ADG and feed conversion efficiency with time on feed have been frequently reported (Smith et al., 1976; Thonney et al., 1981). The heavier body weights associated with each successive slaughter group may have resulted in higher maintenance requirements and reduced ADG. The increases in carcass weight, backfat, and REA in each successive slaughter group were expected (Smith et al., 1976; Thonney et al., 1981). The decrease in REA/carcass weight in slaughter group 3 would be expected as muscle deposition decreases in relation to fat deposition as the animal matures. Estimated carcass fat closely paralleled the differences in marbling score observed in successive slaughter groups. Carcass protein and moisture were inversely related to carcass fat ($R = -.78$ and $-.99$, respectively; $P < .01$).

Breed group x year least squares means for quality grade and estimated carcass composition illustrate variation in carcass characteristics between years for cattle treated alike. Unselected Hereford steers required 1.5% less carcass fat in year 2 to attain the same quality grade. Shorthorn-sired steer carcasses increased one-third of a quality grade with a decrease of 2.5% in carcass fat and crossbred Gelbvieh carcasses increased quality grades with increased carcass fatness. These results further indicate that quality grades are influenced by a number of factors, including breed and

genetics, and external fat or percentage carcass fat. A single indicator appears to be a poor predictor of carcass quality. This observation is critical to the current discussions about changing the quality grading system. Slaughter group x year interaction means also revealed an increase in marbling and quality grade with a decrease in percentage carcass fat for slaughter groups 1 and 2 in year 2.

The complexity of factors involved in the development of the various tissues involved in body growth make interpretation of hormone data in this study difficult. Due to the design of this study, hormone data are only available over a short window in each steer's life. Despite these complications, hormone data from this study are in general agreement with the literature in regard to the role of specific hormones and their interactions in the control and regulation of meat animal growth and development.

The nutritional status of the steers utilized in this study should not have had an effect on reported GH concentrations. Level of intake, fasting, and energy balance can all play a role in determining GH concentrations in the bovine animal (Trenkle, 1976; Villa-Godoy, 1987; Ellenberger et al., 1989). Although the cattle were subject to stress while in the metabolism stalls and during the sampling period, there is no evidence to suggest that malnutrition affected circulating GH concentration.

Growth hormone concentrations declined over time in this study as evidenced by a significant correlation between GH and

bleed group ($R = -.35$, $P < .01$). A decline in circulation GH over time has been observed by several workers using cattle of the same age (Trenkle, 1971; Trenkle and Topel, 1978; Keller et al., 1979; Anderson, 1987). Trenkle and Topel (1978) attributed the decline in ADG as cattle approach slaughter weight to decreases in circulating concentrations of GH. The significant decrease in GH across bleed groups in this study did coincide with an observed decrease in rate of gain over the same time period.

Larger breeds of beef cattle have been reported to have higher mean GH concentrations than smaller breeds (Ohlson et al., 1981; Verde and Trenkle, 1982; Grigsby and Trenkle, 1986). The fact that GSH steers had higher GH concentrations than either SH or SAH steers in this study would support these observations. Grigsby and Trenkle (1986) also found Simmental steers to have higher GH concentrations than British-bred steers, which is in agreement with the differences observed between the Gelbvieh-crossbred steers and straightbred Herefords in this study. In contrast to what has been previously reported, the larger cattle with higher GH concentrations did not demonstrate an advantage in rate of gain in this study.

The reasons for higher concentrations of GH in UH steers is not apparent to the authors. Elevated GH concentrations have been reported in slow growth strains of chickens (Goodard et al., 1988) and normal concentrations have been measured in dwarf Hereford cattle (Dev and Lasley, 1969). Selection for

growth has been shown to increase GH concentrations (Davis et al., 1983; Dodson et al., 1983). Growth hormone concentrations of the UH in this study do not support these findings. However, the possibility that many of the actions of GH are mediated by IGF-I does not make it surprising to find inconsistent relationships between GH values reported both in this study and in the literature. In addition, measurement of circulating concentrations of any hormone does not provide insight into other factors such as receptors and interaction with other hormones involved in growth and development.

Breed group x bleed group interaction means may provide insight as to the differences found in GH concentration between cattle type. All breed groups declined in GH concentration over time ($P < .01$). Unselected Hereford and SAH steers exhibited the sharpest decline in bleed group 3, while SH and GSH steer GH concentrations declined the most from bleed group 1 to bleed group 2. The influence these declines in GH concentration have on cattle performance and composition are unknown; but may have a role, in combination with other hormones, in partitioning of nutrients into specific tissues.

Patterns of GH secretion have been implicated as explanations for differences in growth rate and body size between sexes (Afinson et al., 1975; Keller et al., 1979; Gluckman et al., 1987). Higher peak amplitudes and baseline values are found in males which are known to have a larger

body size and later maturity pattern than females. The largest framed, latest maturing steers in this study (GSH) had numerically higher baseline GH concentrations and higher peak amplitude than other breeds, although these differences were not statistically significant.

Of more importance may be the analysis of GH secretory patterns over time and relationships to growth and development. Baseline GH concentrations, peak number, peak amplitude, and peak frequency all declined over time. Higher baseline concentrations and a greater number of peaks with higher amplitudes have been associated with increased growth rate and higher lean:fat ratios (Afinson et al., 1975; Keller et al., 1979). The pattern of GH secretion over time observed in this study would coincide with growth and compositional changes that occurred during the same period, as the steers declined in growth rate while fat deposition increased.

Insulin-like growth factor I concentrations paralleled GH, and declined over time in this study. Insulin-like growth factor I was negatively correlated with bleed group ($R = -.43$, $P < .01$). One would expect IGF-I concentrations to decline over time as concentrations of IGF-I in serum are directly related to GH (Clemmons et al., 1987; Gluckman et al., 1987). Davis and Bishop (1991) and Hammond et al. (1990) also reported IGF-I concentrations to decline with age in cattle. The close relationship between GH and IGF-I concentrations is also influenced by the role of IGF-I in GH secretion. Insulin-like growth factor I inhibits GH release from the

anterior pituitary through negative feedback (Berelowitz et al., 1981). Consequently, GH release from the anterior pituitary is inhibited by elevated IGF-I concentrations. Analysis of IGF-I concentrations for breed groups shows an association between IGF-I and GH concentrations. Those breed groups with higher serum concentrations of GH also had higher IGF-I concentrations.

Insulin-like growth factor I concentrations would be expected to parallel GH since many of the biological actions of GH are mediated by IGF-I. Administration of GH to humans (Clemmons et al., 1987) and sheep (Underwood et al., 1982) resulted in increased blood concentrations of IGF-I. Growth hormone may directly stimulate release of IGF-I from the liver and other tissues, thus explaining the tight relationship between concentrations of the two hormones in this study and others. Correlation analysis in this study revealed a positive relationship between GH and IGF-I ($R = .24$, $P < .01$). However, it explained only a small proportion of the variation.

Care should be exercised when interpreting the IGF-I results. The assay used is specific for IGF-I, but measures total immunoreactive IGF-I, including the large portion bound to transport proteins in serum. These transport proteins provide short term storage and transport IGF-I to target tissue. The transport proteins also render IGF-I inactive. Since the transport proteins were removed prior to hormone determination, the values reported in this study represent

total IGF-I and not necessarily the activity or use of the hormone. This may partially explain why GSH steers had significantly lower concentrations of IGF-I than UH steers when GH concentrations were similar. The higher concentrations of circulating IGF-I in UH steers may reflect a lower uptake of IGF-I from the circulatory system and more storage of the hormone compared to GSH steers. The association between serum IGF-I concentrations and metabolic utilization of the hormone by the animal requires further research.

The ratio of IGF-I:GH may give insight as to the utilization of IGF-I. A lower IGF-I/GH ratio would indicate a lower concentration of circulating IGF-I if GH concentrations were comparable. Therefore, a lower ratio may indicate greater tissue utilization with less of the hormone being stored bound to transport proteins. Insulin-like growth factor I is thought to have positive effects on bone and lean tissue deposition, with little influence on adipose tissue development. A high ratio of IGF-I/GH was found in the UH steers, and a low ratio was demonstrated in the GSH steers, who were larger framed and physiologically less mature at the time of sample collection. Advantages in carcass composition demonstrated in the GSH steers may have been partially attributed to increased utilization of IGF-I.

Circulating concentrations of insulin in the bloodstream are largely a function of the fed state of the animal (McAtee and Trenkle, 1971b; Weekes, 1986). Due to the great variation

in eating patterns of the steers in this study, hourly samples from each animal were pooled for determination of insulin. The pooled sample would minimize the secretory increase of insulin that occurs after a meal in cattle.

No significant differences were recorded in insulin concentration over time in this study. Insulin concentrations are lowest in young cattle and gradually increase with age and weight (Trenkle, 1970; Trenkle and Topel, 1978; Verde and Trenkle, 1987). However, these researchers measured concentrations of the hormone over a longer time period than used in this study. Consequently, the age of the steers in this study may not have been sufficiently variable to detect differences.

Serum insulin concentrations of breed groups revealed that straightbred Hereford steers had higher concentrations of insulin than the Shorthorn and Gelbvieh-sired crossbred steers. Grigsby and Trenkle (1986) reported similar results in Angus versus Simmental steers.

Insulin is thought to be one of the major regulatory hormones in determining body composition (Prior and Smith, 1982). The importance of insulin in the regulation of growth is made apparent by the effects of diabetes (Romsos et al., 1971). Although insulin is important in normal growth and development of muscle tissue, insulin has its greatest effects on adipose tissue through stimulation of lipogenesis (Prior and Smith, 1982; Weekes, 1986). The differences observed in carcass fat between breed groups may be partially attributed

to differences in insulin concentration.

Of major interest and importance may be the relationship between GH and insulin, as the two hormones are thought to have opposite effects on adipose tissue. Growth hormone is thought to be lipolytic (Eisemann et al.,1986) whereas insulin is generally thought to be lipogenic (Prior and Smith, 1982). How these two hormones interact may influence tissue deposition and ultimately carcass composition in the animal. Elevated concentrations of GH and low insulin concentrations in larger, leaner breeds of cattle may favor increased and more prolonged growth of skeletal muscle rather than shifting energy to adipose tissue. In addition, smaller breeds of cattle have higher insulin concentrations which is associated with increased fat deposition at an earlier age (Trenkle, 1981). Relationships between insulin and GH in this study would generally confirm these observations. Across all slaughter groups, the larger framed, leaner GSH steers had a significantly lower ratio of insulin/GH than other breed groups. However, the small framed, early maturing UH steers did not differ in insulin/GH ratio when compared to SH or SAH steers due to their high concentrations of GH. Although the role of IGF-I in fat deposition is less clear than for GH, one would expect the ratio of insulin:IGF-I to be similar to that of insulin/GH since IGF-I and GH are tightly coupled. Indeed, ranking of breed groups was the same for insulin/GH and insulin/IGF-I.

