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GROWTH AND MUSCLE DEVELOPMENT OF FEEDLOT CATTLE OF DIFFERENT GENETIC BACKGROUNDS

By

Dan Edward Eversole

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ABSTRACT

GROWTH AND MUSCLE DEVELOPMENT OF FEEDLOT CATTLE OF DIFFERENT GENETIC BACKGROUNDS

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Four cattle types of steer calves (averaging 220.8 kg) from a common source were used in a two-year study to evaluate the role of genetic selection and crossbreeding and ration energy level on feedlot performance and muscle growth. The selection-crossbreeding scheme resulted in the following cattle types: Unselected Hereford (UH); Selected Hereford (SH); Angus x Hereford x Charolais (AHC); and Angus x Hereford x Holstein (AHH). One hundred and nineteen steers were randomly allotted within cattle type to either an 80% corn grain (HG) or a 90% corn silage (HS) ration.

Steers from each cattle type were slaughtered on day 1 and at the termination of the feedlot trial. The semitendinosus (ST) muscle was removed from the hindquarter immediately after slaughter, trimmed, weighed and then frozen. The ST was later assayed for nucleic acids, moisture, protein, lipid and muscle protein fractions. Pituitary glands were also removed from the initial and terminal slaughter cattle and analyzed for growth hormone concentration and content.

Average daily gains increased with increasing frame size for the UH, SH and AHC cattle types. However, the AHH steers gained less than

expected based on their frame size. Averaged across cattle types, steers fed HG gained 26% faster than the HS fed steers. Smaller-framed steers tended to be more efficient than the larger-framed steers.

Semitendinosus muscles from terminal slaughter steers had less moisture, more intramuscular lipid and slightly more protein than muscles from initial slaughter steers. Ration and cattle type had no effect on the proximate analysis of the ST from terminal slaughter steers.

Nucleic acid concentrations across cattle types declined with advancing age but total DNA, RNA and protein were higher in the terminal than the initial slaughter cattle. Differences in total content were a reflection of variations in ST weights. Steers fed HS had significantly heavier ST weights, more total protein/ST and higher protein/DNA than HG fed steers. Total RNA/DNA among cattle types was lower for the terminal slaughter steers than those slaughtered initially. However, protein/DNA increased with age.

Ration had no significant effect on the various muscle protein fractions. The total amount of sarcoplasmic and myofibrillar protein across cattle types increased substantially between the initial and terminal slaughter. The larger-framed AHC and AHH cattle types had more total protein in each muscle protein fraction than the smaller-framed UH and SH steers. Relative differences among cattle types were a function of ST weight. The SDS-polyacrylamide gels revealed that there were no differences in myofibrillar protein composition in ST muscles among the cattle types.

Blood samples from all steers were taken on days 1, 29, 57, 113 and 169 of the feedlot trial and analyzed for insulin and growth hormone using the radioimmunoassay technique. Overall mean serum insulin levels were not consistently affected by cattle type but increased in all steers during the trial. Steers fed HG had significantly higher serum insulin concentrations than HS fed steers except on day 1. Serum insulin was positively correlated to ADG.

Serum growth hormone levels throughout the trial were not consistently affected by cattle type or ration. Serum concentrations of growth hormone in steers fed HG were lower and tended to fluctuate more erratically than in steers fed HS. Serum growth hormone and ADG were not related. Similar observations were noted between serum insulin and serum growth hormone.

The absolute anterior pituitary weight increased with increasing frame size. Initial slaughter steers had higher pituitary concentrations of growth hormone than the terminal slaughter steers. Total pituitary growth hormone tended to level off between 215.2 and 519.4 kg to live weight. Steers fed HS had significantly higher pituitary concentration and content of growth hormone than HG fed steers.

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INTRODUCTION

The ultimate goal of all livestock production programs should be to produce enough red meat to feed a hungry world and do it on the least acreage with a diminutive amount of feed. However, regardless of livestock production technologies, there has not yet been developed a meat animal species to fit the above needs. Too many inferior meat animals are still being maintained in breeding programs as seedstock that require considerable capital and land which could yield higher returns if the quantity and quality of the end product were upgraded. Consequently, these inferior meat animals are unsuitable to practical economic and management systems.

Livestock producers must attempt to produce meat animals that will yield a higher percentage of edible lean with a minimal amount of fat and still have acceptable palatability to satisfy the consumers. Within the last decade alterations in market demands have placed more and more emphasis on inherent muscling and freedom from adipose tissue. Human health advocates have insisted that excessive animal fat in human diets can lead to cardiovascular diseases. As a result of the aforementioned factors, consumers have become more and more conscious in selecting meat that has a high proportion of lean. Meat and animal scientists are, therefore, concerned primarily with efficient meat production and those processes regulating growth and muscle development

as well as the fattening of livestock. Needless to say, these body mechanisms are of fundamental importance to the livestock producer and consumer.

The research study reported herein involved four genetic types of feedlot steers from the same herd. This two-year study was undertaken to better understand the effects of selection, genetic type and ration energy level on the variation of growth and muscle development of steers from four genetic backgrounds. The growth and development of bovine muscle was examined by analyzing the moisture, protein and fat content, muscle protein fractions and nucleic acids in semitendinosus muscle tissue. This project was also designed to measure serum levels of growth hormone and insulin during different stages of growing and finishing. In addition, the components of the fibrillar protein fraction were examined by SDS-polyacrylamide gel electrophoresis for any detectable differences among the four genetic steer types.

LITERATURE REVIEW

Growth and Development

Growth and development are of utmost importance to the livestock and meat industry because production of meat is dependent upon this complex, universal process. People associated with livestock production rely upon all the various phases of growth and development but are primarily concerned with that period from birth to slaughter. A thorough understanding of the growth and development of an animal is essential if animal agriculture is to continue to provide food for an increasingly hungry world. Various parameters of growth such as the initiation of growth, the termination of maturity and the regulation of growth rate are not entirely understood. However, the knowledge that is implicit about growth and development remains to be applied at the production level.

Growth and development are so complex that they are not easily defined by animal scientists. There have been wide differences of opinion on what should constitute growth of an individual. Schloss (1911) broadly defined growth as a correlated increase in the mass of the body in definite intervals of time in a way characteristic of the species. Brody (1945) later proposed that growth was the aspect of development in which new biochemical units were synthesized either during cell multiplication, cell enlargement or the incorporation

of materials from the environment. Other authors have other schemes of classifications. Hammond (1952) and McMeekan (1959) have defined growth as an increase in bodyweight until mature size was attained. However, most researchers are in agreement that growth is more than an increase in weight or size. The most commonly accepted definition of growth has been proposed by Maynard and Loosli (1969). They have indicated that "true" growth involved an increase in muscles, bones and organs and should be distinguished from any increase resulting from fat deposition. Hence, any propagation in water, protein or ash content of structural tissues constituted true growth.

Since growth and development are so closely related, it is important that both are defined as one entity. Generally speaking, development includes growth but also accentuates cellular differentiation of a body. Lewis (1939) had explained that development was a process involving growth, whereas Hammett (1936) had defined growth to include development. Development is a gradual progression from a lower to a higher stage of complexity as well as a gradual expansion in size. More specifically speaking, development is defined as those alterations in body shape or conformation until the various parts reach maturity (Hammond, 1952; McMeekan, 1959).

The relative importance of the functions of the parts or tissues for survival of an animal directs which parts and which tissues will be formed first. The order of tissue growth and development is synonymous in all species (Brody, 1945). Tissues that are most vital toward the growth and development of an animal are formed

before birth. Thus, the order of tissue growth and development follows a sequential trend according to physiological importance commencing with the central nervous system and progressing to bone, tendon, muscle, intermuscular or seam fat and subcutaneous fat (Palsson and Verges, 1952a; McMeekan, 1959). Although the growth and development of the central nervous system is nearly complete at birth, Berg and Butterfield (1968) have shown that the major tissues of the animal body, bone, muscle and fat, grow at relatively different postnatal rates. Bone completes a greater proportion of its growth early in life than either muscle or fat. Fat makes the greatest proportion of its growth later in life. This was also earlier demonstrated in sheep by Hammond (1921), Wallace (1948) and Palsson and Verges (1952b) and in swine by McMeekan (1940).

Tulloh (1964) stated that bone in cattle increased with body weight but at a decreasing rate which caused the percentage of bone to decline as body weight increased. Weiss et al. (1971) and Johnson (1974) also agreed with this finding stating that postnatal bone growth was considerably slower than muscle or fat growth and was not proportional to body growth. Cuthbertson and Pomeroy (1962) found similar results in swine. They concluded that the bone production rate from 50 to 68 kg of carcass weight was the growth in length predominating over growth in thickness and density. Thereafter, the rate of bone growth between 68 and 92 kg of carcass weight was characterized by a thickening and ossification of the bones and the rate of bone production decreased during the latter stage indicating that muscle and fat were comprising a higher percentage of the carcass weight.

Quantitatively, adipose tissue is the most variable constituent in the body and in muscle (Callow, 1947; 1948). Most animals have very little body fat stores at birth. Palsson (1955) stated that during early development adipose tissue has the lowest nutrient priority and is, therefore, the latest maturing tissue. The accretion of fat in the body during postnatal growth and development is the result of animals being fed for extended periods of time at intervals in excess of their maintenance requirement. Consequently, a greater proportion of nutrients are available to be utilized by the lowest priority tissue. Other factors such as sex, physiological age and breed of animal may also influence fat deposition in the body and muscle. Schemmel et al. (1970) found that the effect of ration contributed 40% of the total variation of body weight and 74% of the variation of body fat for rats of the same age and sex.

Cuthbertson and Pomeroy (1962) noted that the body fat content increased as the liveweight of the animal increased. Zucker and Zucker (1963) demonstrated under normal dietary conditions in the growing rat that the accretion of body adipose tissue was closely related to body weight and independent of age. Bergen (1974) illustrated that animals reach compositional maturity at a given body weight rather than age. He schematically showed that the rate of fat accretion became greater than protein accretion at some body weight before the protein accretion curve plateaued. He further summarized that there was little or no change in lean body mass and an excessively obese animal may eventually develop.

Filer et al. (1973) stated that the carcass body fat of Pitman-Moore miniature pigs increased from 1.7% at birth to 23.9% at day 28. The percentage of fat in the carcass declined thereafter to 21.5% at day 56. Richmond and Berg (1971) reported changes occurring in the patterns of fat deposition among the depot types as liveweight increased in swine. They found that as liveweight increased from 23 to 114 kg, subcutaneous fat increased from 77.7 to 82.4% of the total fat and the intermuscular fat decreased from 18.1 to 13.4%. This was in agreement with earlier work done by Hedrick (1968) who indicated that fat deposition occurred initially around the vital organs followed by intermuscular, subcutaneous and finally intramuscular fat.

Skeletal muscle is the most abundant body tissue on a weight basis (Hedrick, 1968) and is of great economic importance to the livestock industry. Consequently, the sequence of events associated with differentiation and development of skeletal muscle cells will be discussed. Mesoderm, the middle third of the embryonic primary germ layer, gives rise to skeletal muscle tissue. During embryonic development, tissue of mesoderm origin exists as presumptive myoblasts which are mononucleated cells committed to becoming a muscle cell but incapable of fusion or synthesis of contractile proteins. These cells differentiate into myoblasts, postmitotic cells containing many ribosomes. Mononucleated myoblasts are capable of synthesizing myofibrillar proteins and of fusing with other similar postmitotic cells. The myoblasts may fuse with other myoblasts to form myotubes or may fuse with existing myotubes. The nuclei in the multinucleated myotube

cannot divide mitotically. Consequently, the nuclei number in the newly-formed myotube is indicative of the number of myoblasts that fused to form it. Through maturation and differentiation myotubes will form mature myofibers or skeletal muscle cells (Stromer et al., 1974).

Since domestic animals are born at a relative late stage in their fetal development, these mammals contain approximately all the myofibers that they will ever possess because myoblast fusion discontinues about the time of birth. Work done with swine (McMeekan, 1940; Staun, 1963; Strickland et al., 1975), sheep (Joubert, 1956), and chickens (Smith, 1963) agreed with the above finding. However, Goldspink (1962) and Chiakulas and Pauly (1965) have shown in cytological studies with rodents that the number of muscle fibers increased postnatally during the first two weeks after birth. The small postnatal increase in muscle cell numbers can be regarded as an extension of embryonic muscle differentiation (Stromer et al., 1974).

Winick and Noble (1965) have characterized the growth of muscle tissue into three distinct phases. Hyperplasia or the increase in cell number predominates in the first phase and is succeeded by a concurrent phase of hyperplasia and hypertrophy, the increase in cell size. The third phase is predominately hypertrophy. During early muscle growth hyperplasia is primarily responsible for the growth of muscle tissue before fusion occurs. After fusion and in the absence of mitosis, Goldspink (1972) stated that hypertrophy (characterized by the increase in size and number of individual myofibers in the muscle cell) is entirely responsible for muscle growth.

Postnatal Muscle Growth

There is a substantial postnatal increase in muscle mass. Elliot and Cheek (1968) stated that one-fourth of the body weight at birth in man and rat was comprised of muscle tissue. Regardless of mature body size, Young (1970) reported that muscle constituted approximately one-half of the body weight of mammals.

Growth of skeletal muscle subsequent to birth occurs through enlargement of existing cells or hypertrophy rather than hyperplasia (Joubert, 1956; Staun, 1963). Although nuclei within the multinucleated cells of skeletal muscle do not mitotically divide, there has been considerable evidence that the total nuclei number within muscle cells increases postnatally (Winick and Noble, 1965; Moss, 1968; Robinson, 1969; Cheek et al., 1971; Williams and Goldspink, 1971; Powell and Aberle, 1975). Small mononucleated cells that lie between the basement membrane and the plasmalemma of muscle cells have been called satellite cells (Mauro, 1961) and are responsible for the postnatal increase in skeletal muscle nuclei. Swatland (1971) reported that hypertrophy in muscle fibers is accompanied by an increase in the number of nuclei as a result of fusion with the satellite cells. MacConnachie et al. (1964) and Moss and Leblond (1971) have shown that satellite cells do mitotically divided and are capable of synthesizing DNA but lose these abilities upon fusing with the mature myofiber. Consequently, the fusion process results in the addition of one nucleus to the skeletal muscle cell (Stromer et al., 1974; Swatland, 1977).

Postnatal muscle growth is also accomplished through growth in length of the myofibrils. Due to the longitudinal bone growth in a body, the muscles attached to these bones must also lengthen. Goldspink (1968; 1972), Bendall and Boyle (1967) and Close (1972) reported that the longitudinal growth of the myofibrils was achieved primarily by the formation of new sarcomeres at the tendons rather than the lengthening of existing sarcomeres. This finding was further substantiated by Griffin et al. (1972). Using labelled adenosine in young rats, they demonstrated that the newly synthesized actin was aggregated at the ends of the myofibers. Consequently, the most-recently made sarcomeres were terminally located and were not as long as those sarcomeres found toward the center of the myofibril.

Other morphological changes such as postnatal increases in myofibril numbers have been reported in normal skeletal muscle growth. Numerous studies by Goldspink (1970; 1971; 1972) have revealed longitudinal splitting of the existing myofibril resulting in two smaller daughter myofibrils. Rodent skeletal muscle cells possessing an approximate diameter of 40 μM frequently split longitudinally during muscle contraction. Electron microscopy has revealed that actin or the thin filaments created an exertion on the Z-disk causing it to tear near its center. As a result, the lengthwise separation of the existing myofibril yielded two daughter myofibrils.

Changes in Protein and Nucleic Acids During Muscle Growth

Protein

Water, protein and minerals constitute about 82%, 14% and 4%, respectively, of the fat-free body weight at birth (Forbes, 1968). However, although minerals remain relatively constant on a percentage basis during growth, protein increases to approximately 21 to 24% while water declines and eventually plateaus at 70 to 75%.

Numerous investigators (Dickerson, 1960; Dickerson and Widdowson, 1960; Sink and Judge, 1971; LaFlamme et al., 1973; Giovannetti and Stothers, 1975) have demonstrated that the proportions of protein and lipid in muscle tissue increased and the proportion of water decreased with increasing animal age. The percentage of water in muscle decreased most rapidly during early growth. However, the change in water percentage was progressively less rapid with age.

The protein nitrogen of skeletal muscle has been divided into a number of fractions by the use of appropriate extractants. Sarcoplasmic proteins have been identified as those muscle proteins that are soluble in water or low ionic strength buffers (Helander, 1957). This protein nitrogen fraction contains the glycolytic and citric acid cycle enzymes, hemoglobin, myoglobin and the electron transport chain enzymes (Robinson, 1952a, 1952b; Forrest et al., 1975) and is characterized by its low viscosity, low molecular weight and globular shape. Hill (1962) found that the sarcoplasmic proteins accounted for 25% of the total nitrogen in ovine, 15 to 20% in bovine and 20 to 25% in porcine muscle, respectively.

The muscle protein fraction that is soluble in high salt concentrations has been identified as fibrillar protein (Helander, 1957). This nitrogen fraction contains the proteins associated with muscle contraction and relaxation. The components are myosin, actin, tropomyosin, troponin, M and C proteins, and α and β actinin (Robinson, 1952a, 1952b; Whitaker, 1959; Forrest et al., 1975). The sarcoplasmic and fibrillar proteins together constitute the intracellular protein of the muscle cell. Approximately 53%, 55% and 56% of the nitrogen in ovine, bovine and porcine muscle, respectively, makes up the fibrillar fraction (Hill, 1962).

The extracellular proteins, collagen, elastin and reticulin constitute another muscle protein fraction called stroma protein (Whitaker, 1959). Helander (1957) found that these proteins were insoluble in both water and high ionic strength buffers. Hill (1962) stated the extracellular proteins comprised 8 to 12% of the total nitrogen in ovine muscle, 12 to 18% in bovine and 7 to 10% in porcine muscle.

The fourth muscle protein fraction, non-protein nitrogen, is separated from the sarcoplasmic protein fraction by its solubility in trichloroacetic acid (Dickerson and Widdowson, 1960). This fraction contains free amino acids, peptides, urea, creatine, creatinine, creatine phosphate, nucleotides and nucleosides (Forrest et al., 1975). The non-protein nitrogen comprised 11 to 13% of the total nitrogen in porcine, ovine and bovine muscle (Hill, 1962).

Development is associated with changes in the relative proportions of the muscle protein fractions of the muscle cell. Both the fibrillar and sarcoplasmic proteins differ according to the stage of development with the rate of change being related to the time of functional development of the muscle (Young, 1970). The increase in the fibrillar protein fraction predominates during the period of rapid protein accumulation (Gordon et al., 1966) and, consequently, the percentage of total muscle protein in the fibrillar fraction increases. As a result, the other fractions decline with continued growth (Young, 1974). Dickerson and Widdowson (1960) demonstrated in the pig, which is mobile shortly after birth, that the fibrillar fraction reached its adult proportion of the total nitrogen during the first 4 to 6 weeks after birth. However, Baril and Herrman (1967) and Dickerson (1960) found that the chick synthesized fibrillar proteins more quickly than the pig. They concluded that the fibrillar protein increased primarily during the first 18 days and by day 28 the chick muscle had reached adult composition.

Even though alterations occur in the concentration of fibrillar protein, the absolute concentration of sarcoplasmic protein is relatively stable. Hartshorne and Perry (1962) showed that although the percentage contribution of the sarcoplasmic fraction to the total nitrogen did not increase markedly, the protein composition of the water-soluble fraction underwent changes during development from fetal to adult rabbit skeletal muscle. Kendrick-Jones and Perry (1967) suggested that the activity pattern of a particular muscle was very

important in determining the time at which the activities of the muscle enzymes rise sharply to adult values. Needham (1931) stated that enzyme activity in the leg muscles of the chick and guinea pig, both of which are born at a more advanced stage of development and are capable of independent existence, was very close to adult value. On the contrary, the leg muscles of the rat and rabbit, animals which are incapable of much movement and are less mature at birth, did not attain adult enzyme activity value until some time post partum. Dickerson (1960) found that the main increase in sarcoplasmic proteins of chick pectoral muscle occurred between day 18 and day 28. Kendrick-Jones and Perry (1967) showed that the total amount of sarcoplasmic protein increased only 20% over a period from birth to 20 days. LaFlamme et al. (1973) stated that total protein, fibrillar and sarcoplasmic proteins in cattle increased 133, 150 and 125%, respectively, of the initial concentrations. Total protein and fibrillar concentrations continued to increase up to a live weight of 445 kg, whereas the concentration of water-soluble proteins changed very little after 145 kg of body weight.

Blaxter (1964) reported that the non-protein nitrogen fraction of muscle tissue constituted a greater proportion of the total nitrogen in the young animal because of the higher water content in the fetal tissue than the adult tissue. Kendrick-Jones and Perry (1967) demonstrated with rabbit skeletal muscle that the non-protein nitrogen fraction was relatively stable from 18 days prior to birth to day 48 of the adult state. Dickerson (1960) reported that the

non-protein nitrogen of the fowl pectoral muscle had reached its adult proportion of the total nitrogen by day 18.