Serum hormone relationships over time may also explain

compositional changes over the same period. Steers in bleed group 1 had a significantly lower ratio of insulin:GH than those sampled in bleed groups 2 and 3. The correlation between insulin/GH and bleed group was positive ($R = .18$, $P < .05$). Since insulin did not change over time, the higher ratios associated with bleed groups 2 and 3 are a function of lower GH concentrations in each successive bleed group. The ratio of insulin:IGF-I exhibited the same trend. The increase in these ratios over time coincides with a shift away from lean tissue deposition towards fattening as steers across all breed groups became physiologically more mature.

Results of this study indicate a breed group effect on triiodothyronine and thyroxine. Thyroid hormones are primarily involved in the control of metabolic rate and are important in permitting normal growth, as reduced growth is associated with hypothyroidism and hyperthyroidism (Goldberg et al., 1980; Bowen et al., 1987). Optimal bone and muscle growth are dependent on a euthyroid state. Efforts to relate differences in circulating concentrations of thyroid hormones to different cattle types have been unsuccessful, making interpretation of results from this study difficult. Across breed groups, T3 and T4 values appear to be normal for the age and type of cattle evaluated (Davis et al., 1983; Grigsby and Trenkle, 1986). Relationships between thyroid hormones and other hormones, and resulting influence on growth and development need further clarification.

Significant negative correlations existed between GH

concentration and fat thickness, yield grade, marbling score, and percentage carcass fat. These results are consistent with research done by several workers (Purchas et al., 1970, Trenkle, 1970; Purchas et al., 1971; Trenkle and Topel, 1978; Keller et al., 1979; Klindt et al., 1985). In this study, growth hormone was also found to be positively correlated with REA/carcass weight, and estimates of carcass protein and moisture. These results are in agreement with the generally accepted role of GH in stimulating protein synthesis and decreasing the amount of adipose tissue.

Although the effects of GH are thought to be mediated by IGF-I, correlations between IGF-I and carcass characteristics did not reflect these assumptions. Insulin-like growth factor I was found to be correlated with REA/carcass weight, carcass fat, and carcass moisture. These correlations were not as strong as those observed for the same traits when correlated with GH. As many of the anabolic actions of GH on muscle are mediated by IGF-I, one would expect IGF-I to be positively related to carcass protein content. There is little evidence to suggest that GH affects adipose tissue via IGF-I or that IGF-I has a direct effect on adipose tissue. Thus, the negative correlation between IGF-I and carcass fat may not indicate existence of a true relationship.

Correlations reported between insulin and carcass characteristics are opposite those reported for GH. Insulin has been shown to be strongly correlated with carcass fatness (Trenkle and Topel, 1978). In agreement with these findings,

insulin was positively associated with fat thickness, yield grade, marbling score, and carcass fat in this study. Insulin favors lipogenesis and is thought to decrease the breakdown and mobilization of stored fat (Prior and Smith, 1982; Martin et al., 1984; Weekes, 1986). Since insulin has these strong effects on adipose tissue, positive correlations between concentrations of the hormone and carcass fat measurements are likely. Likewise, negative correlations between insulin and carcass protein would be expected given the negative association among the estimates of carcass composition.

Correlations between insulin and IGF-I with carcass characteristics support earlier discussion on the relationships of these hormones and different effects across breed type and time. The theory that the interaction of hormones play a vital role in determining composition are supported by these correlations. Higher ratios of insulin:GH and insulin:IGF-I would favor fattening in relation to protein deposition when estimated on a carcass basis. Indeed, the ratios of insulin to GH and IGF-I were positive with fat thickness, yield grade, marbling score and carcass fat; while being negatively correlated with percentage carcass protein and moisture.

The correlations for insulin:GH and insulin:IGF-I ratios are similar to those reported for insulin concentration alone for the same carcass characteristics. This may imply that insulin concentration has the most effect on determining the compositional traits evaluated. However, insulin ratio

correlations are slightly stronger suggesting that the relationship between insulin and other hormones explains more of the variation in carcass characteristics.

No significant correlations were found between thyroid hormones and carcass characteristics. Similar reports can be found in the literature (Purchas et al., 1971). Although thyroid hormones are undoubtedly involved in the regulation of animal growth, they may have much less of a direct effect when compared to the hormones previously discussed. Thyroid hormones may be more involved in permission of animal growth and also influence production and activity of other hormones. These roles are made evident as maximum growth of various tissues is dependent on a euthyroid state.

Serum hormone concentrations were not significantly correlated with ADG which is in agreement with other studies (Purchas et al., 1970; Irvin and Trenkle, 1970; Etherton and Kensinger, 1984). Attempts were made to relate hormone status to both ADG over the entire trial as well as current ADG in the metabolism room when serum samples were taken. Serum hormone concentrations were related to neither and a high correlation existed between the two measurements of ADG ($R = .84$, $P < .01$). With the exception of the UH steers, little variation in ADG was observed among the cattle, making significant correlations between ADG and hormone concentrations difficult to obtain.

Due to the design of this study, serum hormone concentrations were only measured during a short period in

each steer's growth curve. This small window may or may not be reflective of hormone concentrations from birth to slaughter. If the concentration of hormones early in life sets the stage for rate and composition of growth, measuring hormones later in life may not be related to variables of interest. Therefore, correlations between hormone concentration and their relationships with growth and carcass measurements are difficult to interpret. Since hormones were measured approximately 90 days prior to slaughter, the time at which hormone data were taken may not be most appropriate for drawing conclusions of how these hormones affected carcass composition. This may explain why stronger correlations between GH, IGF-I and insulin and carcass characteristics have been reported in the literature. Despite these complications, the hormone concentrations, relationships, and correlations reported in this study are in general agreement with the roles each hormone is thought to have in influencing growth and development and with what has been previously reported in the literature in similarly designed studies.

CONCLUSIONS

Results from this study provide support for the changes in cattle type that have occurred in the past three decades. These changes in type have been accomplished by intense selection for growth. This study confirms that selection for growth is effective, and that changes in carcass conformation have been primarily associated with slaughtering cattle that are physiologically less mature. Selection for growth has resulted in larger framed, faster growing cattle that are heavier throughout their life span. Decreased carcass fat thickness, marbling scores, and quality grades are realized in the carcass along with increased percentages of carcass protein and moisture and decreased carcass fat.

Differences in growth hormone, IGF-I, and insulin concentrations were noted for biological types and selection for growth in this study. The correlation coefficients calculated in this study indicate relationships between the measured parameters as cattle grow and do not necessarily indicate the specific metabolic functions of any of the hormones. However, the correlations between the measurements of growth, carcass characteristics and hormone concentrations are in general agreement with accepted roles of the hormones in the regulation of growth and development.

Measurements of hormone concentrations over the entire growth curve for the diverse population of cattle utilized in this study may provide a clearer understanding of the effects of biological type and selection on the endocrine system. In this study, the relationships reported support our previous understanding of how these hormones and their relationships interact with growth, carcass traits, and measures of carcass composition.

Growth and development of meat animals is a complex process. This process is under the influence of hormones and one hormone may have multiple actions while one function is likely under the control of multiple hormones. For these reasons, relating one hormone to a specific growth or carcass trait may be over-simplified. Further research is needed to determine the precise functions of individual hormones and how these hormones interact in the regulation and control of tissue growth and development, and ultimately carcass composition.

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APPENDIX

APPENDIX

List Of Abbreviations Used In Appendix

ADG = average daily gain over entire feeding period, kg

ADJ BF = adjusted 12th rib fat thickness, mm

AND = animal days

BEG WT = initial weight, kg

BEG AGE = initial age, d

BF = 12th rib fat thickness, mm

BG = breed group; 1 = UH, 2 = SH, 3 = SAH, 4 = GSH

BL AGE = age at blood collection, d

DMI = dry matter intake, kg

FIN WT = final weight, kg

FS = frame score

GH = growth hormone, ng/ml

GH BS LN = baseline GH, ng/ml

GH AUC = GH area under curve, ng x min/ml

GH PK INT = GH inter-peak interval (time between peaks), min

GH PK LN = GH peak length, min

GH PK AMP = GH peak amplitude, ng/ml

GH PK NO = GH peak number

GH PK FREQ = GH peak frequency, peaks/min

HCW = hot carcass weight

IGF-I = insulin-like growth factor I, ng/ml

List Of Abbreviations Used In Appendix (cont'd.)

INS = insulin, μU/ml

KPH = kidney, pelvic and heart fat, %

MR IADG = average daily gain for entire feeding period prior to blood collection

MR ADG = metabolism room average daily gain, kg

MR WT = metabolism room weight, kg

MR ADFI = metabolism room average daily feed intake, kg dry matter

MS = marbling score; 400 = Slight 0, 500 = Small 0

NO = individual steer identification number

PN = Beef Cattle Research Center pen number

QG = quality grade; 11 = high Select, 12 = low Choice

REA = ribeye area, cm^2

RIB H2O = 9-10-11 rib moisture, %

RIB BONE = 9-10-11 rib bone, %

RIB PROT = 9-10-11 rib protein, %

RIB EE = 9-10-11 rib ether extract, %

SG = slaughter group

SL AGE = slaughter age, d

T3 = triiodothyronine, ng/ml

T4 = thyroxine, ng/ml

WW = weaning weight, kg

YG = yield grade

Table 22. Individual performance and carcass characteristics of Lake City steers born in 1989

NO	BG	SG	PN	WW	BEG AGE	BEG WT	FIN WT	ADG	FS	SL AGE	HCW	BF	ADJ BF	REA	KPH	YG	MS	QG	RIB H2O	RIB EE	RIB PROT	RIB BONE
101	2	3	60	267	237	256	594	1.275	4.78	503	376.9	12.7	19.1	80.6	1.5	3.83	450	10	39.87	48.01	12.10	15.21
102	1	2	74	151	236	141	342	0.801	0.76	488	210.0	16.5	17.8	67.7	2.5	3.15	490	11	45.84	40.70	13.28	16.33
103	1	1	66	163	235	157	367	0.913	1.02	466	218.6	12.7	17.8	62.6	3.5	3.68	510	12	41.35	45.44	12.90	14.01
105	4	1	62	260	232	256	536	1.219	5.20	463	341.1	10.2	10.2	89.7	3.0	2.51	500	12	50.61	34.35	14.11	16.07
106	3	1	56	251	232	244	514	1.173	6.08	463	314.8	3.8	5.1	82.6	1.5	1.84	490	11	50.35	34.15	14.27	17.60
107	2	1	68	195	227	192	502	1.347	3.99	458	308.4	14.0	17.8	74.2	2.5	3.65	500	12	46.90	39.05	13.59	14.37
108	4	3	70	285	224	281	635	1.335	5.28	490	394.6	7.6	7.6	94.8	2.5	2.35	400	10	53.60	30.99	13.41	16.19
110	4	2	58	276	223	267	587	1.274	6.29	475	361.1	8.9	8.9	100.0	2.5	1.94	500	12	50.29	34.01	13.66	16.07
111	1	3	52	161	222	152	478	1.229	1.27	488	296.2	17.8	19.1	79.4	3.0	3.52	710	14	41.36	47.12	10.50	13.18
113	3	2	64	317	220	300	622	1.285	5.57	472	406.9	14.0	17.8	80.6	3.5	4.36	570	12	41.84	45.87	15.72	13.79
114	3	3	72	242	220	247	555	1.164	5.44	486	345.6	12.7	12.7	88.4	2.5	2.76	600	13	46.47	40.11	12.60	16.78
117	2	2	54	185	219	180	523	1.370	4.57	471	322.5	11.4	12.7	79.4	2.0	2.92	440	10	46.34	40.20	13.82	16.10
118	3	3	72	265	219	257	495	0.900	5.70	485	311.6	5.1	7.6	77.4	2.5	2.52	700	14	45.64	42.61	11.50	18.38
119	2	2	54	201	218	197	518	1.279	4.83	470	318.4	6.4	8.9	80.6	1.5	2.34	420	10	49.60	36.41	12.38	15.40
120	1	3	52	161	218	156	432	1.041	1.19	484	265.8	20.3	20.3	70.3	3.0	3.84	590	12	42.48	46.27	10.53	12.49
126	2	1	68	251	215	241	508	1.160	4.61	446	314.3	7.6	11.4	71.0	2.0	3.14	340	9	44.32	39.34	14.86	14.63
128	4	3	70	312	215	299	665	1.383	7.25	481	403.7	3.8	5.1	100.6	1.5	1.69	520	12	53.63	30.48	14.70	15.82
129	3	2	64	244	215	242	555	1.247	6.12	467	342.5	6.4	7.6	83.2	2.5	2.49	480	11	49.38	34.55	13.43	16.93
130	2	3	60	265	214	252	518	1.003	4.87	480	323.0	15.2	16.5	78.1	2.0	3.36	500	12	45.23	41.05	12.46	15.03
133	2	1	68	206	212	199	492	1.272	4.64	443	299.4	10.2	12.7	63.9	2.0	3.49	440	10	43.90	44.55	12.73	15.56
134	4	1	62	290	211	277	539	1.140	4.15	442	338.4	10.2	10.2	94.2	2.0	2.06	580	12	45.39	40.70	12.09	13.37
135	2	3	60	242	211	234	630	1.494	4.65	477	393.7	20.3	22.9	81.3	2.0	4.42	510	12	38.60	50.51	10.36	14.43
136	4	2	58	317	211	306	684	1.505	6.28	463	423.2	12.7	19.1	95.5	3.5	3.88	460	11	42.43	44.36	12.86	13.39
137	4	3	70	283	211	268	629	1.363	7.29	477	386.9	5.1	5.1	105.8	2.5	1.49	490	11	51.62	33.26	15.17	16.56
140	3	2	64	265	210	256	530	1.093	5.54	462	336.6	7.6	10.2	91.6	2.5	2.28	500	12	47.98	37.63	13.92	14.70
141	3	3	72	278	209	270	636	1.380	6.68	475	409.6	7.6	8.9	90.3	3.0	2.93	540	12	45.14	42.79	12.35	15.34
143	4	1	62	265	218	254	550	1.288	5.59	449	345.6	6.4	7.6	93.5	2.5	2.01	490	11	53.60	29.20	15.48	16.13
144	2	2	54	195	208	189	512	1.289	4.43	460	311.6	7.6	12.7	79.4	2.5	2.92	490	11	47.69	39.06	13.84	15.06
145	3	1	56	208	208	201	528	1.420	4.68	439	325.2	11.4	12.7	80.0	2.5	3.01	500	12	44.82	40.68	14.36	15.58
146	2	3	60	299	208	288	662	1.412	5.43	474	415.9	7.6	8.9	87.7	1.5	2.81	400	10	45.86	41.36	12.07	13.21

Table 22 (cont'd.).

NO	BG	SG	PN	WW	BEG AGE	BEG WT	FIN WT	ADG	FS	SL AGE	HCW	BF	ADJ BF	REA	KPH	YG	MS	QG	RIB H2O	RIB EE	RIB PROT	RIB BONE
147	3	1	56	258	208	248	557	1.347	6.94	439	344.7	5.1	7.6	81.9	2.5	2.57	490	12	48.03	36.51	14.66	16.13
148	1	2	74	131	207	124	361	0.945	1.43	459	206.4	8.9	8.9	65.8	3.5	2.54	420	10	48.26	36.74	14.33	13.63
149	3	2	64	258	206	244	543	1.189	5.96	458	336.1	7.6	7.6	72.9	2.5	2.95	500	12	48.69	36.01	15.87	17.35
151	1	1	66	172	205	163	401	1.035	1.45	436	248.1	8.9	12.7	64.5	2.5	3.13	510	12	42.05	47.51	10.72	15.02
152	3	3	72	303	205	293	570	1.046	5.97	471	355.6	10.2	12.7	74.2	3.0	3.65	650	13	43.35	43.32	12.70	16.16
156	3	2	64	294	204	283	666	1.527	7.48	456	414.1	7.6	11.4	90.3	2.5	3.11	500	12	46.50	38.85	16.58	16.53
159	3	1	56	231	203	226	541	1.367	5.49	434	329.8	8.9	10.2	80.6	2.5	2.76	510	12	48.69	35.63	14.22	15.44
160	4	1	62	288	202	277	554	1.205	7.00	433	346.5	10.2	10.2	88.4	3.0	2.62	500	12	51.12	33.15	14.37	16.67
161	2	2	54	222	201	219	532	1.243	4.25	453	321.6	15.2	15.2	82.6	2.5	3.10	470	11	45.62	40.40	11.60	13.55
162	3	2	64	301	201	292	593	1.198	6.26	453	365.6	10.2	12.7	83.2	3.0	3.28	460	11	47.56	38.59	14.24	14.87
163	3	3	72	326	200	319	691	1.404	6.52	466	448.6	7.6	8.9	102.6	2.5	2.55	490	11	46.57	40.20	13.06	13.36
164	1	3	52	161	200	152	420	1.012	0.62	466	263.5	17.8	20.3	70.3	2.0	3.62	540	12	44.92	43.00	11.60	13.88
165	2	2	54	219	199	210	554	1.370	5.53	451	342.0	12.7	15.2	67.7	1.5	3.81	510	12	43.32	44.85	12.14	13.71
166	2	1	68	242	199	235	525	1.258	4.27	430	323.0	12.7	17.8	81.3	2.5	3.42	590	12	42.44	45.25	11.95	15.42
167	2	3	60	208	198	201	511	1.169	5.41	464	318.4	10.2	14.0	72.3	2.0	3.36	440	10	43.30	44.58	12.03	16.43
169	2	2	54	185	198	177	479	1.204	4.28	450	290.8	10.2	15.2	72.3	2.5	3.35	540	12	44.83	41.11	12.48	13.70
170	3	1	56	260	197	245	541	1.284	6.68	428	347.0	3.8	10.2	85.8	3.0	2.75	510	12	43.87	43.86	11.65	13.53
172	4	3	70	335	196	319	666	1.309	6.31	462	419.6	7.6	7.6	122.6	1.5	0.98	430	10	56.86	26.32	15.79	14.81
173	3	3	72	235	196	236	570	1.260	6.56	462	356.1	7.6	8.9	83.9	2.5	2.70	520	12	45.75	42.01	12.22	16.91
174	4	2	58	260	196	239	602	1.448	7.44	448	364.2	7.6	7.6	94.2	3.0	2.23	470	11	48.40	37.63	12.97	14.61
176	4	3	70	310	194	293	665	1.405	6.33	460	406.4	10.2	10.2	87.1	2.0	2.98	600	13	50.99	33.99	14.03	17.42
177	3	2	64	242	193	231	511	1.117	5.09	445	317.1	5.1	8.9	78.7	2.0	2.53	520	12	48.48	35.79	13.78	16.80
178	1	2	74	165	192	156	430	1.090	2.09	444	264.9	10.2	16.5	69.0	3.0	3.52	590	12	40.07	47.63	10.69	13.16
179	3	1	56	251	191	245	548	1.317	4.74	422	337.5	12.7	12.7	76.8	2.0	3.17	480	11	49.81	35.34	15.26	13.84
182	2	1	68	210	189	200	495	1.284	4.76	420	308.0	10.2	12.7	73.5	1.5	2.98	520	12	45.98	40.93	13.20	16.91
183	4	2	58	210	189	201	475	1.090	6.26	441	296.2	7.6	7.6	78.7	2.5	2.33	520	12	54.82	29.55	13.73	16.54
184	4	1	62	244	188	233	514	1.223	5.65	419	321.6	8.9	14.0	69.0	2.5	3.65	500	12	47.40	37.50	13.51	17.49

Table 22 (cont'd.).