The extracellular proteins or the stroma fraction attained a maximum level at birth and decreased thereafter in the bovine (Bendall and Voyle, 1967) and also in the pig (Widdowson and Dickerson, 1960). LaFlamme et al. (1973) stated that cattle weighing 445 kg had a collagen concentration of 30% less than those measured initially at 45 kg and decreased very little after 345 kg. Blaxter (1964) reported that stroma protein accounted for 17% of the total nitrogen in porcine muscle at birth and decreased to approximately 3.5% in the adult pig. Bendall and Voyle (1967) stated that collagen content of the longissimus and the semitendinosus muscles was lower in the six-month-old animal (0.43 and 0.77% for the respective muscles) than in the calf (1.3 and 1.2% of the wet weights of muscles, respectively).

Nucleic Acids

Muscle cell nuclei like those of other tissues are diploid in numbers. By measuring the amount of deoxyribonucleic acid (DNA) in tissues, the number of nuclei can be estimated. Vendrely (1955) reported that the DNA content per diploid nucleus in the bovine ranged from 6.4 to 7.1 picograms per nucleus and in the pig from 5.1 to 6.8 picograms per nucleus. Work done with rat muscle and brain by Enesco and Leblond (1962) indicated that the amount of DNA per diploid nucleus was a constant 6.2 picograms per nucleus.

It is difficult to estimate cell numbers by the amount of DNA present in skeletal muscle because the cells are multinucleated. On the contrary, DNA content is a good index of cell numbers in tissues comprised of mononucleated cells. However, Robinson (1971) and Goldspink (1972) reported that the nuclei number of muscle tissue is a meaningful measure of postnatal growth because of the maximum cytoplasmic volume that is governed by one nucleus (Cheek et al., 1971, and Goldspink, 1972). Enesco and Leblond (1962) and Winick and Noble (1965) suggested that protein:DNA and weight:DNA ratios can be used to determine the physiological cell size per nucleus.

Ribonucleic acid (RNA) is a good measure of the available synthetic machinery in a tissue capable of synthesizing protein (Wannamacher, 1972). Winick and Noble (1965), Powell and Aberle (1975) and Munro (1969) suggested that a low ratio of RNA to DNA within a tissue is indicative of a low protein synthesizing capacity. However, Harbison et al. (1976) reported that RNA:DNA ratio may be valid for comparisons among different tissues but was not valid for comparisons within a single tissue. This was further substantiated by Sarkar (1977a). He noted that the pig liver on a unit weight basis had a higher RNA:DNA ratio and higher levels of RNA, and was considerably more active than muscle in synthesizing protein. Sarkar et al. (1977b) ranked the following pig organs and tissues at birth from the highest to lowest for their protein synthesizing capacity: liver, muscle, brain, heart, kidney and lung. In addition, the brain exhibited the greatest progressive increase of any tissue in the

ratio of RNA:DNA from 0 to 50 days of age (1.42 to 1.74, respectively). They suggested that the high RNA:DNA ratio was a reflection of cell function rather than specifically protein synthesizing capacity.

Both RNA and DNA concentrations in skeletal muscle decrease during postnatal muscle growth. Tsai et al. (1973) reported a continuous decrease in DNA concentration in porcine skeletal muscle from birth to 16 weeks of age. RNA concentration reflected an immediate rapid increase succeeded by a decline to a rather constant level. Robinson (1969), Gilbreath and Trout (1973) and Powell and Aberle (1975) found similar results. They showed that the concentrations of DNA and RNA in porcine skeletal muscle decreased up to 100 days of age and remained at a constant level thereafter. Harbison et al. (1976) reported that both muscle RNA and DNA concentrations in porcine skeletal muscle decreased significantly ($P < .05$) by nearly 50% between 23 and 91 kg of body weight and remained constant between the live weights of 91 and 118 kg. Similar findings with rodents (Devi et al., 1963; Robinson and Lambourne, 1970; Lipsey, 1973) and ruminants (LaFlamme et al., 1973; Johns and Bergen, 1976) have been reported. LaFlamme et al. (1973) noted a 50% decrease in the DNA concentration of bovine longissimus muscle at 445 kg compared to 45 kg of body weight. However, the concentration of DNA changed very little after a live weight of 345 kg.

Increase in body weight and size of muscle contribute to the decrease in the tissue concentration of DNA. Tsai et al. (1973) reported that a relatively constant amount of DNA was continuously

being diluted by the increasing accumulation of protein. Postnatal skeletal muscle growth was the result of an increase in muscle mass per nucleus or per unit of DNA (Moss et al., 1964; Enesco and Puddy, 1964) because the amount of DNA per nucleus in diploid cells was constant (Vendrely, 1955).

Content of muscle DNA and RNA during development has also been investigated and has been well-documented. An increase in total skeletal muscle DNA may indicate that the number of muscle fiber nuclei is increasing if it is assumed that the amount of DNA per nucleus is constant. Gordon et al. (1966) measured a gradual increase in total DNA content of rat quadriceps between 6 and 9 weeks of age. Between 9 and 11 weeks of age a sharp inclination in total DNA was observed. Finally the DNA content plateaued at 13 weeks. Numerous other investigators have reported an increase in total DNA content in rodents (Robinson and Bradford, 1969; Enesco and Puddy, 1964; Buchanan and Pritchard, 1970; Howarth and Baldwin, 1971); in chickens (Moss et al., 1964; Moss, 1968); in cattle (LaFlamme et al., 1973); in sheep (Johns and Bergen, 1976) and in swine (Robinson, 1969; Powell and Aberle, 1975; Harbison et al., 1976; Sarkar et al., 1977b). Powell and Aberle (1975) reported that total RNA, DNA and protein increased markedly during growth up to 210 days of age. In a study using pigs no older than 120 days, Robinson (1969) noted that the total DNA in the semitendinosus muscle appeared to level off at 4 months of age whereas the triceps brachii continued to increase. On the other hand, Harbison et al. (1976) noted a substantial increase in total muscle RNA

between the live weights of 23 and 104 kg and, also a 2.7-fold increase in total DNA between 23 and 118 kg.

Mechanism of Protein Synthesis

Protein synthesis consists of two major stages (Lehninger, 1975). The first stage is referred to as transcription in which deoxyribonucleic acid (DNA) serves to direct the formation of the following ribonucleic acids (RNA): ribosomal RNA (rRNA), messenger RNA (mRNA) and transfer RNA (tRNA). The inherited genetic information stored in DNA (Munro, 1976) that is needed to provide for the primary structure of specific proteins can be transmitted to the protein synthesizing apparatus in the cytoplasm. Jacob and Monod (1961) observed that the mRNA was transcribed from nuclear DNA and possessed a nucleotide sequence that coded for amino acids. The second stage is called translation. Once the newly mRNA is synthesized, it then moves out of the nucleus via the endoplasmic reticulum into the cytoplasmic portion of the cell where the polypeptide chains are fabricated (Young, 1974). Messenger RNA, in association with ribosomal RNA (rRNA), is decoded and serves as a template to direct the synthesis of specific cellular proteins.

The *in vivo* protein translational process (polypeptide synthesis) has four primary steps (Lehninger, 1975): (1) activation of amino acids; (2) initiation; (3) elongation; (4) termination. Each step requires certain cofactors and specific enzymes as necessary components.

The first step in the utilization of amino acids for protein synthesis requires their activation. This step occurs in the cytoplasm and requires ATP, tRNA, amino acids, aminoacyl-tRNA synthetases and Mg++ (Lehninger, 1975). Energy from ATP is used to enzymatically esterify amino acids to their respective tRNA. Spadoni and Gaetani (1972) stated that there was a specific tRNA molecule for each amino acid that is to be incorporated into the primary structure of a protein molecule.

The second step in the formation of the polypeptide chain is initiation (Lehninger, 1975). The necessary components are the initiating charged tRNA (methionyl tRNA), three initiation factors, Mg++, GTP, 40 S ribosomal subunit, 60 S ribosomal subunit, mRNA and its initiation site (codon). This process, in the presence of the initiation factors, GTP and Mg++, involves the formation of an initiation complex whereby mRNA, which serves as the template for protein synthesis, binds with the initiating charged tRNA and the 40 S ribosomal component. The 60 S ribosomal subunit attaches itself to the initiation complex and forms a functional ribosome (Iwasaki et al., 1968).

Elongation of the polypeptide chain is the third step in protein synthesis (Lehninger, 1975). The protein chain elongates by the sequential addition of amino acids transferred from charged tRNAs in response to a particular codon in the mRNA. After each peptide bond is formed, the ribosomes move along the RNA template to bring the next coded trinucleotide site on the template (mRNA)

into that position on the ribosome where it can receive the tRNA complex of the next amino acid to be added to the growing peptide chain. This step in protein synthesis requires two elongation factors, GTP, Mg++ and specific charged aminoacyl-tRNAs (Lehinger, 1975; Haselkorn and Rothman-Denes, 1973).

Capecchi and Klein (1970) and Lehninger (1975) reported that the final step in protein synthesis (termination) requies three polypeptide releasing factors and a termination codon on the mRNA. This process involves the termination and release of the polypeptide chain from the ribosome-mRNA complex when the termination codon on the mRNA and the releasing factors are present. The ribosome comes off the message either in the form of subunits (60 S and 40 S) or as an intact ribosome (80 S) which later dissociates into its free, smaller subunits. In the former case, factor DF or IF_3 binds to the smaller subunit (40 S) and prohibits the reassociation with the larger subunit (60 S). In addition, factor DF seems to increase the affinity of the 40 S subunit for mRNA. In the latter case, factor DF enhances the dissociation of larger subunit into its smaller subunits (Weissbach and Brot, 1974; Davis, 1971). Falvey and Staehelin (1970) observed that the 60 S and 40 S ribosomal subunits reenter the ribosomal pool and reassociate if mRNA is present.

Rate-Limiting Factors Influencing the Rate of Muscle Protein Synthesis

An alteration in the rate of degradation or synthesis can change the protein content in a muscle cell (Munro, 1970). Consequently, the amount of protein synthesized can be regulated by a rate-limiting step at any point in the chain of events leading to the formation of a protein molecule.

Aminoacyl-tRNA Supply

It is well established that the different species of tRNA in the cell are present in widely varying amounts (Caskey et al., 1968). Cohen and Grinblat (1972) observed that the activity and amount of the synthetase enzymes rather than the limiting quantity of available tRNA are primarily responsible for a decreased formation of aminoacyl-tRNA in the diabetic rat. Mushinski and Potter (1969) reported that certain species of leucyl-tRNA from mouse liver were missing when plasma cell tumors were induced. In addition, Gaetani et al. (1964) observed that the level of amino acid-activating enzymes in gastrocnemius muscle from protein-depleted rats was less than normal. However, Young (1974) and Stephen (1968) disagreed with the finding of Gaetani et al. (1964). The former investigators found that the activity of aminoacyl-tRNA synthetase in muscle was not reduced during dietary protein depletion. However, one should be aware that different synthetases may react differently under similar conditions.

Amount of Messenger RNA

The stability of mRNA in mammalian cells appears to vary widely (Revel and Hiatt, 1964). Palmiter (1973) reported that mRNA of animal cells had a longer half-life than bacterial cells. He also found that the physical half-life of ovalbumin mRNA was one day and was translated 20,000 times. A rapid turnover of mRNA was observed when high levels of actinomycin D were present (Staehelin et al., 1963). Actinomycin D is known to inhibit DNA-dependent RNA synthesis in both mammalian (Goldberg and Rabinowitz, 1962) and bacterial (Hurwitz et al., 1962) systems. Low doses of actinomycin D prevent rRNA synthesis, whereas higher doses selectively inhibit mRNA synthesis (Munro, 1970). Manchester (1976) stated that cortisol, estrogen and testosterone stimulated the production of RNA in the target cell nucleus. The entry of the receptor-steroid complex into the nucleus initially stimulated transcription of nuclear DNA to produce the mRNA for the nucleolar polymerase responsible for transcribing rRNA. Consequently, the overall production of RNA was enhanced.

Florini and Breuer (1966) reported that the administration of growth hormone to hypophysectomized animals increased the activity of the ribosomes as well as the number of polyribosomes present. They concluded that the absence of growth hormone could indicate a deficient amount of mRNA transcribed. Breuer and Florini (1966) suggested that growth hormone promoted RNA synthesis by causing an increase in the activity or synthesis of RNA polymerase.

Ribosome Availability and Capacity

More than 80% of muscle RNA is ribosomal and this proportion appears to be maintained during protein depletion; consequently, a change in RNA also reflects an alteration in ribosome content (Young, 1970). Millward et al. (1973) reported that muscle synthetic rates in rat skeletal tissue fell markedly on the dietary regimes which involved starvation or a protein-free diet. In a similar study, the protein synthetic rate in skeletal muscle was reduced with a concurrent reduction in total RNA content when rats were fed a restricted protein diet (Young and Alexis, 1968). Young (1974) indicated that a decrease in total RNA also caused a reduction in ribosomal RNA. Stivastava (1969) reported that as the rate of protein accretion in the mouse became negligible during later growth, there was a smaller proportion of ribosomes associated with polyribosomes.

Wool and Cavicchi (1966) found that skeletal muscle ribosomes from alloxan-diabetic rats were approximately 60% less effective than ribosomes from control animals in the transfer of radioactivity from tRNA-^{14}C-phenylalanine into protein. However, the injection of insulin into the alloxan-diabetic rats, when compared to the untreated diabetic animals, caused reaggregation of the ribosomes and restored their activity to normal or near-normal levels. In another experiment Martin and Wool (1968) determined whether or not ribosomal subunits from normal and diabetic rats differed in potential activity. They reported that the reassociated ribosomes containing the 60 S subunit from the muscle of diabetic rats were less effective in polyuridylic acid-directed polypeptide synthesis.

Amino Acids

Protein synthesis in liver is stimulated by feeding an imbalanced amino acid diet (Sanahuja and Rio, 1967; Noda et al., 1975); however, the opposite is true in skeletal muscle (Closa et al., 1974; Noda et al., 1975). The free amino acid pool in skeletal muscle is depleted of the most limiting amino acid because the liver has an increased need for this amino acid for its protein synthesis. Ellison and King (1968) reported a marked depression of the muscle concentration of the most limiting amino acid in the diet within 3 to 5 hours after feeding. A reduction in diet consumption appeared 20 hours later. The latter finding of Ellison and King (1968) was in agreement with Devilat et al. (1970) who showed a marked depression in growth rate and feed intake in pigs fed a protein-deficient and an incomplete diet. Akinwande and Bragg (1974) observed reduced growth rates in day-old chicks when either an excessive or deficient level of dietary lysine was fed. Reduced growth rate occurred because of a reduction in feed intake. In addition, they reported lower concentrations of protein, RNA and protein:DNA in the gastrocnemius muscle of one-month-old chicks fed a deficient or excessive level of dietary lysine.

Factors Influencing Growth Rate, Body Composition and Muscle Growth

Carcass composition and growth rate of meat-producing animals are affected by a variety of interacting hormonal, dietary and environmental factors. Individual variation in animal age, genetics, health and exercise will also influence postnatal muscle growth, body

composition and/or growth (Martin and Ezekwe, 1975). The existence of the meat animal variation within a certain species is due primarily to the interaction between environment and heredity (Hedrick, 1975). Environment minimizes or maximizes the potential for growth and development while heredity dictates and provides for this potential. In addition, environmental factors such as nutrition govern the rate and extent to which the phenomenon of growth and development is attained.

Genetics

Red meat animals within a given breed and among breeds vary in growth rate, feed efficiency, postnatal muscle growth, body composition and/or reproductive efficiency. Harbison et al. (1976) developed an obese line of pigs by mating unimproved, inbred Poland China boars to Duroc x Yorkshire females. In an attempt to study total muscle content in the two genetic lines of pigs, they reported that the obese and muscular pigs weighing less than 45 kg did not differ in total physically separable fat when expressed either as total weight or as a percentage of carcass weight. However, the muscular genetic line of pigs possessed more separable muscle at 45 kg of live weight and did have less separable fat than the obese line after a live weight of 45 kg was reached. The results also indicated that the total DNA in the longissimus muscle was significantly greater in the muscular line than in the obese line of pigs, and total DNA in physically separable muscle was the only measurement consistently correlated with the estimate of total muscle. In contrast, Bergen et al. (1975) reported that

nucleic acids were not precise indicators for determining the rate of muscle growth involving lean and genetically-obese mice. The hindlimbs of lean mice had higher rates of RNA and DNA accretion during 6 and 18 weeks of age. However, total limb DNA was not significantly different between the two groups during this time period. These workers assumed that some of the DNA accretion in the genetically-obese mouse was due to hyperplastic obesity. They concluded that there may be a deficit in the muscle development of the genetically-obese mouse.

In a study involving light and heavy muscled pigs, Powell and Aberle (1975) reported that the heavy muscled pigs exhibited greater muscle development resulting in larger loineye areas and greater weights of separable lean in the ham region. In addition, total DNA and total protein in the biceps femoris, when adjusted for differences in live weight, were significantly lower in the light muscled than the heavy muscled pigs when differences over all age groups were analyzed.

Ezekwe and Martin (1975) further substantiated the above findings by reporting a similar pattern in the cellular characteristics of skeletal muscle of fast-growing Yorkshire pigs and slow-growing Ossabaw pigs. The lean Yorkshire pigs were 31.8 kg heavier than the Ossabaw pigs at six months of age and also had significantly heavier ($P < .005$) semitendinosus muscles than the feral Ossabaw pigs. The lean domestic Yorkshire pigs had 2.3 times as much total muscle DNA as well as a two-fold increase in both the total muscle protein and the protein:DNA ratio than did the feral obese pigs. Morevoer, the Ossabaw pigs had one-fourth as much of the total RNA content in the semitendinosus muscle

as did the Yorkshire swine. These data indicated that there was greater muscle growth in the lean pigs, and the heavier muscle weights of these pigs were a reflection of larger cell size (protein:DNA) and cell number (DNA).

Several investigators have reported the results of selecting for mature body size or growth rate. However, only a few cases have reported the alterations at the cellular level in skeletal muscle when selecting for mature body size or growth rate. Luff and Goldspink (1967) first observed that mice selected for large body size had larger muscles than mice selected for small body size. The increased muscle weight was accompanied by increased mean fiber diameter and fiber number. Luff and Goldspink (1967) concluded that the main component of muscle weight response to selection for body weight was fiber number and that fiber number, but not fiber diameter, is genetically determined (Luff and Goldspink, 1970).

Byrne et al. (1973) and Hanrahan et al. (1973) also studied how selection for body size affected muscle weight and its components. Both investigators found that selecting for body weight in mice either at 5 or at 10 weeks of age increased muscle weight, fiber number and mean fiber diameter. However, neither investigator reported body composition at the time of selection. Byrne et al. (1973) observed that changes in both fiber number and fiber diameter generally tended to be positively correlated with the direct response in body weight.

In contrast to Luff and Goldspink (1970) and Aberle and Doolittle (1976), Hanrahan et al. (1973), Ezekwe and Martin (1975),

Luff and Goldspink (1967) and Byrne et al. (1973) reported that some portion of the total variation in both fiber number and fiber diameter is genetically determined. Smith (1963), Byrne et al. (1973) and Hanrahan et al. (1973) indicated heterosis accounted for fiber diameter for various muscles from animals selected for increased body weight. However, Aberle and Doolittle (1976) studied two different genetic lines of mice and found similar body compositions at 60 days of age. They also indicated that the strain selected for large body size at 60 days had heavier muscle weights due to increased number of fibers and not to enlarged fiber diameter. Mean fiber diameters were similar in the two lines and did not contribute to changes in muscle weights.

The discrepancy of enlarged fiber diameters accompanying the selection for increased body size and consequently, increased muscle weight can be attributed to differences in physiological maturity and to the possibility of work-induced hypertrophy. At a given level of activity, muscles from more obese animals would be required to do more work per unit muscle. On the other hand, larger fiber diameters would also be observed in muscles obtained from animals that were more physiologically mature.

Exercise, Compensatory Muscle Hypertrophy and Work-Induced Hypertrophy

Because of its cellular hypertrophy and its morphological structure, skeletal muscle is a most suitable tissue to study the effects of exercise, work-induced hypertrophy or compensatory hypertrophy (that which occurs in response to an increased work load) on

muscle growth. Helander (1961) and Malina (1969) stated that a muscle when exercised will increase its strength by undergoing hypertrophy and, conversely, inactivity gives rise to muscular atrophy. Goldspink (1970) reported that the proliferation of energy-producing and mitochondrial enzyme systems resulted from repetitive low intensity exercise, whereas the synthesis of myofibrillar protein is enhanced by intensive exercise. Helander (1961) observed that restricted activity reduced the myofilamental density and increased the sarcoplasmic content in the muscle cell, whereas exercise enhanced the myofilamental density.

In a more recent study, Hubbard et al. (1975) induced compensatory hypertrophy in the rat plantaris muscle by removing the synergistic gastrocnemius muscle. During the first post-operative week, there was an increase in stromal and sarcoplasmic proteins, an elevation in total DNA and a corresponding decline in the myofibrillar protein concentration. However, there was an increase in myofibrillar protein and a decrease in sarcoplasmic protein from day 7 to day 60. After 60 days post-surgery, the hypertrophied rat plantaris muscle doubled the wet weight of the contralateral controls.

Lesch et al. (1968) reported a rapid increase in muscle mass when an acute increase in work load was imposed on the soleus muscle of the rat by tenotomy of its synergistic plantaris and gastrocnemius muscles. Christensen and Crampton (1965) demonstrated an increase in RNA, DNA and protein in the gastrocnemius muscle of preconditioned rats that were forced to exercise. Crews et al. (1969) observed

that exercise resulted in a significant decline ($P < 0.01$) in the percentage of carcass fat in rats.