NO	BG	SG	PN	WW	BEG AGE	BEG WT	FIN WT	ADG	FS	SL AGE	HCW	BF	ADJ BF	REA	KPH	YG	MS	QG	RIB H2O	RIB EE	RIB PROT	RIB BONE
185	1	2	74	140	188	133	405	1.084	2.14	440	244.9	14.0	19.1	66.5	3.0	3.73	600	13	40.64	47.64	10.58	11.62
186	2	3	60	190	188	191	557	1.383	5.27	454	333.4	12.7	12.7	74.2	2.0	3.26	600	13	46.58	39.63	12.53	16.13
188	4	2	58	276	186	265	591	1.298	6.80	438	363.3	7.6	10.2	100.6	3.0	2.15	500	12	46.80	39.55	12.95	15.85
189	1	1	66	158	186	145	381	1.026	1.41	417	231.3	10.2	15.2	69.0	3.0	3.11	470	11	46.57	39.88	12.70	14.30
190	2	2	54	201	184	198	489	1.162	4.94	436	298.0	10.2	15.2	69.0	2.0	3.47	500	12	44.67	41.66	12.52	18.11
191	1	3	52	165	184	156	408	0.950	1.93	450	255.4	16.5	17.8	54.8	2.5	4.17	780	14	38.70	51.06	10.16	13.84
192	1	1	66	176	181	164	383	0.953	1.84	412	236.8	12.7	20.3	61.3	3.5	4.14	510	12	40.84	45.46	11.91	12.92
193	4	3	70	278	177	265	614	1.318	6.15	443	373.3	6.4	6.4	80.0	2.0	2.68	410	10	50.76	34.78	13.65	18.47
195	1	3	52	156	177	147	380	0.878	1.89	443	231.8	15.2	15.2	58.7	3.0	3.63	540	12	43.21	44.39	11.96	12.58
196	2	1	68	210	176	206	508	1.311	5.66	407	316.6	3.8	5.1	75.5	1.5	2.21	450	10	50.06	34.64	13.79	16.63
197	4	1	62	276	176	259	539	1.219	5.78	407	341.1	12.7	12.7	81.3	2.5	3.08	500	12	51.23	33.48	14.10	15.93
198	4	3	70	288	176	273	600	1.234	6.53	442	376.9	8.9	8.9	87.1	2.5	2.71	470	11	51.30	33.62	14.58	15.28
199	1	2	74	140	176	127	378	0.999	1.27	428	233.1	14.0	16.5	69.7	3.0	3.22	520	12	47.50	38.38	12.60	13.42
201	2	3	60	195	173	191	576	1.452	5.32	439	345.6	7.6	10.2	75.5	2.5	3.15	580	12	44.59	43.17	11.52	15.77
203	2	1	68	181	168	174	513	1.475	5.12	399	311.6	7.6	8.9	76.1	3.0	2.81	520	12	48.18	37.75	13.01	17.18
204	4	3	70	238	167	225	544	1.203	5.76	433	346.1	7.6	7.6	99.4	2.5	1.72	460	11	53.70	30.87	14.56	17.27
205	3	3	72	215	167	208	485	1.044	5.39	433	308.4	8.9	10.2	74.2	3.5	3.10	480	11	44.17	43.81	11.18	15.91
206	2	3	60	226	167	225	566	1.285	5.89	433	343.4	17.8	17.8	74.8	1.5	3.71	520	12	42.99	45.65	11.71	14.29
207	1	1	66	167	166	159	424	1.150	2.89	397	255.8	6.4	10.2	69.7	2.0	2.59	530	12	46.46	37.62	13.96	13.54
208	3	1	56	224	166	213	489	1.203	5.52	397	301.2	8.9	11.4	76.1	3.0	2.97	455	11	46.15	39.29	12.40	17.60
209	4	2	58	263	166	256	584	1.307	7.53	418	362.9	3.8	3.8	102.6	2.5	1.33	460	11	56.54	25.75	18.15	16.51
210	4	1	62	240	166	234	464	0.998	5.90	397	290.3	5.1	5.1	74.2	1.5	2.05	380	9	56.68	25.74	16.12	20.06
211	4	2	58	254	166	239	562	1.289	6.90	418	342.0	7.6	7.6	81.9	1.5	2.35	480	11	50.29	34.69	16.66	15.37
213	2	2	54	151	159	149	529	1.514	4.98	411	316.6	10.2	10.2	71.0	1.0	2.83	495	11	45.04	40.81	12.10	16.06
214	4	1	62	233	157	222	487	1.154	5.88	388	305.7	5.1	8.9	69.7	3.0	3.08	470	11	48.16	36.60	13.65	15.52

Table 23. Individual performance and carcass characteristics of Lake City steers born in 1990

NO	BG	SG	PN	WW	BEG AGE	BEG WT	FIN WT	ADG	FS	SL AGE	HCW	BF	ADJ BF	REA	KPH	YG	MS	QG	RIB H2O	RIB EE	RIB PROT	RIB BONE
60	2	1	80	179	150	185	477	1.326	6.25	397	298.0	5.1	6.4	73.5	2.0	2.37	500	12	52.91	31.53	13.80	16.66
85	3	3	64	188	200	209	509	1.176	6.72	482	316.2	3.8	6.4	81.9	2.0	2.11	500	12	47.02	39.89	12.51	19.11
101	3	1	76	224	227	229	513	1.291	4.08	474	311.6	12.7	15.2	77.4	3.5	3.47	530	12	44.64	42.04	12.75	15.39
102	3	2	58	272	223	288	625	1.387	5.89	493	391.9	11.4	14.0	98.1	3.0	2.89	770	14	48.83	36.29	14.38	15.21
104	1	1	52	145	217	156	377	1.006	1.65	464	224.5	6.4	7.6	60.6	2.5	2.62	600	13	49.13	36.96	12.85	14.95
106	1	2	78	163	213	174	324	0.616	1.06	483	202.3	7.6	14.0	63.9	2.5	2.90	760	14	45.43	41.65	13.29	17.62
109	1	3	60	229	211	236	528	1.144	3.35	493	317.5	20.3	21.6	77.4	2.5	3.95	470	11	41.23	47.73	10.10	13.66
110	2	1	80	215	210	226	557	1.501	5.74	457	344.3	15.2	16.5	74.2	1.5	3.63	520	12	46.42	39.73	11.86	16.03
111	3	3	64	281	210	291	641	1.375	6.88	492	399.6	8.9	11.4	85.8	2.5	3.22	480	11	45.31	41.38	12.56	16.14
113	3	3	64	335	209	356	635	1.094	7.14	491	401.9	10.2	8.9	69.7	3.5	3.99	570	12	42.73	46.06	11.06	16.14
114	3	1	76	315	207	333	647	1.429	6.02	454	401.0	10.2	11.4	78.7	2.5	3.58	455	11	48.08	37.17	12.02	17.70
115	1	3	60	233	207	239	527	1.131	2.63	489	329.3	14.0	16.5	87.7	3.5	3.23	500	12	42.38	45.95	11.52	14.01
116	1	2	78	154	207	164	449	1.172	1.87	477	275.8	25.4	27.9	74.8	2.5	4.35	700	14	41.06	46.03	12.30	12.68
117	4	2	66	249	207	269	584	1.294	5.65	477	367.0	10.2	8.9	85.8	2.5	2.69	580	12	49.42	36.10	14.65	15.34
120	3	2	58	229	205	257	562	1.256	6.17	475	351.1	12.7	12.7	79.4	3.0	3.36	600	13	46.11	39.52	15.12	14.43
122	3	2	58	319	203	337	666	1.353	7.19	473	415.9	12.7	12.7	80.0	3.0	3.87	590	12	46.88	39.43	14.22	16.02
123	3	1	76	254	203	263	532	1.223	5.43	450	329.3	6.4	8.9	74.2	3.0	3.05	640	13	45.30	41.47	10.36	14.24
124	4	3	56	292	202	310	568	1.010	5.95	484	355.2	12.7	16.5	81.3	3.0	3.67	560	12	45.23	41.07	12.95	16.02
125	4	1	74	308	201	318	605	1.305	6.20	448	387.8	3.8	3.8	94.2	2.5	1.95	500	12	50.40	33.65	15.26	15.25
127	4	3	56	265	200	283	618	1.313	7.22	482	369.7	5.1	6.4	74.8	3.0	3.11	560	12	49.64	35.50	13.82	15.77
131	4	1	74	301	199	317	600	1.285	7.61	446	378.8	3.8	5.1	94.8	2.0	1.87	470	11	56.90	25.58	16.75	14.80
134	3	3	64	267	199	282	596	1.233	4.97	481	381.0	15.2	16.5	94.2	3.5	3.35	490	11	43.24	44.46	12.11	15.07
135	4	2	66	299	198	317	664	1.428	5.86	468	431.8	10.2	11.4	122.6	2.5	1.66	500	12	51.07	33.61	14.96	14.64
136	2	3	62	208	198	226	566	1.334	4.47	480	353.4	15.2	17.8	74.2	2.0	3.93	600	13	41.74	46.20	12.48	14.55
137	4	3	56	328	198	333	661	1.286	6.86	480	425.5	8.9	14.0	87.1	3.0	3.72	550	12	40.55	48.50	11.27	15.41
138	1	1	52	140	198	148	402	1.157	1.46	445	241.3	10.2	14.0	61.9	3.5	3.52	500	12	47.59	38.28	12.51	14.69
141	4	1	74	276	196	285	564	1.268	6.13	443	355.2	5.1	7.6	78.7	2.5	2.82	490	11	51.57	32.91	13.98	20.56

Table 23 (cont'd.).

NO	BG	SG	PN	WW	BEG AGE	BEG WT	FIN WT	ADG	FS	SL AGE	HCW	BF	ADJ BF	REA	KPH	YG	MS	QG	RIB H2O	RIB EE	RIB PROT	RIB BONE
143	1	2	78	161	196	170	446	1.139	2.86	466	268.1	10.2	15.2	71.0	2.5	3.23	600	13	42.99	44.53	12.86	14.19
144	3	1	76	258	195	267	482	0.975	6.64	442	300.7	6.4	7.6	66.5	1.5	2.77	460	11	50.57	34.73	12.78	18.72
145	3	1	76	242	194	260	570	1.408	6.40	441	342.9	8.9	8.9	71.0	2.0	3.13	480	11	48.97	35.73	14.12	15.89
146	4	3	56	267	194	298	603	1.197	6.02	476	381.0	2.5	5.1	100.0	3.0	1.83	560	12	50.18	34.79	14.86	16.88
148	3	2	58	244	194	254	564	1.275	6.27	464	345.2	5.1	6.4	80.6	2.0	2.42	580	12	50.26	33.53	15.20	18.81
149	4	2	66	299	193	317	647	1.359	5.78	463	407.3	8.9	8.9	96.1	2.5	2.52	610	13	47.67	37.82	14.13	16.43
150	1	3	60	145	192	152	442	1.135	0.89	474	261.3	10.2	14.0	71.0	3.0	3.14	490	11	45.08	42.45	12.64	14.78
152	1	3	60	136	191	142	376	0.916	0.77	473	225.4	10.2	12.7	66.5	3.0	2.94	490	11	45.25	41.78	13.88	14.46
153	2	3	62	226	191	247	498	0.984	4.42	473	308.4	8.9	10.2	67.1	2.5	3.26	500	12	43.00	44.54	12.13	17.87
154	3	2	58	265	190	282	600	1.309	7.44	460	364.2	6.4	6.4	80.0	1.5	2.51	570	12	50.34	34.01	15.20	18.58
155	1	2	78	149	189	161	453	1.204	2.42	459	272.2	11.4	14.0	73.5	2.5	3.01	540	12	44.52	41.88	14.07	14.73
156	2	2	54	229	189	249	554	1.256	4.56	459	339.7	12.7	15.2	74.2	2.0	3.57	599	12	48.79	36.28	15.18	17.86
157	2	2	54	181	188	205	521	1.303	4.57	458	322.5	15.2	16.5	74.2	2.5	3.65	600	13	45.23	41.09	13.28	14.85
159	4	3	56	276	188	295	579	1.114	6.58	470	345.2	7.6	10.2	81.3	2.5	2.86	680	13	46.03	40.58	12.61	16.27
160	4	1	74	274	187	288	571	1.285	6.47	434	369.7	5.1	5.1	92.9	1.5	1.79	490	11	54.23	28.46	15.64	17.52
161	3	3	64	260	187	284	551	1.046	5.96	469	340.2	10.2	12.7	80.6	2.5	3.10	520	12	46.36	40.00	13.68	16.66
163	4	2	66	267	185	279	561	1.159	5.10	455	348.4	5.1	6.4	85.2	2.5	2.32	490	11	51.10	32.73	15.96	17.77
164	3	3	64	224	185	240	532	1.147	6.36	467	325.2	5.1	6.4	89.7	2.5	1.90	455	11	51.92	32.92	15.17	19.12
165	4	2	66	263	185	278	588	1.275	6.23	455	370.6	5.1	6.4	109.0	2.5	1.32	530	12	49.91	34.75	15.44	16.36
166	1	1	52	129	180	136	358	1.010	1.52	427	220.0	11.4	14.0	68.4	3.0	2.93	500	12	48.21	37.86	13.26	17.70
167	4	1	74	278	180	291	569	1.264	6.79	427	352.0	2.5	3.8	84.5	2.5	2.13	450	10	53.76	30.18	14.93	14.20
168	1	3	60	131	176	138	443	1.197	2.56	458	258.1	12.7	12.7	61.3	2.5	3.37	600	13	39.25	49.90	13.05	13.27
170	3	1	76	258	175	274	564	1.320	6.35	422	343.8	8.9	11.4	78.1	3.5	3.33	450	10	46.85	38.83	12.34	14.12
171	1	2	78	111	173	120	322	0.831	0.58	443	186.4	7.6	8.9	55.5	2.5	2.68	455	11	47.75	38.06	13.02	21.17
172	2	1	80	215	172	227	511	1.291	6.00	419	313.4	3.8	6.4	77.4	2.0	2.31	500	12	50.48	34.25	13.07	15.26
173	1	1	52	131	172	147	373	1.027	1.24	419	228.2	14.0	15.2	67.7	3.0	3.15	490	11	48.76	37.11	14.01	14.77
176	3	1	76	201	169	208	443	1.068	5.16	416	267.6	8.9	8.9	67.1	3.0	2.89	530	12	49.67	35.21	14.24	16.16

Table 23 (cont'd.).