Goldberg (1968) demonstrated that work-induced hypertrophy in alloxan-diabetic rats could be induced without insulin. In another study, Goldberg (1967) concluded that pituitary growth hormone was not essential for work-induced hypertrophy in skeletal muscle. However, growth hormone and insulin were essential for developmental growth. Goldspink (1970) reported that increased fiber diameter was accompanied by work-induced hypertrophy. This was earlier substantiated by Goldberg (1967; 1968) who reported that the average diameter of the hypertrophied soleus and plantaris muscles was significantly lower than those of the control rats.

Nutrition

Nutrition contributes significantly to optimal muscle growth (Goldspink, 1964) and has a profound effect on body composition and animal growth (Hedrick, 1975). In respect to muscle growth, Hill et al. (1970) and Cheek and Hill (1970) proposed that muscle protein synthesis was reduced during a protein-deficient diet while a calorie-deficient diet primarily affected muscle DNA synthesis. Hill et al. (1970) observed a reduction in protein synthesis with a concurrent decrease in total DNA (nuclear number), protein:DNA (cell size/nucleus) and RNA:DNA (protein synthesizing machinery) when a protein-deficient diet was fed to weanling rats. Conversely, the same investigators reported that a calorie-deficient diet decreased the rate of DNA

replication. Mendes and Waterlow (1958) earlier found marked reductions in protein:DNA ratio of muscle from rats that received low protein diets.

However, Howarth (1972) did not support the preceding hypothesis proposed by Cheek and Hill (1970) and Hill et al. (1970). Howarth (1972) clearly demonstrated that protein synthesis had priority over DNA synthesis when muscle growth was impaired by feeding a protein-deficient ration. Weanling rats fed 24% crude protein gained more weight within a two-week period than did similar rats fed 6, 12 or 18% crude protein diets. As the dietary protein level decreased, there was a corresponding decline in the weight (1,369, 1,243, 1,011 and 662 mg/muscle) of the gastrocnemius muscle and its constitutents for the respective crude protein diets. The 6% protein ration allowed no muscle DNA accumulation but permitted a small increase in muscle protein accumulation. Relative to the 24% crude protein diet, there was a greater decrease in total DNA accumulation with the decrease in total RNA accumulation being intermediate to DNA and protein. However, there was a loss in the accumulation of total muscle RNA for the weanling rats fed a 6% crude protein diet.

Trenkle (1974) demonstrated that a protein- or calorie-deficient diet in rats caused reduced growth rates as well as reduced muscle growth, RNA, DNA and protein content. Limiting energy, protein or energy and protein did not change muscle weight:DNA or protein:DNA ratios. Muscle continued to grow but the rate of growth was retarded during dietary restriction.

Winick and Noble (1966) documented the effect of food restriction on cell number and/or cell size. They reported that the effect of calorie and protein restriction depended on the phase of growth at the time of restriction. Rats subjected to early malnutrition from birth to weaning were not able to fully recover normal growth and cell division was impeded. However, rats that were malnourished after weaning had a reduction in cell size but were able to recover normal growth during refeeding. They concluded that undernutrition has more of a permanent suppressing effect on DNA synthesis than on RNA or protein synthesis. Undernutrition seemed to cause permanent growth retardation in an animal at a critical time when the accumulation of DNA was rapid.

Restrictions in dietary energy or protein greatly influence muscle growth and fat accretion in the production of red meat. Clausen (1959) reported three basic concepts governing lean meat and fat accretion in the pig. The first concept stated that no pig can form lean meat up to the limit determined by its heredity unless its diet contained sufficient quantities of protein of high biological value. This concept was in agreement with Kropf et al. (1959) who reported equal rate of gain between pigs fed a 12 and 16% crude protein ration supplemented with high quality protein but reduced gains in pigs fed a 16% crude protein ration with low quality protein. The second concept stated that no pig can be forced, by means of extraordinary high levels of protein in its diet, to produce more lean meat than permitted by its heredity. Finally, the third concept stated that

when the daily requirements for maintenance and lean meat production in the pig have been met, the rest of the ration must inevitably be used for the accretion of fat. In summary, the following conclusion can be made. Whether or not a response is attained by supplying additional protein over and above requirements for maintenance and growth to a fixed ration will depend upon the hereditary ability of the pigs to produce lean meat and the level and composition of the proteins supplied in the fixed ration.

Smith et al. (1967), Kropf et al. (1959) and Wallace (1964) reported a linear increase in the percentage of lean cuts as the dietary crude protein level increased at each ratio of amino acids from soybean meal and corn. Smith et al. (1967) observed that pigs fed a ration containing 17.2% crude protein yielded a significantly ($P < 0.05$) higher percentage of lean cuts than pigs fed diets which contained 11.3, 12.8 and 14.3% crude protein. Each of the four rations maintained a similar ratio (60:40) of amino acids from soybean meal and corn. Corn sugar was used to adjust the protein level down to the desired level and to maintain a constant proportion of protein from corn and soybean meal within each ratio.

Body composition has been shown to vary according to ration type. Bond et al. (1972), Johnson et al. (1967) and Garrigus et al. (1967) have demonstrated that feeding high energy rations to cattle resulted in increased carcass fat when fed equal lengths. As a result, carcass quality was higher in cattle fed a high grain ration than those fed a high roughage diet (Richardson et al., 1961; Utley et al., 1975; Oltjen et al., 1971).

Henrickson et al. (1965) reported that the nutritional plane imposed during the last half of the feedlot gain period governed the major variations in carcass composition. They found that feedlot cattle fed a high nutritional level during the last 200 lb of gain possessed 0.8% less bone, 2.8% less lean and 4.0% more fat than the counterparts fed a moderate level of nutrition. Similar findings were reported by Waldman et al. (1971). Waters (1908) was first to illustrate the difference in nutritional demand of body tissues.

Hormones

The secretory products from various endocrine glands play a vital role in regulating growth and development. Their secretions, the hormones, catalyze and control many diverse metabolic processes. Hormones are chemical agents which are synthesized by specialized ductless glands and are released into the blood in minute quantities (Goodman, 1974). Hormones are transported by the blood to various parts of the body where they exert stimulating or regulating actions upon cellular functions.

There are a number of growth and developmental hormones and each have different specificities and varying actions. A detailed discussion will therefore be restricted to insulin and growth hormone and their control of growth and muscle protein synthesis.

Althen (1975) reported that insulin and growth hormone were important growth promoting hormones and both caused an increased rate of protein synthesis and had a positive effect on nitrogen balance. Rabinowitz and Zierler (1963) proposed that insulin and growth hormone

acted singly and together between meals to insure an ample supply of energy to the body tissues or to store excess energy. A later suggestion by Weil (1965) revealed that protein synthesis was stimulated synergistically by growth hormone and insulin. As time after a meal increased, the stimulation of protein synthesis changed from insulin to growth hormone.

Insulin has been shown to stimulate the transport of amino acids into muscle cells (Manchester and Young, 1958; Kipnis and Noall, 1958; Snipes, 1967; and Manchester, 1972). Manchester (1970) administered insulin into rat diaphragm muscle and noticed an increased accumulation of methionine, alanine and histidine. In addition, Reeds et al. (1971) demonstrated with rabbit muscle that insulin administration enhanced valine, lysine, leucine, histidine and arginine transport into the muscle cell. Cahill et al. (1972) reported that insulin was more influential in lowering essential than nonessential amino acids in the circulating plasma.

Earlier work by Manchester and Krahl (1959) showed that the incorporation of intracellular synthesized amino acids into protein was enhanced by insulin. They suggested that the effect of insulin on protein synthesis was independent of amino acid transport into the cell. A stimulation of amino acid uptake and protein synthesis was reported with rat diaphragm in vitro due to insulin (Wool and Krahl, 1959). Wool and Moyer (1964) reported that insulin still stimulated uptake of amino acids into muscle protein after giving doses of actinomycin D sufficient to block RNA synthesis. Goldstein

and Reddy (1970) could not demonstrate a stimulatory incorporation of amino acids into protein when muscle tissue was incubated in adequate concentrations of amino acids and in a sodium free system containing insulin. They suggested that insulin exerted its major mode of action by stimulating muscle protein synthesis through increased amino acid transport.

Insulin has been reported to have three possible effects on tissue protein synthesis (Manchester, 1972): (1) the ribosome to polysome ratio; (2) total number of ribosomes present; and (3) its regulatory role in the movement of ribosomes along the mRNA. He proposed that insulin increased muscle protein synthesis by promoting translation, whereas in liver, insulin promoted protein synthesis by increasing RNA synthesis (transcription).

Leader et al. (1971) and Wool et al. (1966) reported a variation in the protein synthetic rate between the control and diabetic animals. They suggested that a cell sap factor was responsible for the decreased capacity to initiate synthesis in the diabetic animals. Wool et al. (1972) later proposed that a defective 60 S subunit caused inactive ribosomes in muscle tissue from the diabetic rat. Once insulin was administered, the activity of the ribosomes was restored. This was in agreement with earlier work done by Castles et al. (1971). Additional work by Wool et al. (1968) demonstrated that the aminoacyl-tRNA synthetase activity in the diabetic animal was lower than normal, and the administration of insulin restored the synthetase activity.

Alterations in the polysome profile have been reported during an inadequate insulin supply (Wool and Kurihara, 1967; Tragl and Reaven, 1972). The ratio of heavy polysomes to free ribosomes decreased during an insulin deficiency. Wool and Kurihara (1967) suggested that insulin initially enhanced the translation of an existing mRNA for a specific protein.

Hypophysectomy has been shown to decrease tissue protein synthetic rate and tissue DNA content (Cheek and Hill, 1970; Trenkle, 1974). However, these investigators administered growth hormone to the hypophysectomized animals and partially restored tissue DNA to near normal levels. Cheek and Hill (1970) suggested that normal nuclear proliferation in muscle tissue was dependent upon growth hormone injections to hypophysectomized rats. Earlier work by Beach and Kostyo (1968) substantiated the findings of Cheek and Hill (1970) but the former investigators found no variation in the DNA concentration per mg of wet tissue between hypophysectomized rats injected with growth hormone and hypophysectomized controls.

Growth hormone has been reported to affect tissue protein synthesis by enhancing amino acid transport (Snipes, 1967; Jefferson and Korner, 1967). Using intact rat diaphragm muscle in an in vitro study, Snipes and Kostyo (1962) demonstrated a decreased alanine uptake in hypophysectomized animals. This finding was later substantiated using histidine (Snipes, 1967). Kostyo (1964) and Rillema and Kostyo (1971) noted that the treatment of growth hormone to hypophysectomized rats increased the accumulation of amino acids in the diaphragm muscle.

However, Reeds et al. (1971) reported that the stimulation of amino acid transport by growth hormone was not a necessary step to stimulate protein synthesis on a short-term basis. Turner et al. (1976) postulated that the role of growth hormone in the adult was to sustain cellular protein synthesis when there was a decrease in the level of substrate amino acids.

Growth hormone has also been shown to promote peptide bond formation (Kostyo and Rillema, 1971), to increase RNA synthesis (Garren et al., 1967) and to enhance the incorporation of amino acids into protein in vitro (Kostyo, 1964; Jaspar and Brasel, 1973). Cheek and Hill (1970) reported an increase in muscle RNA content of growth hormone-treated hypophysectomized rats. In a similar study using growth hormone-deficient hypophysectomized rats, Barden and Korner (1969) reported inactivity in muscle ribosomes resulting from a defective 40 S ribosomal subunit.

Florini and Breuer (1966) also experimented with hypophysectomized rats treated with growth hormone injections. They reported that a stimulation in RNA polymerase did not occur until after an increase in the ribosomal activity was observed. The previous finding was later shown by Kostyo and Rillema (1971). The latter investigators reported that growth hormone enhanced the ability of ribosomes to promote elongation or peptide bond formation.

Goldberg (1969) reported that growth hormone had no effect on muscle protein turnover. However, Jefferson et al. (1974), Goldberg et al. (1974) and Cahill et al. (1972) have shown that insulin reduced

breakdown of muscle protein. Li et al. (1975) reported that insulin increased the latency of cathepsin D activity in perfused psoas muscle.

Relationship of Growth to Insulin and Growth Hormone

In a review of the literature, conflicting data have been reported in the relationship between body growth and growth hormone. Purchas et al. (1971) obtained jugular venous blood from 40 Holstein heifers at monthly age intervals from 4 to 10 months of age. Although the plasma growth hormone was significantly reduced by feeding melengestrol acetate (MGA), the levels of pituitary growth hormone in the same heifers at slaughter time were not influenced by MGA. As a result, plasma growth hormone was negatively correlated ($r = -0.37$) to growth rate. Earlier work by Siers and Hazel (1970) also showed negative correlations between serum growth hormone and both growth rate and lean cut percentage in swine. On the other hand, serum growth hormone was directly related to carcass fat in pigs weighing 90 kg.

Althen and Gerrits (1976a) measured serum and pituitary growth hormone in Yorkshire and Duroc pigs genetically selected for low and high backfat. These two genetic strains varied mostly in body composition and not in growth rate (Althen and Gerrits, 1976a; 1976b). It was observed that the high backfat lines for both breeds had lower pituitary and serum growth hormone levels as well as lower serum growth hormone levels at weaning than the unselected control line in both the Duroc and Yorkshire breeds. These data indicated that the selection

for high backfat was associated with lower serum growth hormone levels. Likewise, Wangsness et al. (1977) also found that genetically-obese pigs had lower plasma growth hormone concentrations than did lean pigs at 1, 3 and 6 months of age.

On the contrary, Weiss et al. (1974) observed higher levels of plasma growth hormone in genetically-obese pigs than in lean, muscular pigs susceptible to stress and of corresponding weight. They suggested that the difference in circulating growth hormone levels between the two lines of pigs used in the study may be related to the difference in body composition rather than to the stress-susceptible characteristics. In addition, Topel et al. (1972) found no significant differences in circulating growth hormone levels in meat-type pigs that were stress-resistant or stress-susceptible. The results of Weiss et al. (1974) were in agreement with those of Siers and Hazel (1970), who reported lower serum growth hormone levels in muscular pigs than in obese pigs.

Hafs et al. (1971) and Siers and Swiger (1971) have further substantiated a negative relationship between growth hormone and growth rate in cattle and swine, respectively. Johns and Bergen (1976) reported that serum growth hormone levels in growing lambs averaged between 4.8 and 8.8 ng/ml from birth to 90 days but decreased thereafter to 2.4 ng/ml at 4 months of age.

Trenkle and Irvin (1970) observed no significant differences in plasma growth hormone between 18 day-old calves and 13 month-old animals. However, the latter cattle had plasma growth hormone levels

that were positively related to carcass weight, ribeye area and live weight gains but negatively correlated to 12^{th} rib fat thickness ($r = -0.11$).

Hafs et al. (1971) and Siers and Hazel (1970) have suggested that rapidly-growing animals utilized serum growth hormone at a more rapid rate than slow-growing animals and, consequently, had lower levels of serum growth hormone. Trenkle and Irvin (1970) postulated that the target tissues of the mature animal were not as responsive as in the younger animal to the low physiologic levels of growth hormone; hence, growth rate declined with age. Gershberg (1957) had earlier suggested that cessation of human growth was due to an alteration in the responsiveness of the target cells and not to a deficiency of growth hormone.

Turman and Andrews (1955) concluded that true growth was stimulated in pigs injected with growth hormone. Although growth hormone did not increase average daily gain, it did lower dressing percentage, reduced carcass fat, decreased feed/gain ratio and increased nitrogen retention. Machlin (1972) injected a dose of 0.13 mg growth hormone/kg body weight in pigs and observed improved feed conversion, increased rate of gain, higher percentage of lean cuts and a lower dressing percentage. Nalbandov (1963) suggested that growth stasis occurred because there was less growth hormone per unit of body weight in heavier animals. He also postulated that vigorous growth occurred only as long as the ratio of circulating growth hormone per unit of body tissue was high enough to stimulate muscle and bone growth.

In agreement with the aforementioned hypothesis, Althen and Gerrits (1976a) reported that serum growth hormone levels in swine at birth were significantly higher ($P < 0.01$) than either at 2 months of age (weaning) or at time of slaughter (95 kg body weight). Likewise Bassett et al. (1970) noted a 20-fold difference in plasma growth hormone concentration in fetal lambs and adult sheep. Trenkle (1971a) reported a 5-fold decrease in the secretion rate of growth hormone in cattle varying in age from 3 to 17 months. In addition, Purchas et al. (1970) and Swiatek et al. (1968) observed higher plasma growth hormone levels at birth than at any other age in cattle and swine, respectively.

The concentration of growth hormone in circulating blood is a reflection of its clearance rate as well as its rate of secretion from the anterior pituitary (Trenkle, 1976). The assessment of growth hormone status of an animal is based upon plasma or serum growth hormone, anterior pituitary growth hormone, rate of growth hormone turnover, hypothalamic growth hormone releasing hormones, tissue responsiveness to growth hormone or a combination of these (Purchas et al., 1970). In addition, Hafs et al. (1977) reported that somatostatin was a potent inhibitor of growth hormone and insulin secretion. Siers and Hazel (1970) and Purchas et al. (1970) have suggested that the metabolic clearance rate (MCR) of growth hormone by body tissues more adequately described the growth hormone status of an animal. Measuring the MCR in swine, Althen and Gerrits (1976b) reported that clearance per unit of body weight was reduced in larger animals. Trenkle (1977) found that the pituitary growth hormone, MCR and secretion decreased significantly ($P < 0.01$) when related to increased body weight.

Johns and Bergen (1976) reported that the ovine growth hormone concentration in the anterior pituitary (μg pituitary growth hormone/ 45.4 kg of body weight and μg/mg wet weight) was unaltered ($P > 0.05$) from birth to 4 months of age. However, the total growth hormone content of the anterior pituitary significantly increased ($P < 0.01$) from a low of 180 μg/gland at birth to 3,682 μg/gland at 120 days of age. Curl et al. (1968) also noted that the total growth hormone content in the bovine pituitary increased with age because of increased gland size. Expressed as either per unit of body weight or per unit of gland, the concentration of growth hormone decreased with age. These data were in agreement with those obtained from Holstein heifers (Armstrong and Hansel, 1956).

Baird et al. (1952) measured pituitary growth hormone in pigs selected either for rapid or slow gains. They noted that the faster gaining pigs had consistently higher pituitary growth hormone content per unit of body weight but not pituitary growth hormone concentration. However, the total pituitary growth hormone content per unit of body weight eventually plateaued and then decreased with age. These changes were similar to those of live weight gains. Baker et al. (1956) reported heavier pituitary weights with a corresponding increase in total pituitary content as an animal became older. As a result, the total content per unit of body weight decreased with advanced age because the ratio of pituitary weight to body weight decreased.

In a later study Macmillan and Hafs (1968) reported a linear relationship from birth to one year of age between the anterior

pituitary weight and body weight of Holstein bulls. However, there was a decrease in the weight of the anterior pituitary at the onset of puberty (6 months of age). They postulated that the pituitary gland had an increased sensitivity to the increasing androgen titers. In a similar study Purchas et al. (1970) measured plasma and pituitary growth hormone in bulls slaughtered at monthly intervals from birth to one year of age. They observed that the pituitary growth hormone content and concentration, expressed as either μg/mg of gland or per unit of body weight, increased until 3 to 4 months of age and then declined to measured levels at birth and were constant thereafter. These investigators also reported that the growth rate was not closely correlated to plasma growth hormone concentration, pituitary growth hormone concentration, plasma growth hormone content, total pituitary growth hormone per unit of body weight or total pituitary growth hormone content.

Variable results on blood growth hormone levels have been observed when the type of ration or diet has been changed. Meites and Fiel (1965) noted that rats fed a protein-deficient diet for prolonged periods of time exhibited decreased pituitary and plasma growth hormone activity and concentration. Malnourished rat pups have been reported to have decreased growth hormone levels in the blood (Sinha et al., 1973; Stephen et al., 1971). Muller and Pecille (1966) observed fasting caused a decrease in the total content of growth hormone in the anterior pituitary but an increase in the plasma concentration of growth hormone.

Trenkle (1971b) and McAtee and Trenkle (1971) reported that the plasma growth hormone levels were not affected by feeding, fasting or nutrient uptake in sheep and cattle, respectively. In addition, Trenkle (1970) fed high energy rations to fattening steers and reported no effect on plasma growth hormone levels. However, the addition of 10 mg of diethylstilbestrol per day to the ration stimulated plasma growth hormone levels and live weight gains. Hutcheson and Preston (1971) noted an increase of 32% in serum growth hormone when lambs were fed diethylstilbestrol for 14 days. In contrast, Beck et al. (1976) failed to observe any effects of ovarian steroids on growth hormone levels in Holstein heifers. Zeranol, an estrogenic compound, increased serum insulin and growth hormone levels when implanted and not infused into growing wether lambs that were fasted (Olsen et al., 1977). They suggested that Zeranol must be exposed over an extended period of time (up to 3 weeks) to stimulate an increase in serum growth hormone and insulin. Preston (1975) hypothesized that estrogens and estrogenic compounds, when used in ruminants, may cause a release of growth hormone releasing factors from the hypothalamic area. Consequently, there may be an instant release of growth hormone from the anterior pituitary resulting in increased nitrogen retention and growth. One must realize that this is a hypothesis and remains to be accepted or rejected based on more definitive experimental evidence. Davis et al. (1977) suggested that estrogens and androgens exerted their anabolic actions by modulating the episodic secretion of growth hormone.

Contradictory data have been reported on the fluctuation of growth hormone levels during a 24 hr period. Disagreement has arisen as to the nature of the daily secretion pattern. Some investigators have noted a bimodal release (Dunn et al., 1973/1974) while others observed a circadian secretory release of growth hormone (Muller et al., 1970). Anfinson et al. (1975) and Trenkle (1977) have reported an episodic secretion of growth hormone in cattle. The latter investigator observed one to three peaks in plasma growth hormone concentration occurring in each animal during the 5.5-hr sampling period. However, no differences in amplitude or frequency of these peaks were observed. Failure to utilize short sampling intervals and methods have led to equivocal results.

Tannenbaum and Martin (1976) developed a technique for chronic cannulation which allowed frequent blood sampling from unrestrained freely-behaving rats over long periods of time. They reported that the secretion of growth hormone was primarily regulated by an endogenous ultradian rhythm which was not dependent on feeding behavior or serum glucose. In addition, the alternation of dark and light periods probably acted as a cue to entrain the growth hormone secretory rhythm. The light and dark periods were not necessary to maintain the basic ultradian rhythm which had a periodicity of approximately 3.3 hr.

Rabolli and Martin (1977) studied the effects of diet composition on several blood metabolites. Twelve-week old female lean Zucker rats were either fed a high sucrose, high glucose, high protein, high unsaturated fat, high saturated fat or high starch diets. Serum

growth hormone and glucose concentrations were not significantly different among any of the diets. These data suggested that diet composition and heat increment were not influential on the secretion or clearance of growth hormone. In addition, the highest serum insulin levels were obtained from the high sucrose, high glucose and high starch diets.

The effects of dietary alterations on blood insulin levels have been rather consistent among researchers. Trenkle (1966) observed that increasing the energy density of a ration resulted in higher plasma insulin concentrations in sheep. Likewise, Trenkle (1970) reported that plasma insulin levels were related to the amount of concentrate consumed in finishing rations. Bassett et al. (1971) reported that the amount of organic matter digested and the amount of crude protein digested in the intestines were positively correlated (0.74 and 0.74, respectively) with plasma insulin but negatively correlated (-0.62 and -0.63, respectively) with plasma growth hormone levels in sheep. Lower circulating levels of plasma insulin were also observed in low protein fed steers than in the normal fed steers (Borger et al., 1973).

Grigsby et al. (1972) fed a 16% crude protein grower diet to pigs that were fasted for one day. Glucocorticoids were decreased 50% and the level of free fatty acids were reduced within one hr after feeding. Also, blood glucose levels doubled and serum insulin levels increased 17-fold. Chase et al (1977a) found that portal insulin levels in Holstein steers increased within two min of meal initiation and remained elevated during the first 14 min of feeding. Chase et al.

(1977b) measured short-term changes in portal insulin of Holstein steers either given no feed (control), fed ad libitum or restricted fed (½ of the ad libitum amount). Compared to the control, insulin levels increased within 15 min when fed ad libitum and within 5 min when steers had restricted intake. Since the rapid change in insulin preceded changes in other metabolites, Chase et al. (1977a) suggested that the insulin release upon meal initiation appeared to be due to a stimulated vagal reflex rather than a metabolite causing its release from the pancreas.

Glucose is undoubtedly the best effector of insulin release. Machlin et al. (1968) gave glucose either intravenously or orally to pigs that had been fasted for 36 hr. They observed a simultaneous rise in blood glucose and insulin levels. Likewise, Siers and Trenkle (1973) and Davis et al. (1970) reported a positive correlation of 0.54 and 0.95 between glucose and insulin concentrations, respectively. These data indicated there was a strong tendency for the plasma levels of glucose and insulin to fluctuate in the same direction at the same time. Conversely, Machlin et al. (1968; 1970) and Siers and Trenkle (1973) observed a negative relationship between growth hormone and blood glucose. Additional work done by Stern et al. (1971) and Feldman and Jackson (1974) revealed that insulin levels were increased when either the pancreas was perfused with glucose or glucose was infused intravenously.

Many of the amino acids, when infused intravenously to ruminants, can increase serum insulin levels (Stern et al., 1971;

and Davis, 1972). Bassett et al. (1971) reported that valine, isoleucine, phenylalanine and tyrosine were most highly correlated to plasma insulin concentrations in sheep. In addition, Machlin et al. (1968) and Davis (1972) noted that arginine stimulated insulin secretion and leucine and phenylalanine were effective in stimulating insulin release (Bassett et al., 1971; and Davis, 1972).

Although volatile fatty acids have no stimulatory effect on insulin release in non-ruminant species (Horino et al., 1968), propionate and butyrate have been shown to stimulate insulin release but acetate had little stimulatory effect on ruminants (Manns and Boda, 1967; and Horino et al., 1968). Bassett et al. (1971) noted a correlation of 0.51 between plasma insulin concentration and propionate production.

Pharmacologic and not physiologic concentrations of insulin have been shown to significantly provide direct stimulation of growth cartilage (Salmon, 1970). Somatomedin (sulfation factor) had been reported to hormonally direct skeletal growth by stimulating cartilaginous growth (Daughaday et al., 1975). Recent reports by Phillips and Orawski (1977) have stated that insulin was a vital regulator of somatomedin and did contribute to growth via somatomedin.

MATERIALS AND METHODS

Design of Experiment

A 2 x 4 factorial design was employed in a two-year study conducted during the period of mid-November, 1975, to late September, 1977. In each of the two feedlot trials steers of different genetic background and body types were compared to evaluate the role of genetic selection and crossbreeding and ration energy content on bovine growth and muscle development.

Experimental Animals and Rations

One hundred nineteen steers representing four cattle types were utilized in this study. All steers were produced in a beef selection-crossbreeding project developed from the same Hereford herd at the Lake City Experiment Station, Lake City, Michigan. The steers representing the selection-crossbreeding scheme (Table 1) were as follows: unselected Herefords (UH), selected Herefords (SH), Angus x Hereford x Charolais crossbred (AHC) and Angus x Hereford x Holstein crossbred (AHH) steers.

Group 1 or the unselected Herefords (UH) served as the control for the selection-crossbreeding scheme. The first four bull calves born were left as bulls and became sires for the UH group. Consequently, mating was random and no selection pressure was applied.

Table 1. Selection-Crossbreeding Scheme for Experimental Animals

Cattle type[a]	Group	Selection	Mating systems
UH	1	None	Random
SH	2	Yearling weight	Straightbred
AHC	3	Yearling weight	Crossbred
AHH	4	Yearling weight	Crossbred

[a]UH = Unselected Hereford; SH = Selected Hereford; AHC = Angus x Hereford x Charolais; AHH = Angus x Hereford x Holstein.

Bulls used as sires for the SH (group 2), AHC (group 3) and AHH (group 4) were selected from artificial insemination studs primarily on their adjusted yearling weight. Groups 3 and 4 involved a three-breed rotational cross in which each female was mated to a bull from a breed that she was least related to. Female replacement heifers from groups 2, 3 and 4 were selected each year on the basis of their unadjusted yearling weight. Females in group 1 were saved without considering weight but in the same age groups as the replacement heifers in groups 2, 3 and 4 (Magee and McPeake, 1976).

All steer calves were shipped to the Beef Cattle Research Center at Michigan State University in the fall of 1975 and 1976 and were in average condition. To allow recovery from shipment, the calves were full-fed a standard receiving ration consisting of 88% corn silage plus 12% protein-mineral supplement (dry matter basis) for at least 30 days after arrival. According to NRC (1976) recommendations, the ration was

adequate for phosphorus, calcium, vitamins A and D and trace mineralized salt. All steer calves that were sick were treated during this recovery period and all animals were consuming normal amounts of dry matter when the feedlot trials were begun.

Fifty-nine steers were utilized in trial 1 (1975-1976). Steers within each breeding group were stratified by weight and then randomly allotted to one of two ration treatments, resulting in six to eight steers per pen (Table 2). Animals per pen varied due to limited numbers per breeding group. One-half the number of steers within each breeding group were fed either a high grain (20% corn silage and 80% corn grain plus supplement) or a high silage (dry matter averaged 90% corn silage and 10% supplement) ration. Both rations were supplemented with soybean meal at a level calculated for a given cattle type, stage of growth and percentage protein in the bodyweight gain (Table 2).

Sixty steers were utilized in trial 2 (1976-1977) of this experiment. Similar allocation and feeding procedures were employed as outlined in trial 1 (Table 4). Mean nutrient composition of ration ingredients for trials 1 and 2 are presented in Tables 3 and 5, respectively.

Feeding, Weighing and Managerial Procedures

The complete rations were mixed in a horizontal batch mixer and fed once daily. Sufficient ration was fed ad libitum and any unconsumed feed was removed, weighed and recorded. Feed records were maintained daily and samples of all feeds were obtained monthly and

Table 2. Experimental Design and Rations for Feedlot Trial 1 (1975-1976)[a]

Sex	Cattle type[b]	High silage ration: Pen no.	No. anim.	Weight, kg			High grain ration: Pen no.	No. anim.	Weight, kg			
Steers	UH	5	6	To 272	272-318	318+	11	6	To 272	272-318	318-363	363+
	SH	6	7				12	8				
	AHC	9	8	To 318	318-363	363+	13	8	To 318	318-363	363-454	454+
	AHH	10	8				14	8				
Ingredients in ration dry matter, %												
Corn silage				88.0	90.4	92.8			20.0	20.0	20.0	20.0
High moisture corn									67.4	70.3	73.1	76.0
Supplement 174[c]				12.0	9.6	7.2						
Supplement 175[d]									12.6	9.7	6.9	4.0
Percent of dry matter												
Crude protein[e]				13.2	11.7	10.3			14.6	13.5	12.6	11.2
Ca[f]				0.49	0.44	0.40			0.80	0.63	0.47	0.31
P[f]				0.34	0.31	0.29			0.33	0.32	0.31	0.30
Net energy, Mcal/kg[f]												
Maintenance				1.59	1.59	1.58			2.03	2.05	2.07	2.09
Gain				1.02	1.01	1.00			1.33	1.34	1.35	1.36

[a]Harpster (1978).

[b]UH: Unselected Hereford; SH: Selected Hereford; AHC: Angus x Hereford x Charolais; AHH: Angus x Hereford x Holstein. Two steers (pens 5, 10) were discarded during the trial.

[c]Dry matter composition: soybean meal, 92.0%; limestone, 2.2%; defluor. phosphate, 3.3%; trace mineral salt, 2.2%; vitamin A (30,000 IU/g), 0.15%; vitamin D (3,000 IU/g), 0.15%.

[d]Dry matter composition: soybean meal, 81.8%; limestone, 15.6%; trace mineral salt, 2.3%; vitamin A (30,000 IU/g), 0.15%; vitamin D (3,000 IU/g), 0.15%.

[e]Determined by laboratory analysis.

[f]Calculated from average values (NRC, 1976).

Table 3. Nutrient Composition of Ration Ingredients for Feedlot Trial 1[a]

Ingredient	Int. ref. no.	Percent of dry matter				
		Dry matter[b]	Crude protein[b]	Ca[c]	P[c]	K[c]
Corn, aerial pt, w ears, w husks, ensiled, mature, well-eared mx 50% mn 30% dry matter	3-08-153	32.60	7.70	0.28	0.21	0.95
Corn, dent, yellow, grain, gr 2 US	4-02-931	69.80	10.58	0.02	0.35	
Soybean, seeds, meal solv-extd	5-04-604	90.00	53.56	0.36	0.75	2.21
Limestone, grnd	6-02-632	100.00		33.84	0.02	
Phosphate, defluorinated, grnd	6-01-780	100.00		33.07	18.04	0.09
Trace mineral salt		100.00				
Vitamin A[d]		90.00				
Vitamin D[e]		90.00				

[a]Harpster (1978).

[b]Determined by laboratory analysis.

[c]Average nutrient composition (NRC, 1976).

[d]30,000 IU vitamin A per g.

[e]3,000 IU vitamin D_3 per g.

Table 4. Experimental Design and Rations for Feedlot Trial 2 (1976-1977)[a]

Sex	Cattle type[b]	High silage ration					High grain ration				
		Pen no.	No. anim.	Weight, kg			Pen no.	No. anim.	Weight, kg		
Steers	UH	5	6	To 272	272-318	318+	11	6	To 272	272-318	318-363	363+
	SH	6	8				12	8				
	AHC	9	8	To 318	318-363	363+	13	8	To 318	318-363	363-454	454+
	AHH	10	8				14	8				
Ingredients in ration dry matter, %												
Corn silage				86.0	88.4	90.7			20.0	20.0	20.0	20.0
High moisture corn									66.3	69.2	72.2	75.1
Supplement 176[c]				14.0	11.6	9.3						
Supplement 276[d]									13.7	10.8	7.8	4.9
Percent of dry matter												
Crude protein[e]				13.3	12.3	11.2			14.3	13.5	12.5	11.5
Ca[f]				0.50	0.46	0.42			0.69	0.56	0.44	0.31
P[f]				0.36	0.34	0.31			0.34	0.33	0.33	0.32
Net energy, Mcal/kg[f]												
Maintenance				1.60	1.59	1.59			2.02	2.04	2.06	2.08
Gain				1.02	1.02	1.01			1.32	1.33	1.35	1.36

[a]Harpster (1978).

[b]UH: Unselected Hereford; SH: Selected Hereford; AHC: Angus x Hereford x Charolais; AHH: Angus x Hereford x Holstein.

[c]Dry matter composition: soybean meal, 93.2%; limestone, 1.5%; deflour, phosphate, 3.2%; trace mineral salt, 1.8%; vitamin A (30,000 IU/g), 0.15%; vitamin D (3,000 IU/g), 0.15%.

[d]Dry matter composition: soybean meal, 80.3%; limestone, 10.9%; potassium chloride, 6.6%; trace mineral salt, 1.8%; vitamin A (30,000 IU/g), 0.20%; vitamin D (3,000 IU/g), 0.20%.

[e]Determined by laboratory analysis.

[f]Calculated from average values (NRC, 1976).

Table 5. Nutrient Composition of Ration Ingredients for Feedlot Trial 2[a]

Ingredient	Int. ref. no.	Percent of dry matter				
		Dry matter[b]	Crude protein[b]	Ca[c]	P[c]	K[c]
Corn, aerial pt, w ears, w husks, ensiled, mature, well-eared mx 50% mn 30% dry matter	3-08-153	37.90	7.66	0.28	0.21	0.95
Corn, dent, yellow, grain, gr 2 US	4-02-931	72.60	10.60	0.02	0.35	
Soybean, seeds, meal solv-extd	5-04-604	90.00	52.40	0.36	0.75	2.21
Limestone, grnd	6-02-632	100.00		33.84	0.02	
Phosphate, defluorinated, grnd	6-01-780	100.00		33.07	18.04	0.09
Potassium chloride, KCl	6-03-756	100.00				52.44
Trace mineral salt		100.00				
Vitamin A[d]		90.00				
Vitamin D[e]		90.00				

[a]Harpster (1978).

[b]Determined by laboratory analysis.

[c]Average nutrient composition (NRC, 1976).

[d]30,000 IU vitamin A per g.

[e]3,000 IU vitamin D_3 per g.

analyzed for nitrogen and dry matter. Changes in ration dry matter content of ingredients were adjusted at the beginning of each 28-day weigh period.

All steers in both trials were initially implanted with Synovex S and were re-implanted every subsequent 120-day period. Implants were withdrawn prior to slaughter according to FDA regulations.

Initial and final weights for steers in both trials were obtained after a 16 hr shrink without water and feed. Intermediate weights were recorded every 28 days after a 16 hr shrink without water.

All steers in both trials were housed in partially-covered, concrete, bedded pens.

Initial and Terminal Slaughter Animals

All animals, both of the initial and of the terminal slaughter groups, were slaughtered at the Michigan State University Meat Laboratory to facilitate the removal of both the semitendinosus (ST) muscle and the pituitary gland. The comparative slaughter technique was used to compare differences in moisture, fat, protein, nucleic acids, muscle protein fractions in the ST muscle and total growth hormone content in the adenohypophysis.

Combining both trials 1 and 2, there were six steer calves from each of the breeding groups 2, 3, and 4 and five steer calves from group 1 that were selected for the initial slaughter. All steer calves were randomly chosen to be representative of each breeding group at the start of the experiment. Thus, a total of 23 male castrated calves were killed as soon as possible after the start of the trials.

The finished steers fed high grain were slaughtered when they reached an estimated 28 to 32% carcass fat or fat enough to grade low choice. The high silage fed steers were killed at approximately the same weight as those within the same genetic type fed high grain. As a result, finished steers from both trials were marketed in three groups. The first cut contained one-half of the high grain fed steers per genetic type. The second group consisted of the remaining high grain fed steers and one-half of the high silage fed steers per genetic type. The last group slaughtered were the remaining high silage fed steers.

A total of 36 finished steers, nine from each genetic type, were slaughtered at the meat laboratory at the termination of the two feedlot trials. Each genetic type was represented by 4 high grain and 5 high silage fed steers. The remaining steers were trucked to a commercial packing plant where they were slaughtered according to normal procedures. Actual carcass quality and yield grade data on all steers were collected by a government grader. Harpster (1978) determined carcass chemical composition for all initial and terminal slaughter steers, using either 9-10-11 rib cuts or specific gravity determinations.

Of the total 36 steers slaughtered at the termination of both trials, 12 steers were from trial 1 and 24 from trial 2. Steers from each genetic type and ration were represented at each slaughter time.

Collection and Preparation of Samples

Blood Serum

To determine the levels of blood growth hormone and insulin during different stages of growing and finishing, blood samples from all steers were collected on days 1, 29, 57, 113, and 169 of the feedlot trials. These collection periods except on day 1 corresponded to intermediate weigh days. Using a 2.5 cm 18 gauge needle, whole blood was drawn into a clean 20 ml plastic syringe via jugular vein puncture while the animal was haltered and secured in a restraining chute. Blood was then transferred to clean, labelled Corex tubes and allowed to stand at room temperature for 1 hr and then overnight in a coldroom at 4°C. The clot was rimmed and serum was harvested by centrifugation at 2,500 x g for 30 min. The serum was transferred by disposable pasteur pipettes into clean labelled, small test tubes and stored at -20°C until hormone analyses were performed.

Semitendinosus Muscle

Immediately after exsanguination of the animal, the hide was peeled back from the left hindquarter and the ST muscle was removed. The ST muscle was selected because it was large and easily exposed during carcass dressing. Once it had been excised from the carcass, the ST muscle was trimmed free of any adhering lean, fat or connective tissue. The ST muscle was then weighed to the nearest gram and subsampled by alternately taking 4 to 5 cross-sectional slices approximately 2.5 cm thick. These slices were quickly frozen in a mixture

of isopentane and dry ice in a styrofoam container. Muscle slices were transferred by tongs to labelled plastic bags and stored at -70°C.

To assure homogeneity, the muscle slices from each sample were pulverized with dry ice using a Waring Blendor. This entire procedure took place in the walk-in freezer (-30°C) in the meat laboratory. Each powered sample was manually sifted, mixed and transferred to a plastic bag (Whirl Pak, Nasco, Fort Atkinson, Wisconsin). Each bag was left open for 24 hr in the -30°C freezer to allow the dry ice to sublime. Bags were then securely closed and stored at -70°C until analyzed for nucleic acids, moisture, ether extract, protein and muscle protein fractions.

Pituitary Gland

The head of the animal was immediately skinned and severed at the atlas joint after the animal was stunned, hoisted and exsanguinated. With the use of a power saw, the skull cap was removed by sawing approximately 4 cm above the eyes directly back to the top of the occipital condyles. Located under the base of the brain and in the concavity of the sphenoid bone, the pituitary gland was then carefully removed with a scapel and forceps by severing the pituitary stalk and the connective tissue surrounding the hypophysis. The intact pituitary gland was placed in a labelled plastic bag (Whirl Pak, Nasco, Fort Atkinson, Wisconsin) and frozen immediately on dry ice. Intact glands were stored at -70°C until the anterior pituitary was removed and homogenized.

The frozen pituitary gland was exposed to room temperature 10 to 15 min to facilitate the removal of the anterior pituitary from the gland. A longitudinal dissection through the pituitary stalk was made to remove the adenohypophysis from both halves of the pituitary gland. After the anterior pituitary was weighed and the weight recorded, it was cut into smaller pieces to facilitate homogenization. The pieces were transferred to a glass homogenizing tube and 5 ml of cold 0.02 M borate buffer containing 0.5% lauryl sulfate, pH 8 (Appendix Table A.1) were added to extract the growth hormone. This mixture was homogenized on ice with the use of a motorized pestle. The homogenized contents were poured into a 30 ml Pyrex test tube, and the rod and homogenizing tube were washed with an additional 15 ml of borate buffer. To assure a homogeneous mixture, the Pyrex test tube was inverted 4 times and frozen immediately on dry ice. The 20 ml homogenate was stored at -30°C until analyzed for total growth hormone content.