NO	BG	SG	PN	WW	BEG AGE	BEG WT	FIN WT	ADG	FS	SL AGE	HCW	BF	ADJ BF	REA	KPH	YG	MS	QG	RIB H2O	RIB EE	RIB PROT	RIB BONE
177	2	2	54	181	169	197	568	1.527	4.76	439	330.2	6.4	7.6	87.7	2.0	2.06	455	11	48.26	37.43	13.37	16.37
178	4	1	74	260	168	270	576	1.388	7.42	415	357.0	8.9	8.9	88.4	2.0	2.38	460	11	52.18	32.28	14.63	16.66
179	3	2	58	222	167	238	517	1.148	6.04	437	326.1	6.4	7.6	80.6	1.5	2.28	499	11	50.09	34.86	13.86	19.47
181	2	3	62	174	165	186	545	1.407	5.43	447	317.1	8.9	8.9	83.9	2.0	2.27	470	11	50.82	34.76	13.44	17.64
182	4	3	56	278	165	297	551	0.996	6.69	447	342.5	3.8	5.1	80.0	2.0	2.30	500	12	49.28	37.29	13.17	17.02
184	2	3	62	183	164	195	550	1.389	5.32	446	323.9	7.6	7.6	85.8	2.0	2.11	500	12	49.90	36.26	14.67	18.71
185	3	3	64	242	164	257	603	1.357	5.82	446	376.0	10.2	14.0	82.6	3.5	3.63	490	12	40.83	48.42	11.13	14.69
186	2	1	80	192	164	205	487	1.285	6.72	411	304.4	7.6	8.9	74.2	2.5	2.74	500	12	50.20	34.61	14.36	14.57
187	2	1	80	163	163	175	488	1.425	5.85	410	291.2	7.6	7.6	71.0	2.0	2.57	540	12	49.55	35.62	13.17	15.41
188	2	2	54	215	163	229	551	1.322	6.21	433	336.1	17.8	17.8	79.4	2.0	3.53	600	13	42.65	44.13	11.88	14.63
189	2	1	80	188	161	207	532	1.476	6.50	408	321.6	6.4	7.6	81.3	2.5	2.41	510	12	51.88	32.64	14.64	16.94
190	4	1	74	244	159	261	542	1.274	7.78	406	332.5	5.1	6.4	86.5	2.0	2.02	480	11	53.16	32.14	15.14	18.95
191	3	3	64	238	159	256	595	1.331	6.25	441	375.1	8.9	10.2	82.6	2.5	3.05	490	11	46.65	40.63	13.61	16.32
192	3	1	76	213	158	227	483	1.161	5.41	405	304.8	12.7	15.2	76.1	2.5	3.28	500	12	46.94	39.01	13.35	14.10
193	2	3	62	235	158	245	588	1.345	5.76	440	362.0	3.8	6.4	91.6	1.5	1.91	520	12	50.27	34.98	14.48	18.02
194	3	2	58	226	157	248	508	1.070	5.65	427	305.3	8.9	8.9	78.7	2.0	2.43	470	11	50.25	34.10	15.14	19.09
195	2	3	62	158	157	171	557	1.517	6.15	439	324.8	2.5	5.1	73.5	2.0	2.47	500	12	52.34	32.64	14.66	18.16
196	2	1	80	181	156	197	464	1.216	5.05	403	288.0	10.2	10.2	76.1	2.5	2.64	520	12	50.43	34.68	12.91	15.12
197	2	2	54	167	151	191	552	1.486	5.71	421	327.0	7.6	7.6	79.4	1.5	2.35	460	11	48.59	36.45	14.02	17.65
199	2	2	54	190	150	213	517	1.251	6.73	420	314.3	10.2	10.2	78.1	2.0	2.66	460	11	47.36	38.17	13.23	16.29
200	4	3	56	213	149	224	498	1.076	5.99	431	299.4	5.1	5.1	80.6	2.0	1.91	500	12	54.94	28.68	15.24	20.55
201	2	3	62	156	150	157	478	1.259	5.73	432	281.2	10.2	10.2	71.0	2.5	2.84	520	12	52.44	32.42	14.78	16.76
9085	2	1	80	188	162	204	581	1.717	7.12	409	359.2	6.4	6.4	86.5	2.0	2.25	430	10	53.48	30.92	12.31	15.82

Table 24. Individual hormone parameters of Lake City steers born in 1989

						GH										
NO	BG	SG	PN	BL AGE	GH	BS LN	PK NO	PK AMP	PK LN	PK FREQ	PK INT	AUC	IGF-I	INS	T3	T4
101	2	3	60	438	3.04	2.72	1	5.04	30	0.0021	0	1471	486	22.60	2.330	89.27
102	1	2	74	402	4.32	4.02	2	3.39	30	0.0042	180	2069	428	30.81	2.213	94.17
103	1	1	66	373	4.44	2.94	5	4.31	36	0.0104	112	2172	814	22.07	2.154	92.92
105	4	1	62	370	4.05	2.58	3	6.16	70	0.0062	165	1982	707	29.62	1.881	93.86
106	3	1	56	370	5.22	3.39	2	12.1	60	0.0042	210	2581	813	23.61	2.135	89.58
107	2	1	68	365	8.14	5.30	3	13.2	60	0.0062	210	3876	900	29.04	1.816	85.93
108	4	3	70	425	3.60	3.19	2	4.01	45	0.0042	270	1674	985	25.07	1.907	99.61
110	4	2	58	389	2.71	2.45	1	3.09	60	0.0021	0	1312	806	13.76	2.252	83.52
111	1	3	52	423	3.12	2.88	1	3.65	30	0.0021	0	1489	859	39.81	1.595	82.20
113	3	2	64	386	3.44	3.34	1	1.77	30	0.0021	0	1650	615	34.44	1.966	93.62
114	3	3	72	421	2.75	2.49	2	1.51	45	0.0042	150	1323	511	33.68	2.402	88.26
117	2	2	54	385	4.28	3.31	1	10.9	60	0.0021	0	2126	860	46.94	2.363	80.18
118	3	3	72	420	3.10	3.01	1	1.68	30	0.0021	0	1497	504	25.52	1.731	71.56
119	2	2	54	384	2.76	1.90	2	6.41	45	0.0042	120	1342	620	27.39	1.835	95.64
120	1	3	52	419	2.83	2.72	1	1.81	60	0.0021	0	985	748	31.89	2.037	105.5
126	2	1	68	353	4.45	3.64	1	10.5	90	0.0021	0	2178	880	28.47	1.614	62.16
128	4	3	70	416	3.96	2.45	3	7.11	60	0.0062	180	1960	562	21.97	1.894	86.09
129	3	2	64	381	3.24	2.64	2	4.41	45	0.0042	240	1562	524	20.64	2.154	78.71
130	2	3	60	415	3.26	2.76	3	2.63	30	0.0062	210	1571	655	31.90	2.207	115.9
133	2	1	68	350	4.17	3.20	2	5.24	60	0.0044	90	1803	910	17.20	2.369	89.74
134	4	1	62	349	4.20	3.05	2	7.62	60	0.0042	90	2070	1157	27.15	2.884	103.9
135	2	3	60	412	3.11	2.46	2	4.26	45	0.0042	210	1505	598	41.54	2.395	101.4
136	4	2	58	377	4.40	2.80	3	6.79	70	0.0062	180	2133	978	27.52	2.604	98.98
137	4	3	70	412	3.55	3.49	1	5.00	30	0.0021	0	1724	575	37.46	1.809	83.83
140	3	2	64	376	2.86	2.50	1	6.38	30	0.0021	0	1390	609	52.27	1.933	94.63
141	3	3	72	410	3.43	2.83	2	3.41	60	0.0042	270	1677	423	17.73	2.174	84.69

Table 24 (cont'd.).

NO	BG	SG	PN	BL AGE	BL GH	GH BS LN	GH PK NO	GH PK AMP	GH PK LN	GH PK FREQ	GH PK INT	GH AUC	IGF-I	INS	T3	T4
143	4	1	62	356	4.04	4.34	1	4.27	30	0.0021	0	1955	1056	27.82	2.324	87.33
144	2	2	54	374	3.17	2.57	2	4.70	30	0.0042	210	1558	810	64.66	2.128	107.6
145	3	1	56	346	3.13	2.54	2	3.70	60	0.0042	330	1506	971	24.99	2.194	88.88
146	2	3	60	409	5.73	4.21	1	24.4	30	0.0021	0	2836	885	32.14	1.861	94.63
147	3	1	56	346	4.37	3.36	4	3.71	45	0.0083	120	2075	811	21.53	1.966	93.93
148	1	2	74	373	6.71	2.67	3	17.4	70	0.0062	150	3297	812	31.75	2.291	98.75
149	3	2	64	372	2.57	2.50	1	2.76	30	0.0021	0	1228	632	17.75	2.350	98.05
151	1	1	66	343	3.65	2.53	2	5.73	45	0.0042	120	1810	1018	20.97	2.649	108.5
152	3	3	72	406	4.18	3.85	1	5.45	30	0.0021	0	2038	583	23.38	1.790	94.48
156	3	2	64	370	4.11	3.30	3	4.92	30	0.0062	165	1947	917	33.21	2.519	114.7
159	3	1	56	341	4.52	4.21	2	2.87	30	0.0042	420	2132	685	13.61	1.686	118.6
160	4	1	62	340	5.37	4.24	2	9.90	30	0.0042	360	2399	934	22.63	1.783	80.03
161	2	2	54	367	3.47	2.22	1	12.6	90	0.0021	0	1706	684	28.4	2.395	100.3
162	3	2	64	367	2.69	2.64	0	0	0	0	0	1302	558	34.37	2.526	105.2
163	3	3	72	401	3.40	3.27	1	1.54	30	0.0021	0	1650	604	37.33	2.167	96.42
164	1	3	52	401	2.63	2.29	2	2.68	45	0.0042	90	1264	632	13.27	2.005	95.33
165	2	2	54	365	3.27	3.00	1	2.33	90	0.0021	0	1578	718	29.57	2.467	99.76
166	2	1	68	337	3.30	2.98	2	2.77	30	0.0042	360	1540	811	32.15	2.044	96.81
167	2	3	60	399	3.00	2.87	1	1.38	60	0.0021	0	1435	798	24.72	2.102	85.31
169	2	2	54	364	3.56	3.62	1	2.43	30	0.0021	0	1660	838	33.90	2.324	88.57
170	3	1	56	335	3.92	3.73	1	2.46	30	0.0021	0	1853	675	33.29	1.634	107.3
172	4	3	70	397	2.43	2.17	1	4.60	30	0.0021	0	1164	560	13.73	2.252	76.61
173	3	3	72	397	3.10	2.66	2	3.49	45	0.0042	90	1509	780	14.24	2.259	89.43
174	4	2	58	362	3.16	2.80	1	4.02	60	0.0021	0	1525	924	40.13	2.148	77.23
176	4	3	70	395	3.67	3.39	2	3.11	30	0.0042	270	1722	798	22.80	2.096	85.15
177	3	2	64	359	4.49	3.40	1	15.4	60	0.0021	0	2196	763	20.73	2.597	99.76

Table 24 (cont'd.).

						GH										
NO	BG	SG	PN	BL AGE	GH	BS LN	PK NO	PK AMP	PK LN	PK FREQ	PK INT	AUC	IGF-I	INS	T3	T4
178	1	2	74	358	5.91	3.43	2	14.5	75	0.0042	300	2871	924	52.35	2.311	56.02
179	3	1	56	329	3.60	3.32	2	3.26	30	0.0042	450	1658	619	36.38	2.220	95.57
182	2	1	68	327	4.64	3.04	1	21.1	90	0.0021	0	2268	792	26.52	2.330	99.37
183	4	2	58	355	3.01	2.40	1	9.08	90	0.0021	0	1464	831	28.21	2.226	84.30
184	4	1	62	326	4.34	3.21	1	10.9	60	0.0021	0	2096	514	20.96	2.558	160.9
185	1	2	74	354	2.29	2.39	0	0	0	0	0	1082	688	30.72	2.304	95.95
186	2	3	60	389	2.66	2.25	1	7.18	30	0.0021	0	1287	769	21.73	1.894	66.58
188	4	2	58	352	5.04	3.37	2	14.2	30	0.0042	90	2461	686	26.05	2.519	79.72
189	1	1	66	324	5.87	3.86	2	10.4	75	0.0042	360	2852	880	29.17	2.037	89.43
190	2	2	54	350	3.62	2.64	2	5.87	60	0.0042	390	1638	608	22.42	2.395	79.64
191	1	3	52	385	2.32	2.28	0	0	0	0	0	1111	545	42.31	1.744	104.8
192	1	1	66	319	2.77	2.49	2	2.02	30	0.0042	390	1339	946	23.28	2.486	97.51
193	4	3	70	378	6.08	4.11	3	7.32	40	0.0062	60	2469	879	16.97	1.608	79.56
195	1	3	52	378	2.67	2.17	1	7.03	60	0.0021	0	1282	717	26.08	2.141	106.8
196	2	1	68	314	4.11	3.14	1	6.92	120	0.0021	0	2010	841	20.20	2.024	55.32
197	4	1	62	314	5.95	2.83	4	9.98	52.	0.0083	130	2777	1120	24.85	2.167	95.95
198	4	3	70	377	3.97	3.01	3	3.59	50	0.0062	75	1936	782	22.28	2.421	105.2
199	1	2	74	342	4.78	3.11	2	9.54	60	0.0042	90	2330	1088	37.87	4.069	125.3
201	2	3	60	374	2.71	2.32	3	2.30	30	0.0062	195	1273	534	27.96	1.959	74.35
203	2	1	68	306	2.63	1.86	2	4.78	45	0.0042	60	1281	996	10.33	1.953	77.07
204	4	3	70	368	4.16	3.09	1	8.49	150	0.0021	0	2033	639	23.85	1.985	99.22
205	3	3	72	368	2.36	2.26	1	1.72	30	0.0021	0	1126	495	28.67	2.539	81.42
206	2	3	60	368	2.11	2.08	0	0	0	0	0	1016	836	32.90	2.434	86.24
207	1	1	66	304	4.91	2.77	2	14.1	60	0.0042	120	2442	1014	38.29	3.059	113.1
208	3	1	56	304	3.12	2.32	3	4.14	40	0.0062	75	1522	950	19.34	2.597	117.1
209	4	2	58	332	5.23	3.53	1	14.8	150	0.0021	0	2534	706	21.02	2.115	80.96

Table 24 (cont'd.).

NO	BG	SG	PN	BL AGE	GH	GH BS LN	GH PK NO	GH PK AMP	GH PK LN	GH PK FREQ	GH PK INT	GH AUC	IGF-I	INS	T3	T4
210	4	1	62	304	8.03	5.70	3	8.91	60	0.0062	150	3720	789	12.67	1.770	72.41
211	4	2	58	332	4.95	2.15	2	13.7	60	0.0042	60	2453	954	27.23	2.395	138.9
213	2	2	54	325	3.23	2.34	2	5.08	45	0.0042	120	1581	990	38.29	2.890	94.17
214	4	1	62	295	4.19	3.80	2	4.34	30	0.0042	90	1994	1057	26.03	1.992	92.22

Table 25. Individual hormone parameters of Lake City steers born in 1990

						GH										
NO	BG	SG	PN	BL AGE	GH	BS LN	PK NO	PK AMP	PK LN	PK FREQ	PK INT	AUC	IGF-I	INS	T3	T4
60	2	1	80	298	5.80	4.16	4	6.16	45	0.0083	150	2739	790	36.49	2.324	92.85
85	3	3	64	404	3.04	2.66	2	2.89	30	0.0042	210	1474	617	35.91	1.894	92.77
101	3	1	76	375	2.76	2.51	2	2.13	30	0.0042	210	1335	869	54.35	2.545	103.9
102	3	2	58	399	2.50	2.39	1	1.89	30	0.0021	0	1203	909	39.60	2.675	99.06
104	1	1	52	365	3.45	1.52	2	6.25	105	0.0042	390	1618	894	32.82	2.343	111.2
106	1	2	78	389	3.52	3.00	2	3.73	45	0.0042	300	1684	781	38.96	2.291	92.77
109	1	3	60	415	2.39	2.09	1	2.36	90	0.0021	0	1151	602	39.92	2.076	114.9
110	2	1	80	358	3.74	3.19	1	6.75	120	0.0021	0	1789	935	42.97	2.343	93.31
111	3	3	64	414	2.13	2.08	0	0	0	0	0	1013	587	39.51	2.467	107.2
113	3	3	64	413	3.02	3.09	1	1.39	30	0.0021	0	1446	598	33.42	2.350	87.87
114	3	1	76	355	2.86	2.55	1	2.35	90	0.0021	0	1357	679	32.10	2.382	110.8
115	1	3	60	411	3.89	3.00	3	4.50	40	0.0062	180	1854	960	40.20	2.428	102.7
116	1	2	78	383	2.53	1.98	1	5.12	60	0.0021	0	1214	1160	61.44	2.317	100.3
117	4	2	66	383	2.79	2.72	1	1.29	30	0.0021	0	1325	718	49.70	2.272	103.4
120	3	2	58	381	2.95	2.35	2	5.14	30	0.0042	180	1439	857	43.17	2.962	101.5
122	3	2	58	379	3.19	2.46	2	4.28	60	0.0042	330	1522	561	23.96	1.946	75.75
123	3	1	76	351	3.50	2.56	2	6.91	60	0.0042	90	1696	1207	29.57	2.5	106.2
124	4	3	56	406	3.54	3.49	1	1.62	30	0.0021	0	1705	627	28.64	2.845	140.4
125	4	1	74	349	4.21	3.28	1	10.0	90	0.0021	0	2053	1079	20.58	2.623	113.5
127	4	3	56	404	3.07	2.80	2	3.27	30	0.0042	90	1451	821	24.48	2.395	92.15
131	4	1	74	347	3.91	3.90	1	5.10	60	0.0021	0	1888	968	39.15	2.682	121.6
134	3	3	64	403	1.80	1.79	0	0	0	0	0	864	519	18.73	2.350	99.30
135	4	2	66	374	2.73	2.77	1	1.34	30	0.0021	0	1318	577	37.28	2.278	93.47
136	2	3	62	402	2.95	2.35	3	2.83	40	0.0062	165	1389	492	55.57	1.783	87.17
137	4	3	56	402	3.08	2.72	2	2.98	45	0.0042	90	1476	760	22.79	2.923	87.72
138	1	1	52	346	4.52	4.43	1	8.78	30	0.0021	0	2228	1032	72.61	1.888	106.1

Table 25 (cont'd.).