Laboratory Analyses

Proximate Analysis

Each powered sample of ST muscle was analyzed for moisture, crude protein (N x 6.25) and ether extract. All chemical analyses were run in duplicate and duplicate determinations agreed within 4%. Moisture was determined by drying approximately 5 g of fresh, powdered sample in a forced air oven at 100°C for 24 hr. Ether extraction was performed on the dried samples from the moisture determinations using

the Goldfisch apparatus and procedure. Crude protein was determined by weighing approximately 1 g of fresh, powdered sample and employing the Technicon Auto-Kjeldahl System.

Muscle Nucleic Acid Extraction

A modification of the method of Munro and Fleck (1966) was utilized to determine RNA and DNA of the ST muscle. Two samples of fresh, powdered muscle tissue were assayed in duplicate. Duplicate determinations for both RNA and DNA agreed within 10%.

Two ml of cold deionized distilled water were dispensed into glass centrifuge tubes containing approximately 0.3 g fresh, powdered muscle tissue. The tubes were stoppered and vortexed. Five ml of cold 2.5% perchloric acid (PCA) were added to all tubes. Tubes were vortexed, placed in ice for 10 min, vortexed again and centrifuged at 34,800 x g for 15 min. The supernatant was decanted and discarded. The pellet was broken apart with a wooden applicator stick and 5 ml of cold 1.0% PCA were dispensed. All tubes were vortexed and centrifuged at 34,800 x g for 15 min. Supernatants were again discarded and pellets were broken up. A 5 ml aliquot of cold 1.0% PCA was added a second time. Tubes were vortexed and centrifuged as previously described.

After the second centrifugation using 1.0% PCA, the supernatant was discarded, the pellet was broken apart, 4 ml of 0.3 N potassium hydroxide were added, and the tubes were vortexed gently. The tubes were incubated for 2 hr at 37°C in a water bath. A marble was placed on top of each tube and the tubes were agitated frequently during digestion of muscle tissue. The tubes were removed from the water

bath at the end of 2 hr and then placed in ice for at least 5 min. Five ml of cold 5.0% PCA were added, and the tubes were vortexed and placed in ice for 15 min after which the tubes were vortexed again and centrifuged at 34,800 x g for 10 min. The supernatant was removed and transferred to 25 ml graduated tubes and saved. The pellet was broken up and washed twice with 5 ml of cold 5.0% PCA. The tubes were vortexed and centrifuged at 34,800 x g for 10 min each time. The pellet washings were added to the 25 ml graduated tubes containing the original supernatant. Volume was brought to 20 ml with 5.0% PCA. This supernatant fraction represented the RNA and was saved. Pellets were also saved for DNA extraction.

DNA extraction commenced by breaking up the pellet as previously described and 5 ml of 10% PCA were added to each tube. It is important to note that this is a convenient stopping place for overnight storage. The extraction of DNA was accomplished by incubating the pellet for 25 min in a 70°C water bath. A marble was placed on top of each tube during the digestion period and samples were agitated frequently. After digestion, the sample tubes were vortexed and placed in ice. When cold, the tubes were centrifuged at 34,800 x g for 15 min. The supernatant was decanted into 10 ml calibrated tubes. The pellet was broken up and washed with 4.8 ml of cold 10% PCA, vortexed and centrifuged for 15 min at 34,800 x g. The washing suspension was added to the original supernatant in the 10 ml calibrated tube. The volume was brought up to 10 ml with 10% PCA. This fraction was saved and represented the DNA supernatant. The pellet was then discarded.

Muscle Nucleic Acid Determination

RNA concentration was determined by a colorimetric procedure utilizing orcinol (Mejbaum, 1939). A 2 ml aliquot of the 20 ml RNA supernatant was run in duplicate and was pipetted into test tubes. Two reagent blanks containing 2 ml of 5.0% PCA in lieu of sample and RNA standards of 6.25, 12.5, 18.75, 25.0 and 37.5 mg/ml were used for deriving a standard curve. Two ml of a 1.0% orcinol reagent (Appendix Table A.2) were dispensed into all tubes and mixed. A marble was placed on each tube and the tubes were incubated in a boiling water bath for 30 min. After boiling, all tubes were cooled in running cold water and allowed to equilibrate to room temperature. Optical densities were determined on all tubes using a Gilford Spectrophotometer at a wavelength of 680 nm.

Diphenylamine and acetaldehyde were utilized (Burton, 1956; 1968) to colorimetrically determine DNA concentration. Two ml aliquots of the 10 ml DNA fraction were run in duplicate and were pipetted into test tubes. Two reagent blanks containing 2 ml of 10% PCA instead of sample and DNA standards of 6.25, 12.5, 18.75 and 25.0 mg/ml were used to prepare a standard curve. Two ml of 4.0% diphenylamine in glacial acetic acid (Appendix Table A.3) and 0.1 ml of acetaldehyde solution (Appendix Table A.4) were added to all tubes and then each was mixed. Marbles were placed on each of the tubes and samples were incubated for 16 hr at 30°C in a water bath. Tubes were removed from the water bath and cooled to room temperature. Optical densities were determined on all tubes using a Gilford Spectrophotometer at a wavelength of 595 nm.

Extraction of Muscle Protein Fractions

The extraction procedure for muscle protein fractionation was similar to the Borton (1969) modification of the Helander (1957) procedure. All extractions were done at 4°C. Each powdered muscle sample and analyses were run in duplicate. Duplicate determinations agreed within 10%.

Approximately 5 g of frozen powdered muscle tissue were weighed into a 250 ml centrifuge bottle. Fifty ml of 0.015 M phosphate buffer, pH 7.5 (Appendix Table A.5), were dispensed into each bottle containing both muscle sample and a magnetic stirrer. Samples were placed on an electric, magnetic rotator and allowed to gently agitate for 3 hr. After the low ionic strength extraction, bottles were centrifuged for 20 min at 1,465 x g with the GSA (Sorvall Inc.) rotor. Supernatants were carefully filtered through 8 layers of cheese cloth into a 100 ml graduated cylinder. The pellet was resuspended in an additional 50 ml of 0.015 M phosphate buffer, pH 7.5, and the extraction procedure was repeated exactly as previously described. After the second centrifugation, the supernatant was filtered and combined with the original supernatant. Cheese cloth was then squeezed and any additional supernatant was collected in the graduated cylinder. Total volume was recorded and fraction I (Figure 1) represented the sarcoplasmic protein nitrogen fraction. The pellet was saved for further extraction. It is important to note that this is a convenient stopping place for overnight storage.

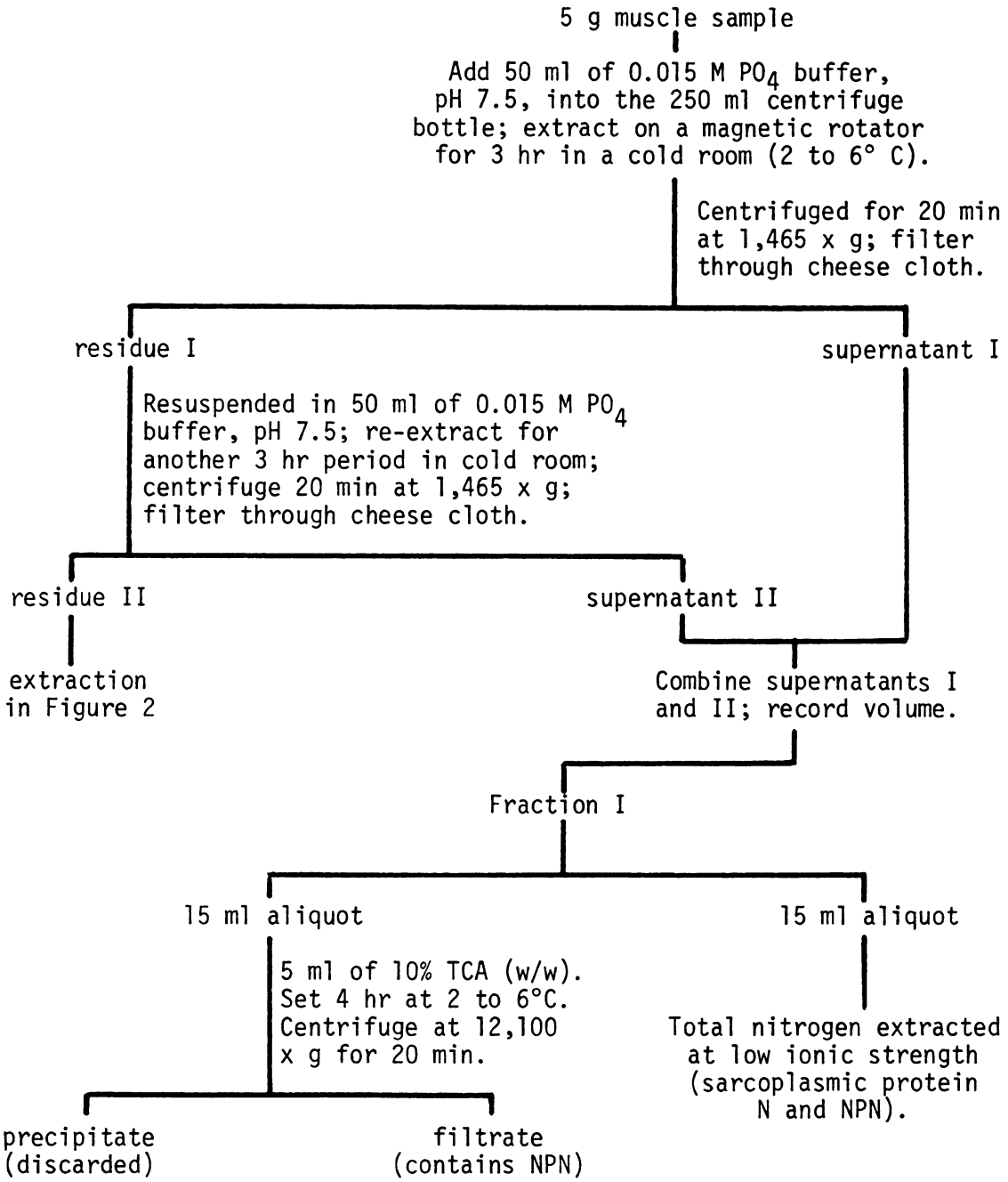


Figure 1. Scheme for sarcoplasmic protein nitrogen and non-protein nitrogen fractionations.

The residue or pellet left from fraction I was resuspended in 50 ml of 1.1 M potassium iodide, 0.1 M phosphate buffer, pH 7.5 (Appendix Table A.6). The extraction of muscle protein nitrogen in the high ionic strength buffer continued for 3 hr on the electric, magnetic rotator. Samples were centrifuged at 1,465 x g with GSA rotor for 20 min. Supernatant was carefully filtered through 8 layers of cheesecloth into a 100 ml graduated cylinder. An additional 50 ml of 1.1 M potassium iodide, 0.1 M phosphate buffer, pH 7.5 were added to resuspend the pellet. The exact same sequence of steps were followed as previously described using the high ionic strength buffer. The supernatant obtained from the second centrifugation was filtered and combined with the original supernatant from the high ionic strength extraction. Cheese cloth was squeezed and any additional supernatant was collected in the 100 ml graduated cylinder. Total volume was recorded and fraction II (Figure 2) represented the fibrillar protein nitrogen fraction. The pellet was discarded after the removal of this fraction.

Determination of Nitrogen Distribution in Muscle Protein Fractions

Total crude protein (N x 6.25) for each muscle sample had previously been determined as described earlier in the proximate analysis procedure.

Fraction I in Figure 1 contained both the non-protein nitrogen (NPN) and the sarcoplasmic protein nitrogen. A 15 ml aliquot was taken from fraction I and 5 ml of 10% (w/w) trichloroacetic acid (TCA) were

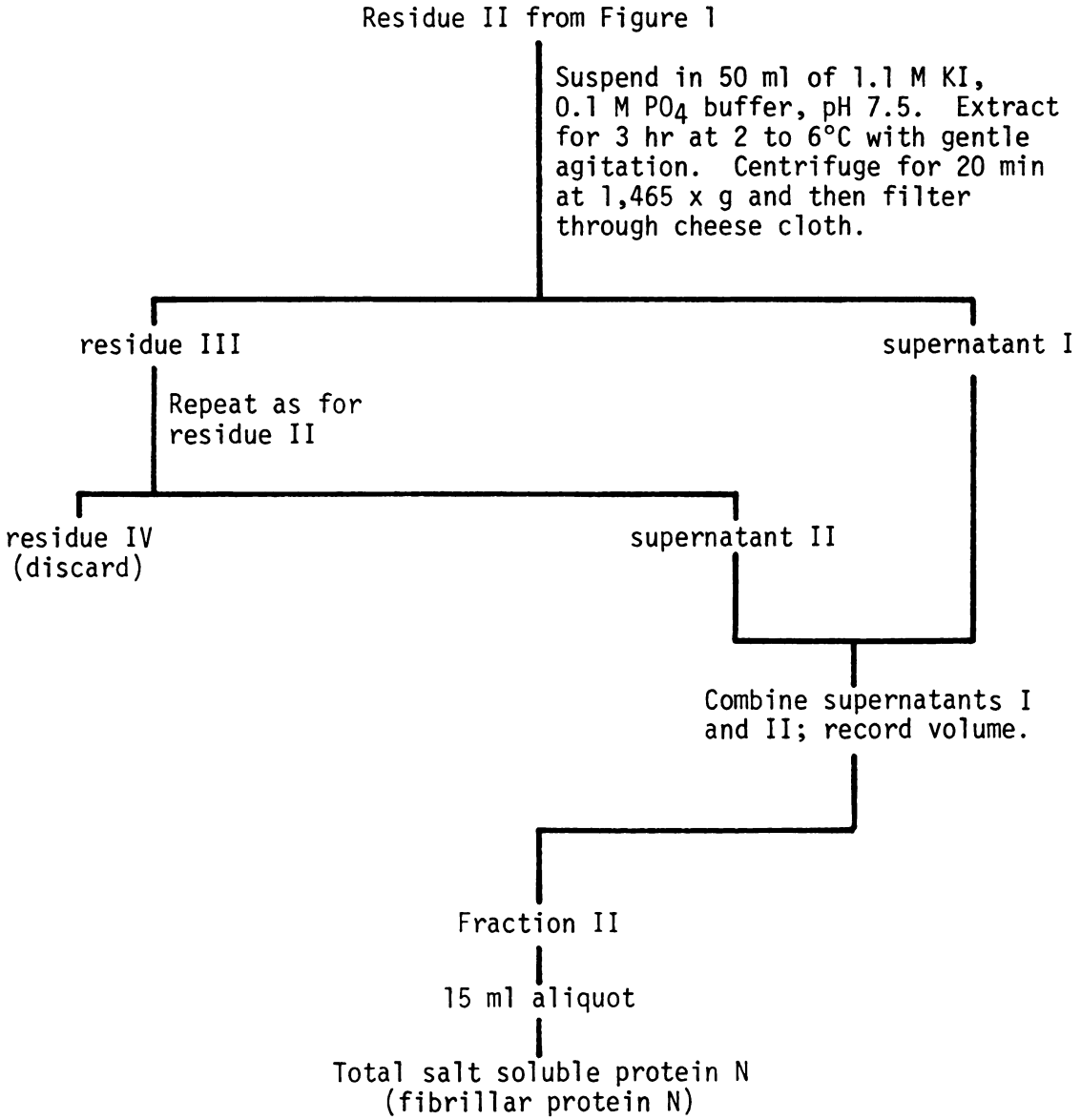


Figure 2. Scheme for fibrillar protein nitrogen fractionation.

added to the aliquot to precipitate the protein. The contents were gently mixed together and kept at 4°C for 4 hr. The tubes were centrifuged at 12,100 x g for 20 min. The filtrate was used for nitrogen analysis and the result recorded as the NPN. All nitrogen analyses were determined by the micro-Kjeldahl procedure as outlined by the American Instrument Company (1961).

Another 15 ml aliquot was taken from fraction I (Figure 1) to determine the total nitrogen extracted in the low ionic strength buffer. The amount of NPN was subtracted from the amount of nitrogen in fraction I with the remainder recorded as the sarcoplasmic protein nitrogen. A 15 ml aliquot was taken from fraction II and used for nitrogen analysis (Figure 2). The result was recorded as the amount of fibrillar protein nitrogen. Stroma nitrogen was found by subtracting the amount of nitrogen in fractions I and II from the total nitrogen in the powdered muscle sample.

Insulin Determination

Serum insulin concentration was determined by using the double antibody radioimmunoassay technique described by Grigsby (1973). Composition of all reagents used are shown in Appendix Table A.7. The assay used guinea pig antibovine insulin serum (GPABI) and sheep anti-guinea pig gamma globulin (SAGPGG) to form an insoluble complex which is precipitated when centrifuged for 30 min at 2,500 x g.

Disposable culture tubes (12 x 75 mm, Scientific Products, McGaw Park, Illinois) were used for all samples and standards. Serum samples described earlier in the collection and preparation of blood

serum samples were thawed to room temperature just prior to use and kept cold (4°C) thereafter until sampling was completed. The pipetting and diluting of samples and standards were accomplished using an automatic pipette (Micromedic Systems Inc., Philadelphia, Pennsylvania).

Standards were prepared from highly purified bovine insulin (Eli Lilly and Company, Indianapolis, Indiana, lot 795372, 24.2 units/mg) with 100 µl of each standard containing 0.1, 0.2, 0.3, 0.4, 0.6, 0.8, 1.0, 1.2, 1.6, 2.0, 2.5 and 3.0 ng of insulin.

In preparation for the insulin assay either 150 or 250 µl of serum sample were pipetted and dispensed simultaneously with 350 or 250 µl of 0.05 M phosphate-buffered saline, 1% bovine serum albumin (PBS-BSA), pH 7.4, into each tube (for a total of 500 µl per tube). Four hundred µl of PBS-BSA were simultaneously added to each 100 µl of bovine insulin standard. Assay tubes 1 and 2 (non-specific binding or background tubes) received 500 µl of the above buffer while tubes 3 and 4 (total count tubes) will contain only 100 µl of ^{125}I-insulin.

On the first day of the assay, 200 µl of GPABI diluted 1:105,000 in normal guinea pig serum (NGPS) were added to each tube (except background and total count tubes). Tubes 1 and 2 received 200 µl of 1:400 NGPS. All assay tubes were gently vortexed and incubated at 4°C for 24 hr.

On day 2, 100 µl of ^{125}I-insulin containing approximately 15,000 cpm were added to each assay tube, gently vortexed and incubated for 24 hr at 4°C.

After this incubation 200 µl of SAGPGG were pipetted to all tubes (except total counts), gently mixed and incubated at 4°C for 96 hr.

On the sixth day, 3 ml of 0.05 M phosphate-buffered saline (PBS) were added to all tubes (except total counts) and centrifuged for 30 min at 2,500 x g in a Sorvall RC-3 refrigerated centrifuge with a swinging bucket rotor. The supernatants were then decanted and the tubes were inverted on absorbant paper for 30 min. Any excess liquid was wiped from the upper half of the tube and then the precipitate was counted for 4 min or 4,000 counts, whichever came first, in a Nuclear-Chicago Model 43-4230 autogamma scintillation counter. The counting time and tube number were simultaneously recorded on a Texas Instrument electromagnetic cassette tape which was mounted onto a Texas Instrument Silent 700 ASR terminal. The cassette tape was later inserted in the terminal for the CDC 6500 computer for calculating unknown insulin concentrations and regression coefficients for the standard curve. The computer program corrected for sample dilution and automatically calculated insulin values of unknown sera.

Growth Hormone Determination

The double antibody radioimmunoassay technique described by Purchas (1970) was utilized for the serum and pituitary growth hormone determinations. The same equipment, materials and reagents were used as previously described in the insulin determination procedure. A standard curve was constructed from standards prepared from NIH-GH-B 2.

One hundred µl of each standard contained 0.1, 0.3, 0.4, 0.5, 0.6, 0.7, 0.8, 1.0, 1.5, 2.0, 2.5, 3.0, 4.0 and 5.0 ng of growth hormone.

One hundred or 150 µl of serum sample were pipetted and dispensed simultaneously with 400 or 350 µl of PBS-1%-BSA into each tube (for a total of 500 µl/tube). Both standards and serum samples were handled in the manner described previously for the preparation of the insulin assay.

On day one, 200 µl of guinea pig antibovine growth hormone serum (GPABGH) diluted 1:3200 were added to each tube (except background and total count tubes), gently vortexed and incubated at 4°C for 24 hr.

On the second day, 100 µl of ^{125}I-GH containing 20,000 cpm were added to each tube, gently vortexed and incubated at 4°C for 24 hr.

On day 3, 200 µl of SAGPGG were added to all tubes (except total counts), gently vortexed and incubated for an additional 72 hr at 4°C.

At the end of the 72 hr incubation period, additions and handling procedures were identical with those described previously for the insulin radioimmunoassay. The calculation of results was also identical to the previous method described for the insulin assay.

Isolation of Myofibrils for Electrophoresis

Five g of frozen muscle tissue were weighed into a 250 ml centrifuge bottle and were suspended in 10 volumes (v/w) of cold 0.25 M sucrose, 0.05 M Tris, 1 mM EDTA, pH 7.6 (Appendix Table A.8).

The slurry was gently mixed and centrifuged at 1,000 x g for 10 min. The supernatant was decanted along with a small, normal, fat layer. If a thick, heavy, fat layer appeared on top, it was saved and resuspended with the sediment because a considerable quantity of trapped myofibrils may be contained in the heavy, fat layer.