						GH										
NO	BG	SG	PN	BL AGE	GH	BS LN	PK NO	PK AMP	PK LN	PK FREQ	PK INT	AUC	IGF-I	INS	T3	T4
141	4	1	74	344	3.80	3.37	2	4.52	45	0.0042	90	1796	981	59.54	3.352	131.7
143	1	2	78	372	3.00	2.50	2	3.51	45	0.0042	90	1465	1007	56.50	2.578	127.7
144	3	1	76	343	2.96	2.49	1	5.30	60	0.0021	0	1441	936	15.08	2.343	99.06
145	3	1	76	342	2.87	2.23	2	4.19	60	0.0042	330	1391	593	34.26	2.506	117.0
146	4	3	56	398	3.97	3.03	2	6.21	60	0.0042	90	1947	684	23.71	2.597	122.3
148	3	2	58	370	3.15	2.89	2	2.28	30	0.0042	360	1501	811	15.53	3.014	103.1
149	4	2	66	369	3.29	2.56	3	3.83	30	0.0062	90	1579	845	21.59	2.877	96.03
150	1	3	60	396	3.80	2.49	2	8.38	75	0.0042	390	1721	1049	44.34	2.395	125.3
152	1	3	60	395	3.22	2.30	2	6.59	60	0.0042	300	1556	878	26.36	2.018	120.1
153	2	3	62	395	3.15	2.79	3	2.08	30	0.0062	195	1509	514	33.61	2.213	83.76
154	3	2	58	366	3.20	2.43	3	3.50	50	0.0062	180	1485	816	16.77	2.363	70.00
155	1	2	78	365	3.57	2.39	3	5.35	50	0.0062	150	1737	1213	35.50	2.350	113.5
156	2	2	54	365	2.98	2.46	1	7.98	90	0.0021	0	1467	802	20.82	2.519	88.34
157	2	2	54	364	2.15	2.05	1	1.63	30	0.0021	0	1032	827	34.23	2.089	106.9
159	4	3	56	392	2.47	2.27	2	1.73	30	0.0042	240	1184	752	31.38	2.194	79.56
160	4	1	74	335	4.29	2.13	1	15.9	150	0.0021	0	2094	920	27.47	2.623	104.8
161	3	3	64	391	2.62	2.21	1	4.35	60	0.0021	0	1265	558	16.85	2.929	84.77
163	4	2	66	361	2.97	2.90	0	0	0	0	0	1428	658	18.31	2.916	115.5
164	3	3	64	389	3.83	2.89	3	3.93	60	0.0062	195	1789	921	13.34	1.972	76.14
165	4	2	66	361	3.15	3.13	0	0	0	0	0	1488	687	17.07	2.428	107.5
166	1	1	52	328	4.84	4.21	2	8.90	60	0.0042	360	2341	1212	34.45	3.098	100.3
167	4	1	74	328	2.88	2.77	1	3.14	30	0.0021	0	1337	1031	28.60	1.770	65.03
168	1	3	60	380	2.99	2.61	1	4.27	90	0.0021	0	1444	751	19.60	2.330	98.21
170	3	1	76	323	3.37	3.15	2	1.47	30	0.0042	90	1621	908	49.97	3.177	102.6
171	1	2	78	349	3.33	2.84	2	4.99	30	0.0042	390	1558	731	20.65	2.910	95.88
172	2	1	80	320	4.93	3.41	3	6.32	40	0.0062	195	2362	740	40.01	2.597	74.59

Table 25 (cont'd.).

						GH										
NO	BG	SG	PN	BL AGE	GH	BS LN	PK NO	PK AMP	PK LN	PK FREQ	PK INT	AUC	IGF-I	INS	T3	T4
173	1	1	52	320	2.78	2.43	2	2.67	45	0.0042	90	1320	877	32.76	2.714	96.34
176	3	1	76	317	2.80	2.53	2	4.60	30	0.0042	60	1371	851	31.13	2.994	96.19
177	2	2	54	345	2.10	1.16	2	6.21	60	0.0042	90	1036	927	34.98	2.317	84.22
178	4	1	74	316	3.79	2.92	2	6.38	60	0.0042	90	1815	1103	23.31	2.200	109.3
179	3	2	58	343	3.69	2.97	1	5.90	120	0.0021	0	1802	925	26.71	1.966	82.59
181	2	3	62	369	2.77	1.58	2	5.33	90	0.0042	390	1262	946	30.17	2.480	82.28
182	4	3	56	369	2.10	2.00	1	1.40	60	0.0021	0	1013	514	40.93	2.180	78.16
184	2	3	62	368	1.84	1.37	1	3.12	120	0.0021	0	896	941	43.04	2.467	76.37
185	3	3	64	368	2.13	2.11	0	0	0	0	0	1020	578	28.35	2.858	103.6
186	2	1	80	312	3.24	3.45	1	5.78	30	0.0021	0	1602	1014	42.62	2.565	92.38
187	2	1	80	311	3.78	1.90	2	10.0	90	0.0042	330	1863	830	45.57	2.526	92.22
188	2	2	54	339	2.14	2.12	0	0	0	0	0	1034	835	47.15	2.226	85.70
189	2	1	80	309	3.12	2.52	2	3.80	45	0.0042	60	1527	718	22.46	2.200	62.31
190	4	1	74	307	4.49	3.73	2	5.84	45	0.0042	240	2214	903	38.34	2.890	121.2
191	3	3	64	363	3.07	2.79	1	4.92	30	0.0021	0	1464	993	25.32	2.141	88.42
192	3	1	76	306	2.21	2.15	1	2.77	30	0.0021	0	1172	571	25.38	2.265	90.52
193	2	3	62	362	2.48	2.17	2	3.11	30	0.0042	240	1185	773	47.73	2.259	81.11
194	3	2	58	333	3.87	2.91	1	10.7	90	0.0021	0	1892	765	11.76	3.274	93.24
195	2	3	62	361	2.67	2.03	1	6.34	90	0.0021	0	1295	960	19.89	2.024	89.89
196	2	1	80	304	4.36	3.04	3	5.68	50	0.0062	180	2086	899	27.65	2.311	97.04
197	2	2	54	327	2.00	1.78	1	4.11	30	0.0021	0	949	675	40.25	2.083	72.26
199	2	2	54	326	2.48	2.03	2	4.37	45	0.0042	330	1219	907	35.87	1.933	79.56
200	4	3	56	353	5.83	3.53	2	13.4	60	0.0042	450	2537	616	20.88	2.213	103.4
201	2	3	62	354	2.08	1.89	1	2.38	60	0.0021	0	1009	991	29.55	2.5	92.92
9085	2	1	80	310	2.61	1.68	2	5.01	60	0.0042	330	1219	915	28.12	2.825	98.44

Table 26. Metabolism room performance and intakes of Lake City steers born in 1989

NO	BG	SG	PN	MR WT	MR ADFI	MR ADG	MR IADG
101	2	3	60	521	8.4	0.92	1.36
102	1	2	74	303	4.5	0.08	0.97
103	1	1	66	303	6.8	1.05	1.05
105	4	1	62	460	8.3	0.79	1.47
106	3	1	56	453	8.0	0.52	1.50
107	2	1	68	410	7.9	1.66	1.57
108	4	3	70	544	7.5	1.18	1.35
110	4	2	58	485	5.7	0.16	1.30
111	1	3	52	417	7.0	1.93	1.36
113	3	2	64	532	9.2	0.55	1.39
114	3	3	72	537	9.5	0.76	1.49
117	2	2	54	408	7.1	0.78	1.36
118	3	3	72	449	5.1	-0.84	0.98
119	2	2	54	403	6.5	0.31	1.23
120	1	3	52	405	6.7	1.34	1.28
126	2	1	68	399	5.6	0.09	1.14
128	4	3	70	576	8.2	0.25	1.42
129	3	2	64	449	5.9	-0.16	1.23
130	2	3	60	464	6.3	1.18	1.09
133	2	1	68	378	7.0	0.96	1.29
134	4	1	62	458	7.4	0.52	1.30
135	2	3	60	548	8.0	1.43	1.61
136	4	2	58	557	7.5	0.47	1.50
137	4	3	70	551	7.5	0.50	1.45
140	3	2	64	478	7.8	0.00	1.32
141	3	3	72	571	9.1	0.67	1.54
143	4	1	62	449	7.4	0.87	1.40
144	2	2	54	401	5.4	0.63	1.26
145	3	1	56	410	7.7	0.96	1.50
146	2	3	60	578	7.2	1.43	1.49
147	3	1	56	442	8.4	1.13	1.40
148	1	2	74	276	4.1	0.31	0.90
149	3	2	64	496	6.5	0.23	1.50
151	1	1	66	299	5.8	0.26	0.98
152	3	3	72	501	7.4	1.43	1.07
156	3	2	64	551	9.0	0.78	1.59
159	3	1	56	426	7.0	0.79	1.44
160	4	1	62	471	8.5	0.00	1.40
161	2	2	54	426	6.8	0.39	1.23
162	3	2	64	517	8.0	0.31	1.34
163	3	3	72	635	10.7	1.34	1.62
164	1	3	52	371	6.1	1.01	1.12
165	2	2	54	464	7.6	0.70	1.51
166	2	1	68	453	7.9	1.66	1.57

Table 26 (cont'd.).

NO	BG	SG	PN	MR WT	MR ADFI	MR ADG	MR IADG
167	2	3	60	449	7.3	1.26	1.27
169	2	2	54	396	7.1	0.86	1.30
170	3	1	56	467	8.7	1.48	1.59
172	4	3	70	587	8.1	0.92	1.37
173	3	3	72	489	7.9	0.67	1.30
174	4	2	58	494	6.5	0.70	1.52
176	4	3	70	557	7.2	0.50	1.36
177	3	2	64	449	8.0	0.31	1.30
178	1	2	74	351	7.1	1.17	1.16
179	3	1	56	435	7.1	0.44	1.36
182	2	1	68	399	7.4	0.70	1.43
183	4	2	58	408	5.5	-0.31	1.23
184	4	1	62	426	8.0	0.52	1.39
185	1	2	74	319	4.6	0.47	1.11
186	2	3	60	460	7.7	1.51	1.38
188	4	2	58	503	7.6	0.78	1.42
189	1	1	66	303	6.1	0.61	1.14
190	2	2	54	385	6.5	0.55	1.12
191	1	3	52	347	5.6	0.92	0.98
192	1	1	66	310	5.5	0.52	1.05
193	4	3	70	544	6.9	0.59	1.43
195	1	3	52	317	5.0	1.26	0.87
196	2	1	68	374	6.5	0.35	1.21
197	4	1	62	444	6.8	0.35	1.33
198	4	3	70	521	7.9	0.92	1.27
199	1	2	74	297	4.7	0.47	1.01
201	2	3	60	492	8.3	2.18	1.54
203	2	1	68	371	6.9	1.05	1.42
204	4	3	70	464	8.6	0.42	1.23
205	3	3	72	446	7.6	0.17	1.22
206	2	3	60	474	7.5	1.09	1.27
207	1	1	66	324	6.7	0.61	1.18
208	3	1	56	399	7.1	0.70	1.34
209	4	2	58	469	6.4	0.39	1.27
210	4	1	62	417	6.8	0.26	1.32
211	4	2	58	462	6.3	0.31	1.33
213	2	2	54	424	8.1	1.33	1.63
214	4	1	62	394	7.0	0.17	1.24

Table 27. Metabolism room performance and intakes of Lake City steers born in 1990

NO	BG	SG	PN	MR WT	MR ADFI	MR ADG	MR IADG
60	2	1	80	361	7.4	1.60	1.42
85	3	3	64	470	8.2	1.10	1.46
101	3	1	76	415	7.0	0.82	1.51
102	3	2	58	496	8.3	1.65	1.37
104	1	1	52	289	4.7	1.47	1.08
106	1	2	78	329	4.5	0.56	1.03
109	1	3	60	454	6.2	1.07	1.21
110	2	1	80	438	8.1	1.69	1.72
111	3	3	64	549	9.3	1.32	1.44
113	3	3	64	555	8.4	1.35	1.11
114	3	1	76	526	8.8	0.95	1.57
115	1	3	60	447	6.4	1.07	1.16
116	1	2	78	348	4.9	1.07	1.21
117	4	2	66	493	7.7	1.55	1.48
120	3	2	58	461	9.6	1.26	1.35
122	3	2	58	552	8.7	1.60	1.41
123	3	1	76	422	7.5	1.16	1.30
124	4	3	56	513	8.2	0.48	1.13
125	4	1	74	497	7.8	1.69	1.45
127	4	3	56	554	8.3	1.31	1.51
131	4	1	74	491	8.0	1.57	1.40
134	3	3	64	543	9.4	1.03	1.46
135	4	2	66	537	8.2	1.65	1.45
136	2	3	62	472	6.9	0.80	1.37
137	4	3	56	587	9.0	1.08	1.41
138	1	1	52	295	5.8	0.99	1.20
141	4	1	74	463	7.4	1.29	1.44
143	1	2	78	341	5.3	1.04	1.13
144	3	1	76	396	6.5	0.37	1.05
145	3	1	76	432	7.6	0.99	1.39
146	4	3	56	532	7.2	0.63	1.31
148	3	2	58	474	7.7	1.53	1.44
149	4	2	66	514	7.2	1.57	1.30
150	1	3	60	360	5.4	1.04	1.15
152	1	3	60	302	4.5	0.80	0.89
153	2	3	62	443	6.1	0.76	1.09
154	3	2	58	508	8.8	1.95	1.49
155	1	2	78	359	5.7	0.68	1.31
156	2	2	54	452	7.2	1.46	1.34
157	2	2	54	424	7.0	1.37	1.45
159	4	3	56	510	7.8	0.44	1.20
160	4	1	74	480	7.5	1.40	1.56
161	3	3	64	472	7.1	1.24	1.05
163	4	2	66	455	6.6	1.02	1.16

Table 27 (cont'd.).

NO	BG	SG	PN	MR WT	MR ADFI	MR ADG	MR IADG
164	3	3	64	449	7.8	1.31	1.16
165	4	2	66	474	7.5	1.27	1.29
166	1	1	52	261	4.9	0.38	1.02
167	4	1	74	472	8.2	1.28	1.47
168	1	3	60	357	6.2	1.03	1.22
170	3	1	76	434	7.4	0.95	1.30
171	1	2	78	268	4.6	1.00	0.98
172	2	1	80	381	6.7	1.79	1.22
173	1	1	52	272	4.9	1.00	1.01
176	3	1	76	341	6.8	0.86	1.08
177	2	2	54	433	7.0	1.57	1.56
178	4	1	74	444	7.4	1.99	1.40
179	3	2	58	417	7.6	1.33	1.18
181	2	3	62	447	7.5	1.31	1.45
182	4	3	56	491	8.3	1.00	1.08
184	2	3	62	449	7.4	1.39	1.41
185	3	3	64	518	9.0	1.07	1.45
186	2	1	80	361	6.6	1.49	1.26
187	2	1	80	337	7.4	1.61	1.31
188	2	2	54	459	7.8	1.57	1.51
189	2	1	80	379	6.6	1.69	1.40
190	4	1	74	435	6.9	1.24	1.41
191	3	3	64	510	8.6	1.63	1.41
192	3	1	76	388	7.3	0.87	1.31
193	2	3	62	481	7.8	1.43	1.31
194	3	2	58	415	7.9	1.50	1.10
195	2	3	62	452	8.4	1.39	1.56
196	2	1	80	343	6.0	1.28	1.18
197	2	2	54	424	7.5	1.28	1.53
199	2	2	54	409	7.5	1.33	1.29
200	4	3	56	428	7.8	1.15	1.14
201	2	3	62	444	8.1	1.15	1.60
9085	2	1	80	398	7.5	1.69	1.57

Table 28. Pen intakes and gains by period of Lake City steers born in 1989

BG	SG	PN	Period 1 DMI	Period 1 AND	Period 1 ADG	Period 2 DMI	Period 2 AND	Period 2 ADG	Period 3 DMI	Period 3 AND	Period 3 ADG	Period 4 DMI	Period 4 AND	Period 4 ADG	Period 5 DMI	Period 5 AND	Period 5 ADG
1	1	66	640	135	1.576	871	140	1.426	714	140	0.518	870	150	1.255	777	130	0.611
1	2	74	548	135	1.381	797	140	1.264	704	140	1.037	803	150	1.331	696	130	0.646
1	3	52	452	135	1.566	796	140	1.345	834	140	0.940	894	150	1.285	685	130	0.872
2	1	68	1106	189	1.730	1562	196	1.909	1353	196	1.018	1524	210	1.274	1263	182	0.922
2	2	54	1059	216	1.474	1902	224	1.934	1672	224	1.104	1995	240	1.767	1356	208	0.981
2	3	60	1273	216	1.627	2053	224	1.934	1725	224	1.205	1918	240	1.370	1470	208	1.385
3	1	56	1348	216	1.758	1788	224	1.893	1658	196	1.470	1492	210	1.210	1382	182	0.860
3	2	64	1416	216	2.071	2387	224	1.914	1701	196	1.539	1881	210	0.950	1553	182	1.570
3	3	72	1501	216	1.674	2227	224	2.136	1654	196	1.053	1893	210	1.534	1532	182	1.358
4	1	62	1308	216	1.758	1808	224	1.843	1855	224	1.195	1856	240	1.484	1528	208	0.436
4	2	58	1390	216	1.909	1859	224	1.752	1815	224	1.579	2187	240	1.247	1578	208	1.265
4	3	70	1390	216	1.884	1947	224	2.005	1913	224	1.205	1934	240	1.455	1657	208	1.461

BG	SG	PN	Period 6 DMI	Period 6 AND	Period 6 ADG	Period 7 DMI	Period 7 AND	Period 7 ADG	Period 8 DMI	Period 8 AND	Period 8 ADG	Period 9 DMI	Period 9 AND	Period 9 ADG	Entire Trial DMI	Entire Trial AND	Entire Trial ADG
1	1	66	913	145	0.938	749	135	1.310	1062	175	0.583				6596	1150	1.015
1	2	74	740	145	0.501	785	135	1.226	839	140	0.761	931	140	0.700	6842	1255	0.984
1	3	52	849	145	0.563	837	135	1.294	858	140	0.664	1548	210	0.797	7754	1325	1.022
2	1	68	1605	203	1.374	1624	189	1.464	2072	245	0.828				12108	1610	1.301
2	2	54	1601	232	0.694	1681	216	1.165	1709	224	1.559	2306	224	1.023	15279	2008	1.304
2	3	60	1875	232	0.772	1649	216	1.375	1840	224	1.073	3239	336	1.153	17040	2120	1.309
3	1	56	1684	203	1.162	1649	189	1.548	2047	245	0.650				13048	1665	1.315
3	2	64	1598	203	0.290	1630	189	1.200	1791	196	0.694	1709	196	0.953	15665	1812	1.254
3	3	72	1825	203	0.883	1525	189	0.600	1776	196	0.856	2650	294	0.705	16583	1910	1.196
4	1	62	1796	232	0.978	1683	216	1.176	2273	280	0.659				14107	1840	1.181
4	2	58	1306	203	0.358	1710	189	1.800	1562	196	0.810	1840	196	1.053	15248	1896	1.318
4	3	70	1857	232	0.860	1690	216	0.661	1873	224	1.083	3295	336	1.285	17556	2120	1.319

Table 29. Pen intakes and gains by period of Lake City steers born in 1990

BG	SG	PN	Period 1 DMI	Period 1 AND	Period 1 ADG	Period 2 DMI	Period 2 AND	Period 2 ADG	Period 3 DMI	Period 3 AND	Period 3 ADG	Period 4 DMI	Period 4 AND	Period 4 ADG	Period 5 DMI	Period 5 AND	Period 5 ADG
1	1	52	482	130	1.127	806	135	1.394	594	116	0.958	611	112	0.850	595	108	1.071
1	2	78	551	130	1.179	791	135	1.327	871	145	1.345	947	140	1.231	773	135	0.504
1	3	60	662	130	1.288	847	135	1.394	773	145	1.032	785	140	0.940	852	135	1.260
2	1	80	1191	208	1.444	1509	216	1.617	1409	232	0.890	1639	224	1.468	1704	216	1.743
2	2	54	780	156	1.611	1262	162	1.638	1156	174	1.343	1255	168	1.350	1171	162	1.204
2	3	62	1101	182	1.508	1436	189	1.956	1295	203	1.095	1552	196	1.030	1509	189	1.764
3	1	76	1412	208	1.681	1797	216	1.071	1751	232	1.828	1815	224	0.850	1690	216	0.892
3	2	58	1331	208	1.335	1825	216	1.848	1458	232	0.763	1947	224	1.093	1819	216	1.470
3	3	64	1404	208	1.537	1825	216	1.176	1686	232	1.212	1914	224	0.952	1660	216	1.753
4	1	74	1272	208	1.228	1825	216	1.606	1561	203	1.631	1780	196	1.308	1490	189	1.680
4	2	66	1310	182	1.690	1213	189	1.068	1087	174	1.238	1485	168	1.039	1310	151	1.547
4	3	56	1392	208	1.437	1767	189	1.692	1375	203	1.106	1726	196	0.949	1661	189	1.680

BG	SG	PN	Period 6 DMI	Period 6 AND	Period 6 ADG	Period 7 DMI	Period 7 AND	Period 7 ADG	Period 8 DMI	Period 8 AND	Period 8 ADG	Period 9 DMI	Period 9 AND	Period 9 ADG	Entire Trial DMI	Entire Trial AND	Entire Trial ADG
1	1	52	723	116	1.251	723	112	0.790	595	104	0.977				5129	933	1.063
1	2	78	822	145	1.236	724	140	0.713	812	145	0.798	578	100	0.431	6870	1215	0.992
1	3	60	951	145	0.876	832	140	1.134	813	145	0.845	1088	160	1.208	7604	1275	1.105
2	1	80	2127	232	1.476	2139	224	0.972	1692	208	1.686				13411	1760	1.405
2	2	54	1447	174	1.655	1338	168	0.904	1308	174	1.369	879	120	1.074	10596	1458	1.357
2	3	62	1535	203	1.229	1307	196	1.122	1560	203	1.296	1480	216	1.023	12775	1777	1.325
3	1	76	1748	232	1.349	1935	224	1.063	1502	208	1.119				13650	1760	1.234
3	2	58	1764	203	1.609	1874	196	1.342	1835	203	0.972	1142	140	0.684	14996	1838	1.251
3	3	64	2052	232	1.369	2010	224	1.144	1943	232	1.007	2066	256	0.914	16561	2040	1.220
4	1	74	1861	203	1.184	1629	196	0.717	1449	182	1.117				12867	1593	1.313
4	2	66	1251	145	1.361	1165	140	1.053	1180	145	1.001	827	100	1.801	10827	1394	1.293
4	3	56	1737	203	0.983	1548	196	0.752	1616	203	1.184	1800	224	0.662	14622	1811	1.150