The sedimented myofibrils were resuspended in 5 volumes (v/w) of cold 0.25 M sucrose, 0.05 M Tris, 1 mM EDTA, pH 7.6 and centrifuged at 1,000 x g for 10 min. The supernatant was decanted and 5 volumes (v/w) of cold 0.05 M Tris, 1 mM EDTA, pH 7.6 (Appendix Table A.9) were added to resuspend the pellet of sedimented myofibrils. The contents were then centrifuged for 10 min at 1,000 x g.

The supernatant was again decanted and the sedimented myofibrils were redissolved twice in 5 volumes of cold 100 mM KCL, 1% Triton X-100 (Appendix Table A.10). The mixture was centrifuged at 1,000 x g for 10 min.

After the second centrifugation, the supernatant was decanted and 5 volumes of cold 100 mM KCL (Appendix Table A.11) were added twice to resuspend the pellet. Each centrifugation lasted for 10 min at 1,000 x g.

After the second centrifugation using the 100 mM KCL, 25 ml of 50 mM KCL, 1 mM sodium azide (Appendix Table A.12) were dispensed into each centrifuge bottle to redissolve the pellet of sedimented myofibrils. Samples were refrigerated and stored at 4°C until protein concentrations of the samples were determined by a modification of the Lowry method (Hartree, 1972).

Sodium Dodecyl Sulfate (SDS)-Polyacrylamide Gel Electrophoresis of Myofibrils

Myofibrils were dissolved in 50 to 100 µl of 1.0% SDS, 0.05 M Tris-HCL, pH 7.1, 20% glycerol, 0.5% mercaptoethanol and 0.01% Pyronin Y (Appendix Table A.13) by heating at 80°C for 20 min. A Hamilton syringe was used to quantitatively load the dissolved samples onto the 8 cm, 10% polyacrylamide gels. Gels were electrophoresed in a chamber buffer consisting of 0.1% SDS, 0.2 M Tris-glycine, pH 8.8 (Appendix Table A.14) at a voltage of 0.5 milliamps per gel tube. A 0.033% Coomassie brilliant blue R250 solution (Appendix Table A.15) was used to stain the gels overnight. A mixture of H_2O:glacial acetic acid:methanol (87.5: 7.5:5 v/v; Appendix Table A.16) destained the gel background.

Preparation of Pituitary Extract

The frozen 20 ml homogenate was thawed and made up to a concentration of 30 mg/ml with additional cold 0.02 M borate buffer, 0.5% lauryl sulfate, pH 8 (Appendix Table A.1). The resulting homogenate was centrifuged at approximately 1,200 x g for 6 min. For the assay of the 30 mg/ml pituitary extracts, a 1:10,000 dilution was made to give a final concentration of 3 µg/ml. This was accomplished by an initial 1 to 100 dilution with PBS (Appendix Table A.7 [E]) and a second 1 to 100 dilution using PBS-1%-BSA (Appendix Table A.7 [F]) as the diluent. Ten, 25, and 50 µl aliquants of the 3 µg/ml solution were assayed for each pituitary.

Statistical Analysis

All data were analyzed on the CDC 6500 computer at the Michigan State University Computer Laboratory. A least squares analysis using a three-way analysis of variance with unequal numbers was used to estimate main effects and interactions for ration and cattle type (Harvey, 1960). When significant differences were observed by least squares analysis, the Tukey's ω test was utilized to determine the group means differing from each other (Steele and Torrie, 1960). Group mean separation was performed on the 9825A Hewlett-Packard calculator. Significant differences in the simple correlation coefficients were determined as described by Snedecor (1946).

RESULTS

Feedlot Performance

Table 6 depicts the feedlot performance of steers fed HS and HG rations. The UH steers fed HS or HG rations had lower average daily gains than the SH, AHC and AHH cattle types. Holstein crossbred steers consumed more dry matter and were less efficient ($P < .01$) than the UH, SH and AHC steers (Harpster, 1978). Additionally, steers within a given type fed HS gained at a slower rate and consumed more dry matter per unit of gain than their counterparts fed HG. Averaged across all cattle types, the HG fed steers gained 26% faster than the HS fed steers (1.33 vs 0.98 kg, respectively). Differences in daily gain among the three beef breed types were consistent with frame size differences.

Initial Slaughter Steers

Twenty-three steer calves from both trials 1 and 2 were slaughtered initially to obtain initial muscle and anterior pituitary data. Ten steer calves (2 to 3 per cattle type) were killed at the beginning of trial 1, whereas 13 steer calves (3 to 4 per cattle type) were slaughtered at the beginning of trial 2. Individual semitendinosus (ST) muscle and anterior pituitary data are presented in Appendix Tables B.5 and B.11, respectively, for all steer calves slaughtered initially. Initial muscle protein fractionation and anterior pituitary data from trial 1 are missing due to freezer malfunction.

Table 6. Effect of Ration Energy Level and Cattle Type on Average Daily Gain and Feed Efficiency of Various Types of Finished Steers

Cattle[a] type	Ration energy level					
	High silage			High grain		
	Final weight, kg[b]	ADG, kg	F/G	Final weight, kg[b]	ADG, kg	F/G
UH	431	0.88	7.73	471	1.28	5.25
SH	490	0.96	7.98	516	1.35	5.46
AHC	541	1.04	7.99	576	1.40	5.87
AHH	565	1.05	8.25	564	1.30	6.50

[a]UH = Unselected Hereford; SH = Selected Hereford; AHC = Angus x Hereford x Charolais; AHH = Angus x Hereford x Holstein.

[b]Final shrunk weights were adjusted to the average dressing percent (61.7) of all steers.

Semitendinosus Muscle Data

Moisture, Fat, Protein and Ash

The proximate analysis data from the initial slaughtered steers are presented in Table 7. The moisture content of the ST muscle comprised approximately three-fourths of the fresh tissue weight. The protein percentage of the ST muscle was constant (20.3 to 20.9%) among steer types. Since the ST muscle was trimmed free of any adhering lean from other muscles, fat or connective tissue, the percentage of fat represented in Table 7 was primarily a reflection of the amount of intramuscular fat present. Fat comprised a very small percentage (1.6 to 2.0% among the 4 steer types) of the fresh ST weight. Ash percentages, which were found by difference, were relatively constant (1.7 to 1.9%) across steer types.

The muscle gross composition data from the terminal slaughtered steers are shown in Table 8. Based on an estimated degree of fatness sufficient for the U.S. Choice grade, the steers in both trials 1 and 2 were slaughtered in three groups and the number of days on feed varied depending on the type of ration fed (Appendix Table B.6). The data presented in Table 8 revealed that there were no significant differences in the least square means between ration and among cattle types. In general, the steers fed HG had ST muscles that were, on the average, 1.2% lower in moisture and 1.1% higher in fat than their counterparts fed HS. Steers fed HG averaged 73 fewer days on feed than those fed HS. There were essentially no differences in either the protein or the ash percentages between the two rations.

Table 7. Mean Proximate Analysis, Nucleic Acids and Protein Fractionation Data of Semitendinosus Muscle from Initial Slaughter Steers (Trials 1 and 2)

Cattle[a] type	No. of animals	ST[b] wt, kg	Proximate analysis %, fresh tissue basis				Nucleic acids mg/fresh tissue		Protein Fractionation[c] g total protein/fresh tissue			
			Moisture	Protein	Fat	Ash	DNA	RNA	Sarco-plasmic	Myo-fibrillar	Stroma	NPN
UH	5	0.67	76.4	20.3	1.6	1.7	253.0	899.8	23.8	71.9	21.1	16.4
SH	6	0.80	75.5	20.6	2.0	1.9	288.6	1,036.7	33.1	86.8	25.8	21.0
AHC	6	1.08	75.5	20.9	1.7	1.9	387.7	1,278.7	39.7	115.6	32.3	27.0
AHH	6	1.07	75.8	20.7	1.8	1.7	392.4	1,317.8	43.4	118.7	28.2	25.2

[a]UH = Unselected Hereford; SH = Selected Hereford; AHC = Angus x Hereford x Charolais; AHH = Angus x Hereford x Holstein.

[b]Weight of semitendinosus muscle.

[c]Three, three, three and four animals/mean for the UH, SH, AHC and AHH cattle types, respectively.

Table 8. Effects of Ration and Cattle Type on the Proximate Analysis of the Semitendinosus Muscle of Finished Steers (Terminal Slaughter) Fed to a Constant Compositional Endpoint[a]

Item	Ration[b]		Cattle type[c]				EMS[d]
	HG	HS	UH	SH	AHC	AHH	
	---------- %, Fresh tissue basis ----------						
Moisture	71.9	73.1	72.6	72.7	72.7	71.9	5.3
Protein	21.0	21.2	21.3	20.9	21.2	21.1	0.6
Fat	6.0	4.9	5.3	5.3	5.1	6.0	7.0
Ash	1.1	0.8	0.8	1.1	1.0	1.0	0.5

[a]Least square means.

[b]HG = high grain (16 animals/mean); HS = high silage (20 animals/mean).

[c]UH = Unselected Hereford; SH = Selected Hereford; AHC = Angus x Hereford x Charolais; AHH = Angus x Hereford x Holstein; nine animals/mean for each cattle type.

[d]EMS = error mean square.

The percentage of water in fresh ST tissue decreased initially from 75 to 76% among the four cattle types (Table 7) to terminal values of 72 to 73% (Table 8). This 3 to 4% decrease in moisture was replaced by a corresponding 3 to 4% increase in intramuscular fat. Although differences were small, the ST muscles from the Holstein crossbred steers were 15% fatter (6.0 vs 5.1%) than those from AHC steers and 12% fatter (6.0 vs 5.3%) than those from UH and SH steers. The percentage of protein increased slightly with advancing age, whereas the ash percentage tended to follow the trend observed for the water content.

Nucleic Acids, Total Protein and Semitendinosus Weight

The average DNA concentrations of the ST muscles from the UH, SH, AHC and AHH steers that were slaughtered initially were 0.39, 0.36, 0.37 and 0.37 mg/g of fresh tissue, respectively. The average RNA concentrations from the same steers were 1.37, 1.32, 1.20 and 1.23 mg/g of fresh tissue (Appendix Table B.5) Average DNA and RNA concentrations were lower in the terminal slaughter steers. Concentration values from the finished steers were 0.28, 0.28, 0.31 and 0.31 mg DNA/g of fresh tissue and 0.79, 0.93, 0.95 and 0.88 mg RNA/g of fresh tissue from the respective UH, SH, AHC and AHH cattle types (Appendix Table B.6). Steers from the UH group had the most dramatic decline in both DNA and RNA concentrations of ST muscle.

Total DNA and RNA data from the initial slaughter steers are presented in Table 7. Total DNA and RNA among all cattle types tended to follow the same pattern observed for the weight of the ST muscle.

The AHC and AHH crossbred groups averaged 119 and 330 more mg of DNA and RNA/muscle, respectively, than the averaged UH and SH groups. Since the UH group had the lightest ST weight, they also had the lowest total DNA and RNA contents.

There was no effect of ration on either the total RNA or DNA content/muscle from terminal slaughter steers (Table 9). However, steers fed HS rations had 9% more total RNA/muscle but 8% less total DNA/muscle than steers fed HG rations. Consequently, the HS fed steers had an 18% advantage ($P = .07$) in the RNA/DNA ratio over their counterparts fed HG.

Since the RNA/DNA ratio is a good index of the available synthetic machinery capable of synthesizing protein within a cell, the 18% increase in the RNA/DNA ratio for HS fed steers most likely contributed to a significant ($P < .05$) 10% increase in the total protein content of the ST muscle from steers fed HS (Table 10). Steers fed HG rations had significantly ($P < .05$) lighter ST weights (Table 10) and significantly ($P < .01$) lower protein/DNA ratios than steers fed HS (Table 9).

There was a significant effect of cattle type on total RNA ($P < .013$), total DNA ($P < .005$), total protein content ($P < .005$) and ST weight ($P < .005$). Tables 9 and 10 revealed that the AHC steers had significantly ($P < .01$) more total RNA/muscle than the UH steers. Additionally, the Charolais crossbred steers had heavier ($P < .01$) ST muscles and possessed significantly ($P < .01$) more total protein and total DNA/muscle than either the UH or SH steers. Also, the ST muscles from the

Table 9. Effects of Ration and Cattle Type on the Cellular Components of the Semitendinosus Muscle of Finished Steers (Terminal Slaughter) Fed to a Constant Compositional Endpoint[a]

	Ration[b]		Cattle type[c]				
Item	HG	HS	UH	SH	AHC	AHH	EMS[f]
RNA (mg/muscle)	1,707.5	1,870.7	1,306.8[d]	1,797.4[de]	2,252.6[e]	1,799.6[de]	309,987.2
DNA (mg/muscle)	614.8	568.1	465.6[dD]	545.3[d]	721.2[e]	633.8[deE]	11,597.9
RNA/DNA	2.8	3.4	2.9	3.3	3.1	3.0	0.8
Protein/DNA	657.8[d]	805.7[e]	774.9	754.6	699.4	697.8	19,589.0

[a]Least square means.

[b]HG = high grain (16 animals/mean); HS = high silage (20 animals/mean).

[c]UH = Unselected Hereford; SH = Selected Hereford; AHC = Angus x Hereford x Charolais; AHH = Angus x Hereford x Holstein; nine animals/mean for each cattle type.

[de]Means in rows differing in superscripts differ significantly, $P < .01$; [DE]$P < .05$.

[f]EMS = error mean square.

Table 10. Effects of Ration and Cattle Type on the Total Protein Content and Weight of the Semitendinosus Muscle of Finished Steers (Terminal Slaughter) Fed to a Constant Compositional Endpoint[a]

	Ration[b]		Cattle type[c]				
Muscle Item	HG	HS	UH	SH	AHC	AHH	EMS[g]
Total protein content, g	399.1^{D}	441.8^{E}	353.3^{dD}	400.2^{d}	497.4^{e}	431.0^{deE}	2,979.6
Weight, kg	1.90^{D}	2.08^{E}	1.66^{d}	1.92^{df}	2.35^{e}	2.05^{ef}	0.06

[a]Least square means.

[b]HG = high grain (16 animals/mean); HS = high silage (20 animals/mean).

[c]UH = Unselected Hereford; SH = Selected Hereford; AHC = Angus x Hereford x Charolais; AHH = Angus x Hereford x Holstein; nine animals/mean for each cattle type.

[def]Means in rows differing in superscripts differ significantly, $P < .01$; [DE]$P < .05$.

[g]EMS = error mean square.

AHH steers were heavier ($P < .01$) and had significantly ($P < .05$) more total protein and total DNA/muscle than the UH steers. No significant effect of cattle type on the RNA/DNA or the protein/DNA ratios was observed among steers slaughtered terminally. However, the comparison of data in Table 11 with that of Table 9 indicated that the RNA/DNA ratio was higher in the initial slaughter steers, whereas the protein/DNA ratio among each cattle type made a substantial increase in the finished steers.

Table 11. Mean RNA/DNA and Protein/DNA Ratios of Semitendinosus Muscle from Initial Slaughter Steers (Trials 1 and 2)

Cattle[a] type	No. of animals	RNA/DNA	Protein/DNA
UH	5	3.6	536.8
SH	6	3.6	572.9
AHC	6	3.3	578.9
AHH	6	3.4	556.3

[a]UH = Unselected Hereford; SH = Selected Hereford; AHC = Angus x Hereford x Charolais; AHH = Angus x Hereford x Holstein.

Protein Fractionation

The data containing the percentage of total protein nitrogen in the ST muscles of the initial and terminal slaughter steers are presented in Appendix Tables B.5 and B.6, respectively. Due to a malfunctioning freezer, muscle samples from trial 1 were lost before any protein fractionation analyses were performed. Consequently, there are missing data in both tables.

Approximately 54% of the total protein nitrogen present in the ST muscles of steers slaughtered initially was contained in the myofibrillar fraction but this percentage decreased slightly to 51% found in terminal slaughter steers (Appendix Tables B.5 and B.6, respectively). The protein nitrogen present in the sarcoplasmic fraction increased from 19 to 23% as age increased, whereas the stroma protein nitrogen remained relatively constant between the initial (15%) and the terminal (16%) kills. The non-protein nitrogen decreased slightly from 12% initially to 10% terminally.

The protein fractionation data, expressed on a total protein basis, are presented in Tables 7 and 12 for the initial and terminal slaughter steers, respectively. Comparing all cattle types, the UH steers in Table 7 consistently had the lowest protein content in each of the muscle protein fractions. The larger-framed AHC and AHH cattle types had more total protein in each muscle protein fraction than the smaller-framed UH and SH steers.

Ration had no significant effect on the distribution of protein in the various protein fractions (Table 12). However, the stroma fraction between the HS and HG fed steers was approaching significance ($P < .055$).

The effect of cattle type on the distribution of protein in the sarcoplasmic, myofibrillar and stroma fractions was significant ($P < .045$, $P < .002$ and $P < .037$, respectively). The Charolais crossbred steers had significantly more sarcoplasmic, myofibrillar and stroma protein than the UH steers. Additionally, the ST muscles from AHC

Table 12. Effects of Ration and Cattle Type on the Distribution of Protein in Various Protein Fractions of the Semitendinosus Muscle of Finished Steers (Terminal Slaughter) Fed to a Constant Compositional Endpoint[a]

Fraction	Ration[b]		Cattle type[c]				EMS[f]
	HG	HS	UH	SH	AHC	AHH	
	g total protein/fresh muscle						
Sarcoplasmic	94.2	103.5	79.8^{D}	95.6^{DE}	110.8^{E}	109.3^{DE}	446.0
Myofibrillar	212.6	218.3	181.0^{d}	203.7^{deD}	253.2^{eE}	223.7^{de}	909.3
Stroma	62.8	75.6	57.7^{D}	61.2^{DE}	81.7^{E}	76.4^{DE}	271.5
NPN	43.2	40.0	37.6	35.3	52.9	40.6	209.9

[a]Least square means.

[b]HG = high grain (12 animals/mean); HS = high silage (16 animals/mean).

[c]UH = Unselected Hereford; SH = Selected Hereford; AHC = Angus x Hereford x Charolais; AHH = Angus x Hereford x Holstein; seven animals/mean for each cattle type.

[de]Means in rows differing in superscripts differ significantly, $P < .01$; [DE]$P < .05$.

[f]EMS = error mean square.

steers contained 20% more ($P < .05$) myofibrillar protein than the muscles from SH steers. No significant interactions between ration and cattle type were observed in Table 12.

SDS-Polyacrylamide Gel Electrophoresis

Aliquots of myofibril preparations of the ST muscle from each of the four cattle types were electrophoresed on 10% SDS-polyacrylamide gels as explained in Materials and Methods. Figure 3 illustrates that the synthesis of the myofibrillar proteins is similar within each cattle type. Also, there was essentially little difference in the isolation and characterization of the proteins within each gel. Each of the four gels was selected to be representative of each cattle type and represented steers that were slaughtered initially.

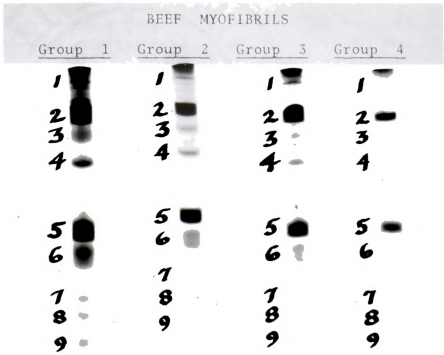
Blood Data

Serum Growth Hormone

Individual serum insulin and growth hormone data from all steers used in trials 1 and 2 are presented in Appendix Tables B.1 through B.4. Mean serum growth hormone levels in steers fed either a HS or a HG ration are graphically presented by days on feed in Figures 4 and 5, respectively. Blood samples from all steers within each cattle type were drawn on days 1, 29, 57, 113 and 169 of the feedlot trial. Serum growth hormone levels in steers fed HG (Figure 5) fluctuated more erratically throughout the feedlot period than in steers fed HS (Figure 4). Serum growth hormone levels in HG fed steers

Figure 3. SDS-polyacrylamide gel electrophoresis of myofibril preparations of ST muscle from Unselected Hereford (Group 1), Selected Hereford (Group 2), Angus x Hereford x Charolais (Group 3) and Angus x Hereford x Holstein (Group 4) steers slaughtered initially. The bands for each gel are numbered 1 through 9 and proteins are labeled according to Porzio and Pearson (1977).

Band	Protein
1	unidentified
2	myosin
3	C-protein
4	α-actinin
5	actin
6	troponin-T and tropomyosin
7	myosin light chain--1
8	myosin light chain--2
9	myosin light chain--3


BEEF MYOFIBRILS
Group 1
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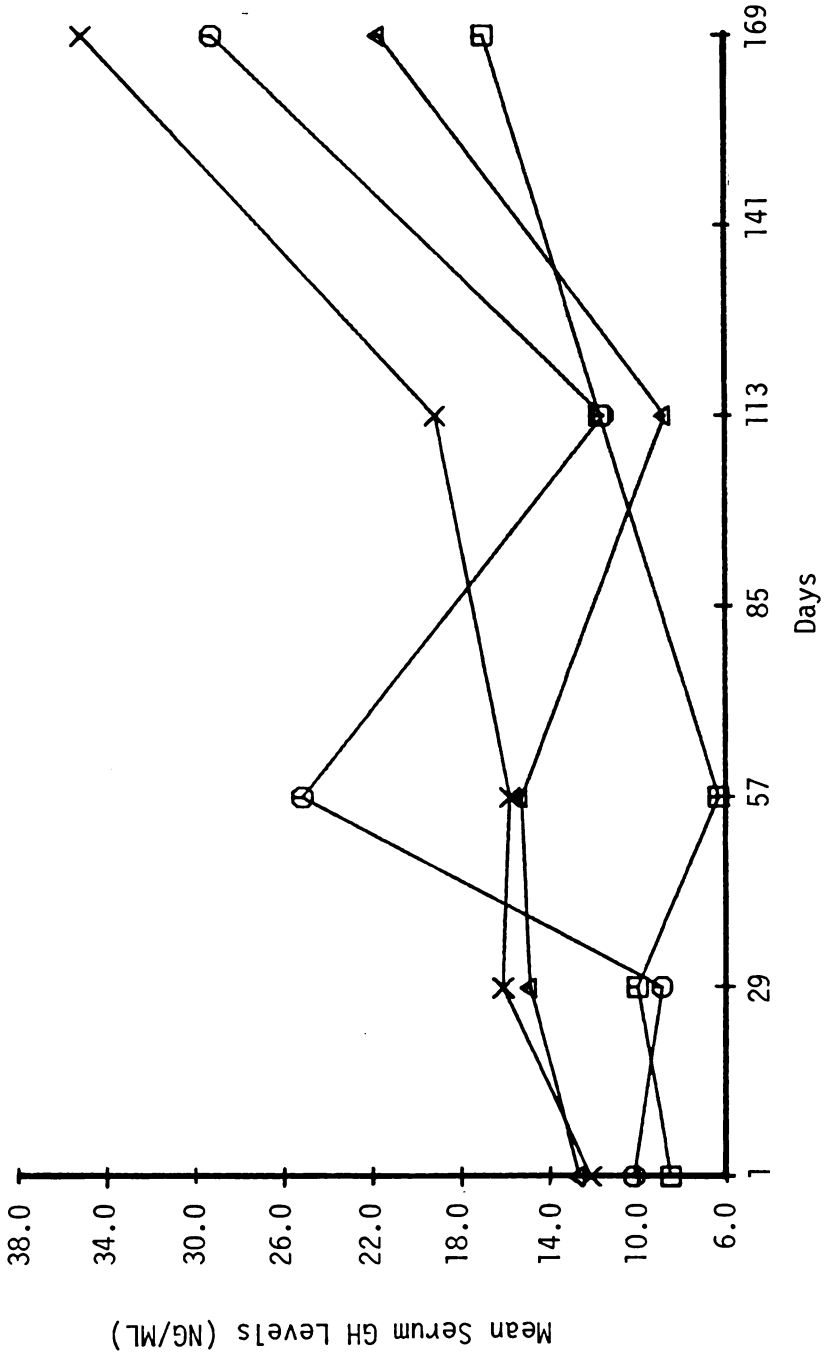

Figure 4. Mean serum growth hormone levels of Unselected Hereford (○), Selected Hereford (□), Angus x Hereford x Charolais (×), and Angus x Hereford x Holstein (△) steers (trials 1 and 2) fed a high silage ration.

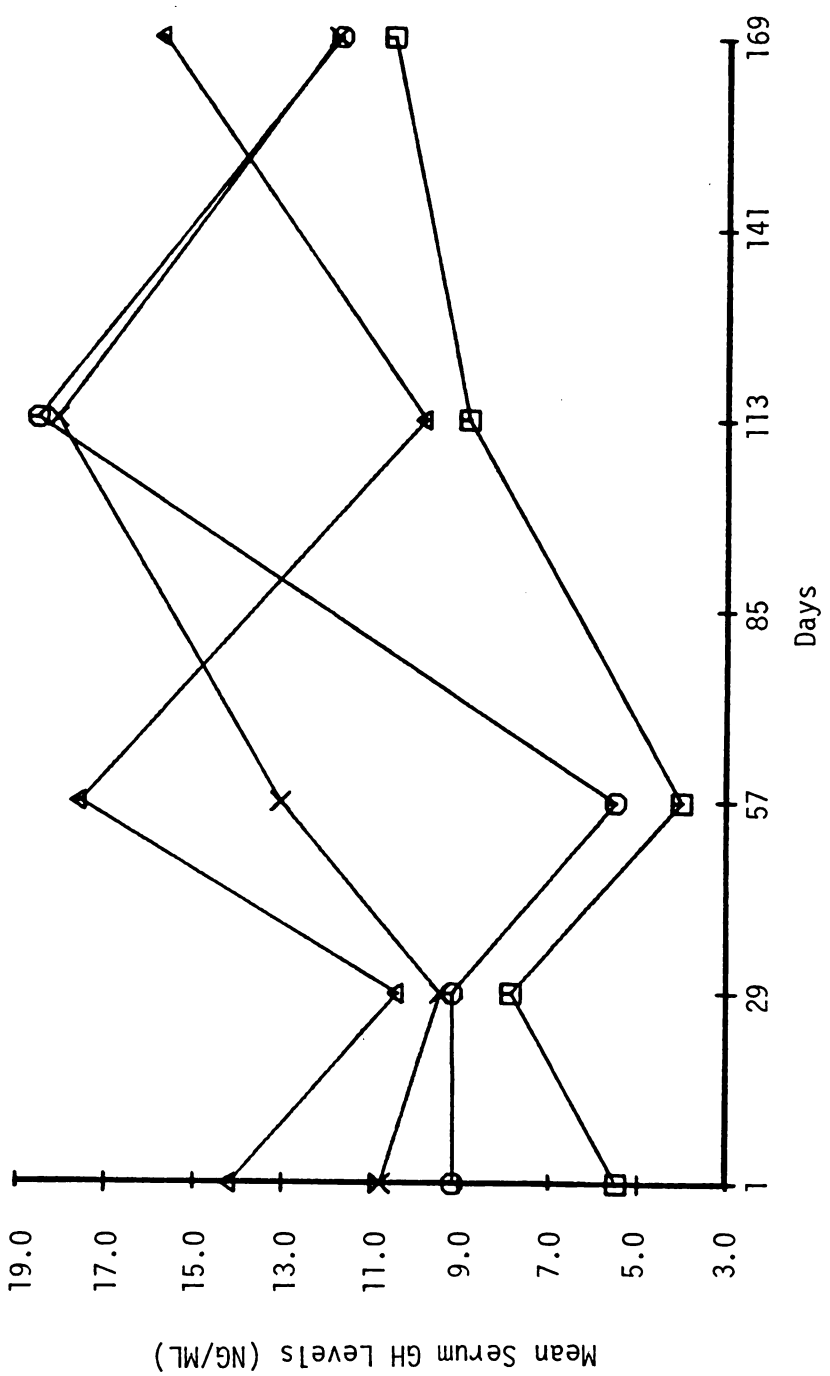

Figure 5. Mean serum growth hormone levels of Unselected Hereford (O), Selected Hereford (□), Angus x Hereford x Charolais (X), and Angus x Hereford x Holstein (Δ) steers (trials 1 and 2) fed a high grain ration.

(Figure 5) increased from 9.2, 5.5, 10.8 and 14.2 ng/ml on day 1 to 11.7, 10.5, 11.8 and 15.8 ng/ml on day 169 for the UH, SH, AHC and AHH cattle types, respectively. The same respective cattle types fed HS (Figure 4) had serum growth hormone levels increasing from 10.2, 8.5, 12.2 and 12.7 ng/ml on day 1 to 29.2, 16.9, 35.0 and 21.6 ng/ml on day 169.

The effect of ration on serum growth hormone levels in steers is shown in Table 13 and is depicted graphically in Figure 6. The type of ration had no significant effect on serum growth hormone levels of steers except on day 169 (Table 13 and/or Figure 6). However, the serum growth hormone levels on days 29 and 57 were approaching significance ($P < .098$ and $P < .067$, respectively). Steers fed HS had significantly ($P < .001$) higher levels of serum growth hormone (25.65 vs 12.47 ng/ml) on day 169 than those steers fed HG.

There was a significant effect of cattle type on serum growth hormone levels (Table 13) for days 1 and 57 ($P < .009$ and $P < .031$, respectively). In addition, day 113 was approaching significance ($P < .078$). On days 1 and 57, the SH steers had significantly ($P < .01$ and $P < .05$) lower levels of serum growth hormone than did the Holstein crossbred steers (Table 13). No significant interactions between ration and cattle type were observed. However, an interaction between the main effects was approaching significance ($P < .087$) on day 57.

Figure 7 graphically illustrates the overall change in the serum levels of growth hormone of steers within each cattle type. Serum growth hormone levels increased from 9.7, 7.0, 11.5 and

Table 13. Effects of Ration and Cattle Type on Serum Growth Hormone Levels of Feedlot Steers[a]

Days on feed	Ration[b]		Cattle type[c]				EMS[f]
	HG	HS	UH	SH	AHC	AHH	
	ng/ml						
1	9.9	10.9	9.7[de]	7.0[d]	11.5[de]	13.4[e]	58.2
29	9.2	12.5	9.0	8.9	12.8	12.7	107.8
57	10.0	15.6	15.3[DE]	5.2[E]	14.4[DE]	16.4[D]	267.1
113	13.8	12.8	15.0	10.3	18.6	9.3	254.1
169	12.5[d]	25.7[e]	20.5	13.7	23.4	18.7	411.8

[a]Least square means.

[b]HG = high grain (60, 56, 60, 59 and 59 animals/mean on days 1, 29, 57, 113 and 169, respectively); HS = high silage (58, 59, 57, 56 and 56 animals/mean on days 1, 29, 57, 113 and 169, respectively).

[c]UH = Unselected Hereford (24, 24, 24, 23 and 22 animals/mean on days 1, 29, 57, 113 and 169, respectively); SH = Selected Hereford (31, 31, 31, 31 and 31 animals/mean on days 1, 29, 57, 113 and 169, respectively); AHC = Angus x Hereford x Charolais (31, 31, 31, 32 and 32 animals/mean on days 1, 29, 57, 113 and 169, respectively); AHH = Angus x Hereford x Holstein (32, 29, 31, 29 and 30 animals/mean on days 1, 29, 57, 113 and 169, respectively).

[de]Means in rows differing in superscripts differ significantly, $P < .01$; [DE]$P < .05$.

[f]EMS = error mean square.

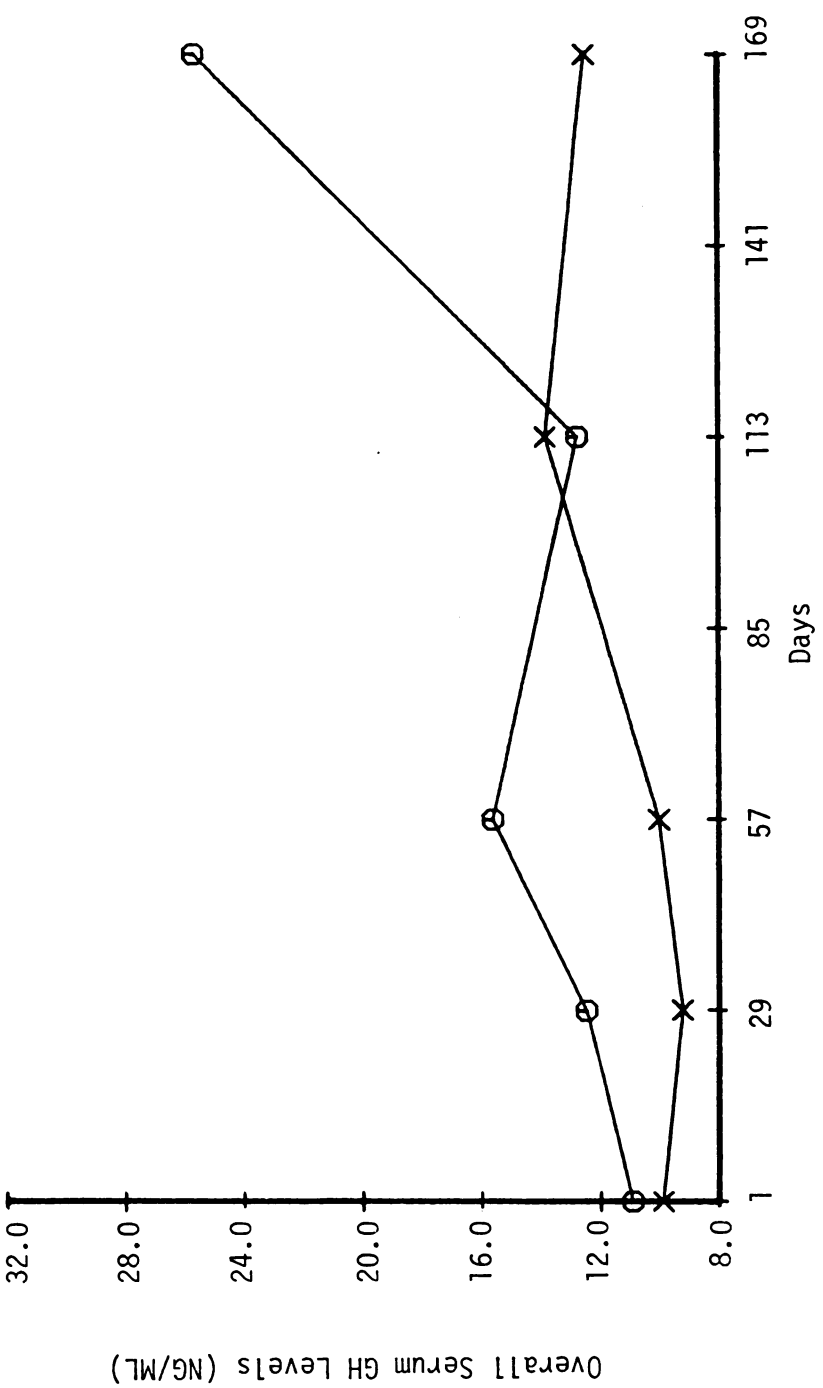

Figure 6. Overall mean serum growth hormone levels of steers (trials 1 and 2) fed either a high silage (O) or a high grain (X) ration.

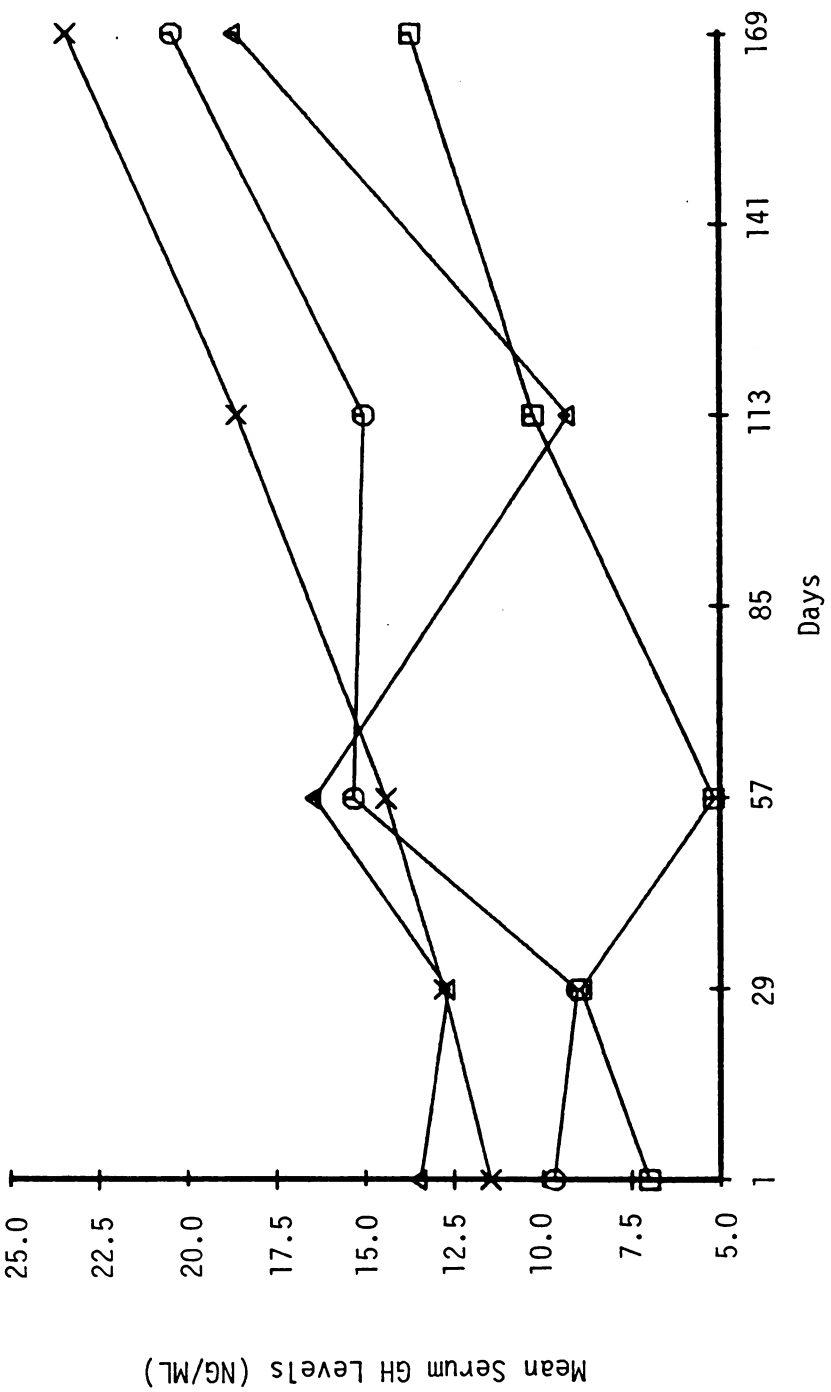

Figure 7. Mean serum growth hormone levels of Unselected Hereford (O), Selected Hereford (□), Angus x Hereford x Charolais (X), and Angus x Hereford x Holstein (Δ) steers (trials 1 and 2).

13.4 ng/ml on day 1 to 20.5, 13.7, 23.4 and 18.7 ng/ml on day 169 for the UH, SH, AHC and AHH cattle types, respectively.

Simple correlation coefficients between serum growth hormone levels and average daily gains of steers within each cattle type are presented in Table 14. Average daily gains from steers utilized in trials 1 and 2 are presented in Appendix Tables B.7 and B.10. Since day 1 represented the first day of the feedlot trial, there were no ADG data to correlate with growth hormone data for day 1. Consequently, no correlation coefficients appear for day 1. It should also be noted that the critical values for each cattle type differ due to the unequal number of observations per cattle type. Serum growth hormone levels across cattle types were not correlated to ADG. For the most part, the coefficients within each cattle type were negative or approaching zero for each observed period. However, the AHH steers seemed to show the most fluctuation in coefficients than the remaining UH, SH and AHC beef breeds. There was no correlation (0.02, -0.07, 0.04 and -0.11) between overall serum levels of growth hormone and overall average daily gains (Table 14).

Serum Insulin

Mean serum insulin levels in steers fed either a HS or a HG ration are graphically presented by days on feed in Figures 8 and 9, respectively. Both graphs had similar initial serum insulin values within a given cattle type on day 1. However, subsequent mean insulin values for each cattle type differed between rations. Figure 8 illustrates that mean serum insulin concentrations for each cattle type

Table 14. Simple Correlation Coefficients Between Serum Growth Hormone Levels and Average Daily Gains

Days on feed	Cattle type[a]				Combined
	UH	SH	AHC	AHH	
29	0.30	-0.47*	0.16	-0.19	0.02
57	-0.45*	-0.18	0.07	0.03	-0.07
113	-0.01	0.08	-0.01	0.29	0.04
169	-0.08	-0.10	-0.14	-0.17	-0.11

[a]UH = Unselected Hereford (n = 24); SH = Selected Hereford (n = 31); AHC = Angus x Hereford x Charolais (n = 32); AHH = Angus x Hereford x Holstein (n = 32).

	Critical values[1]				
	UH	SH	AHC	AHH	Combined
*(P < .05)	0.404	0.355	0.349	0.349	0.1832

[1]Snedecor, 1946.

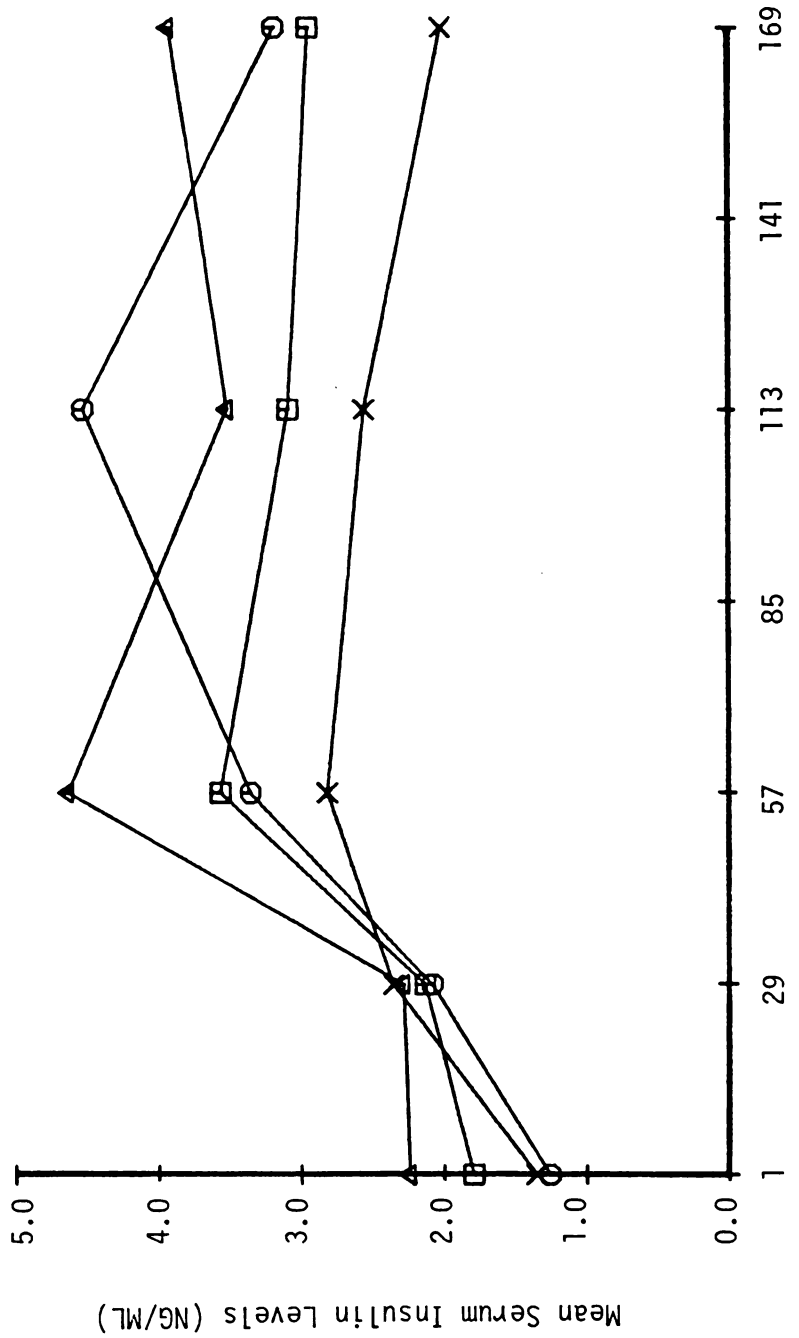

Figure 8. Mean serum insulin levels of Unselected Hereford (○), Selected Hereford (□), Angus x Hereford x Charolais (×), and Angus x Hereford x Holstein (△) steers (trials 1 and 2) fed a high silage ration.

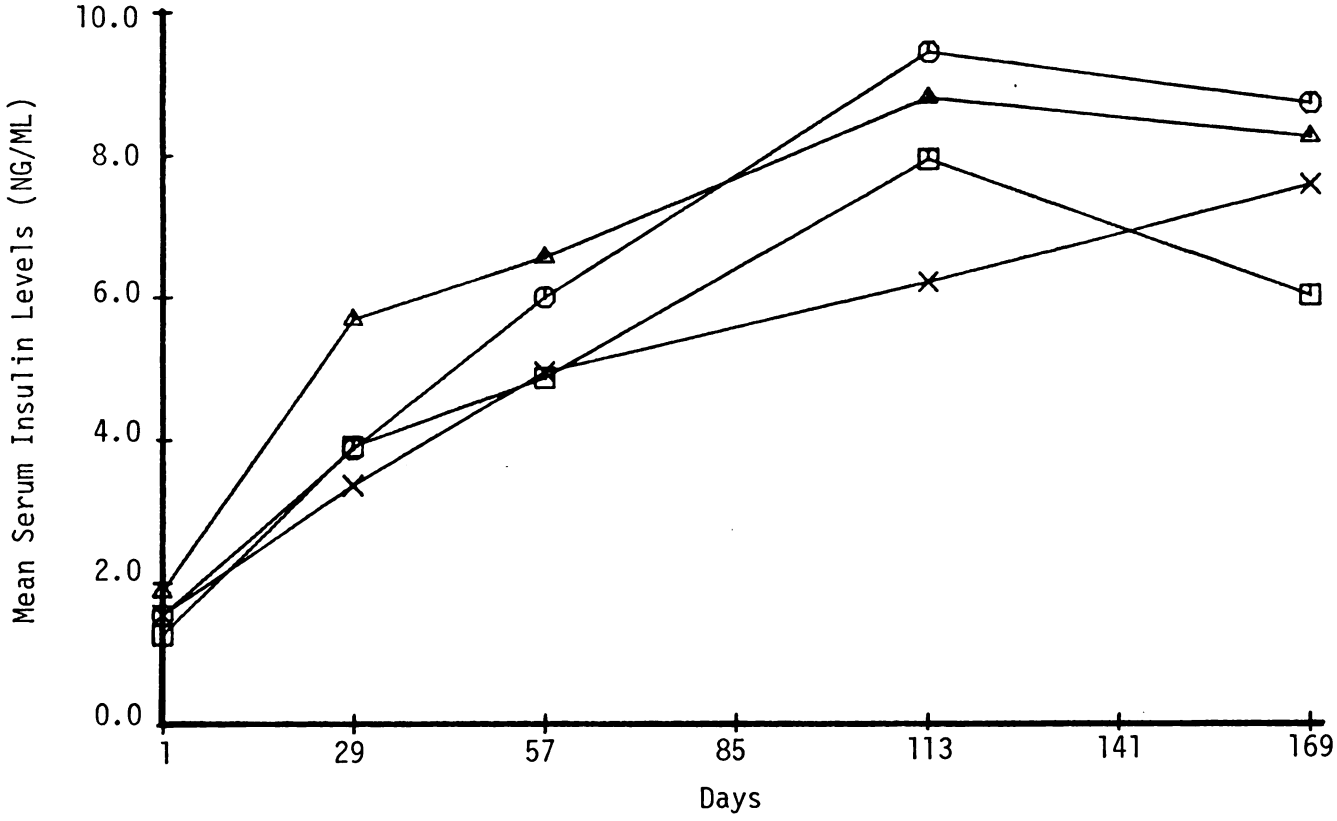

Figure 9. Mean serum insulin levels of Unselected Hereford (○), Selected Hereford (□), Angus x Hereford x Charolais (×), and Angus x Hereford x Holstein (△) steers (trials 1 and 2) fed a high grain ration.

increased gradually from day 1 to 57 but concentrations tended to level off after day 57. Insulin concentrations on day 57 for steers fed HS (Figure 8) were 3.4, 3.6, 2.8 and 4.6 ng/ml for the UH, SH, AHC and AHH steers, respectively, and remained relatively constant (3.2, 2.9, 2.0 and 3.9 ng/ml for the respective cattle types) through day 169.

Figure 9, on the other hand, showed that mean serum insulin concentrations for each cattle type fed HG increased constantly from day 1 through day 113 but decreased slightly on day 169 for all cattle types except the AHC steers. Unlike the HS fed steers, serum insulin concentrations for the HG fed steers increased from day 57 (6.0, 4.9, 4.9 and 6.5 ng/ml) to day 169 (8.7, 6.0, 7.6 and 8.2 ng/ml for the UH, SH, AHC and AHH steers, respectively).

In addition, HG fed steers had significantly higher serum insulin concentrations than their counterparts fed HS. The ration effect is depicted graphically in Figure 10 and the least square means are shown in Table 15. Serum insulin concentrations were identical on day 1 regardless of ration. However, on days 29, 57, 113 and 169, HG fed steers had significantly ($P < .0005$) higher serum insulin concentrations than steers fed HS (Figure 10 and/or Table 15). High silage fed steers had less variation in the serum insulin concentrations (ranged from 1.7 to 3.6 ng/ml) throughout the 169 days on feed, whereas serum insulin concentrations ranged from 1.6 to 8.1 ng/ml for the HG fed steers.

There was a significant effect of cattle type on serum insulin concentrations for days 1 and 113 ($P < .003$ and $P < .014$, respectively).

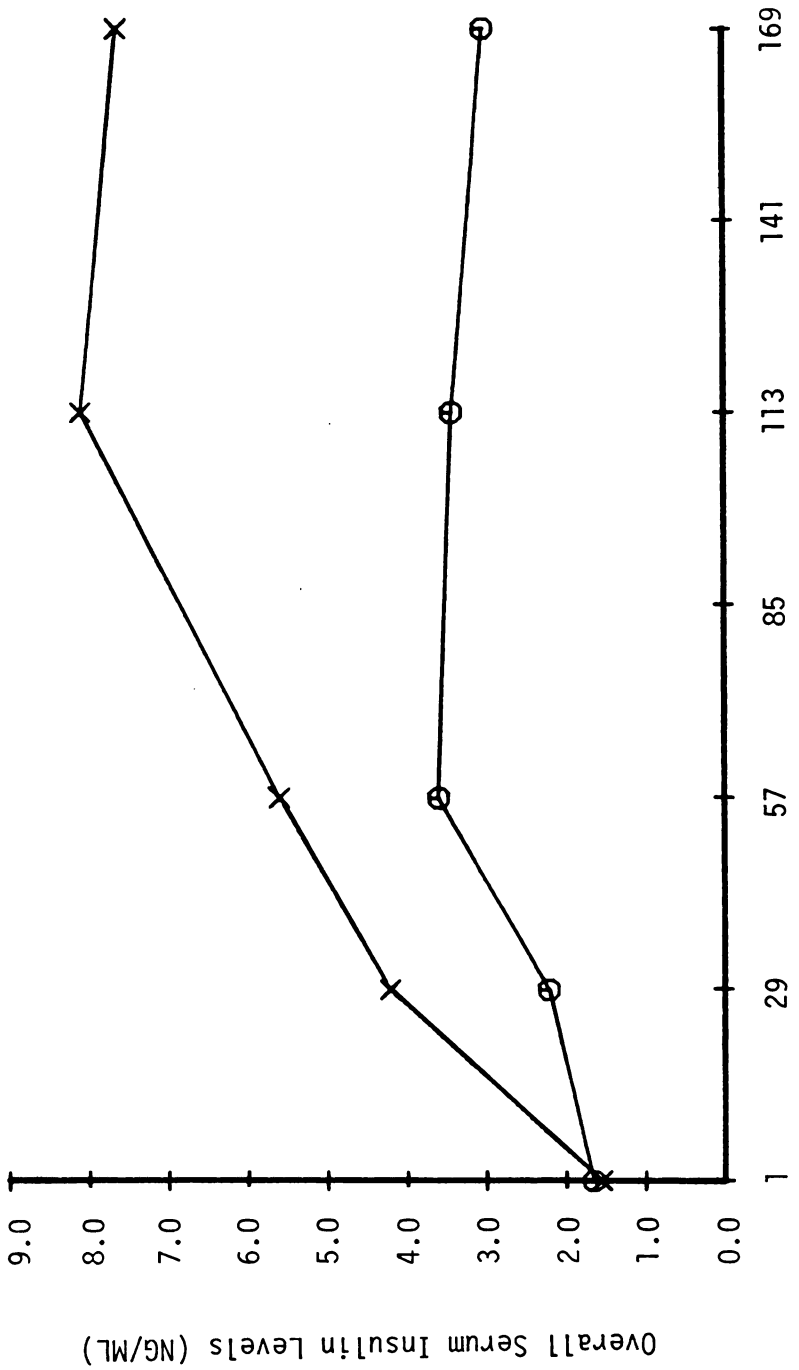

Figure 10. Overall mean serum insulin levels of steers (trials 1 and 2) fed either a high silage (O) or a high grain (X) ration.

Table 15. Effects of Ration and Cattle Type on Serum Insulin Levels of Feedlot Steers[a]

Days on feed	Ration[b]		Cattle type[c]				EMS[f]
	HG	HS	UH	SH	AHC	AHH	
	ng/ml						
1	1.6	1.7	1.4[d]	1.5[deD]	1.5[d]	2.1[eE]	0.6
29	4.2[d]	2.2[e]	3.0	3.0	2.9	4.0	3.7
57	5.6[d]	3.6[e]	4.7	4.2	3.9	5.6	7.9
113	8.1[d]	3.4[e]	7.0[D]	5.5[DE]	4.4[E]	6.2[DE]	9.1
169	7.6[d]	3.0[e]	5.9	4.5	4.8	6.1	10.8

[a]Least square means.

[b]HG = high grain (60, 56, 60, 59 and 59 animals/mean on days 1, 29, 57, 113 and 169, respectively); HS = high silage (58, 59, 53, 57 and 56 animals/mean on days 1, 29, 57, 113 and 169, respectively).

[c]UH = Unselected Hereford (24, 24, 22, 23 and 22 animals/mean on days 1, 29, 57, 113 and 169, respectively); SH = Selected Hereford (31, 31, 30, 31 and 31 animals/mean on days 1, 29, 57, 113 and 169, respectively); AHC = Angus x Hereford x Charolais (31, 31, 30, 32 and 32 animals/mean on days 1, 29, 57, 113 and 169, respectively); AHH = Angus x Hereford x Holstein (32, 29, 31, 30 and 30 animals/mean on days 1, 29, 57, 113 and 169, respectively).

[de]Means in rows differing in superscripts differ significantly, $P < .01$; [DE]$P < .05$.

[f]EMS = error mean square.

Concentrations on days 29, 57 and 169 were approaching significance ($P < .102$, $P < .099$ and $P < .161$, respectively). Holstein crossbred steers had significantly higher concentrations of serum insulin on day 1 than did the UH, SH and AHC cattle types (Table 15 and/or Figure 11). In addition, the AHC steers had significantly ($P < .05$) lower serum insulin concentrations (4.4 vs 7.0 ng/ml) on day 113 than the smaller-framed UH steers. Serum insulin concentrations increased from 1.4, 1.5, 1.5 and 2.1 ng/ml on day 1 to 5.9, 4.5, 4.8 and 6.1 ng/ml on day 169 for the UH, SH, AHC and AHH cattle types, respectively (Figure 11). Overall mean serum insulin concentrations within each cattle type increased consistently through day 113. From day 113 to day 169, serum insulin concentrations for the larger-framed AHC and AHH steers increased and seemed to parallel one another, whereas the smaller-framed UH and SH steers had decreasing insulin concentrations that also paralleled one another during this period (Figure 11).

Simple correlation coefficients between serum insulin and average daily gains of steers within each cattle type are presented in Table 16. The explanation for the critical values is similar to that discussed for Table 14. Serum insulin was positively correlated to ADG throughout the feedlot trial for the UH, SH and AHC cattle types but there was no correlation between serum insulin and ADG on days 29 and 113 for the Holstein crossbred steers (Table 16). Correlation coefficients for the SH and AHC steers were consistently significant for days 57, 113 and 169. The same trend was observed for the combined correlations. The correlation between overall serum insulin

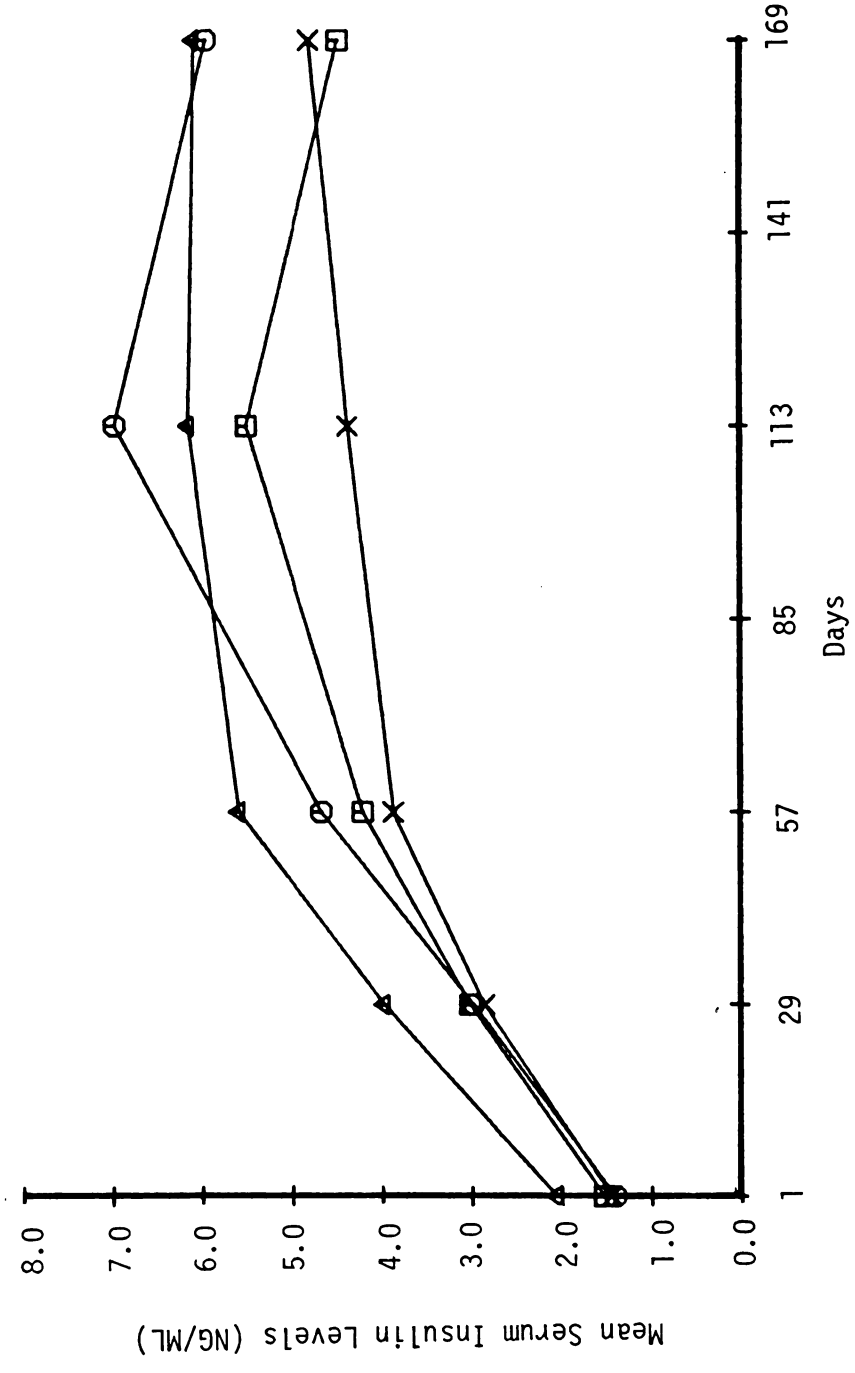

Figure 11. Mean serum insulin levels of Unselected Hereford (○), Selected Hereford (□), Angus x Hereford x Charolais (×), and Angus x Hereford x Holstein (△) steers (trials 1 and 2).

Table 16. Simple Correlation Coefficients Between Serum Insulin Levels and Average Daily Gains

Days on feed	Cattle type[a]				Combined
	UH	SH	AHC	AHH	
29	0.09	0.11	0.13	-0.03	0.09
57	0.44*	0.54**	0.51**	0.31	0.40**
113	0.14	0.50**	0.35*	-0.07	0.22*
169	0.39	0.49**	0.56**	0.39*	0.44**

[a]UH = Unselected Hereford (n = 24); SH = Selected Hereford (n = 31); AHC = Angus x Hereford x Charolais (n = 32); AHH = Angus x Hereford x Holstein (n = 32).

	Critical values[1]				
	UH	SH	AHC	AHH	Combined
*P < .05)	0.404	0.355	0.349	0.349	0.1832
**P < .01)	0.515	0.456	0.449	0.449	0.2394

[1]Snedecor, 1946.

concentrations and overall average daily gains was 0.09, 0.40, 0.22 and 0.44 for days 29, 57, 113 and 169, respectively.

Table 17 presents the correlation between serum insulin and serum growth hormone concentrations. Generally speaking, there was no correlation between the two anabolic hormones. The Holstein crossbred group was the only cattle type that consistently had correlation coefficients that were negative. Disregarding cattle type, the overall serum insulin concentrations were not correlated to serum growth hormone levels (-0.18, -0.09, -0.14 and -0.30 for days 29, 57, 113 and 169, respectively).

Anterior Pituitary Data

The mean weight, growth hormone concentration and total growth hormone content of the adenohypophyses from initial slaughter steers are shown in Table 18. The anterior pituitary data from both the initial and terminal slaughter steers are presented in Appendix Tables B.11 and B.12, respectively. Due to a malfunctioning freezer, the pituitary glands obtained from trial 1 (1975-76) were destroyed; hence, no data were available for the first year of this study. The weights of the anterior pituitary glands from initial slaughter steers tended to be heavier as frame size increased. The gland weights were 0.669, 0.757, 0.778 and 0.854 g for the UH, SH, AHC and AHH cattle types, respectively. Although the UH steers had the lightest anterior pituitary glands, they did have the highest concentration of growth hormone/gland (24.3 μg/mg). The SH steers had the lowest concentration

Table 17. Simple Correlation Coefficients Between Serum Insulin and Serum Growth Hormone Levels

Days on feed	Cattle type[a]				Combined
	UH	SH	AHC	AHH	
29	-0.23	-0.38*	-0.23	-0.14	-0.18
57	-0.37	0.10	0.14	-0.18	-0.09
113	0.04	-0.25	-0.19	-0.10	-0.14
169	-0.41*	-0.35	-0.35*	-0.28	-0.30**

[a]UH = Unselected Hereford (n = 24); SH = Selected Hereford (n = 31); AHC = Angus x Hereford x Charolais (n = 32); AHH = Angus x Hereford x Holstein (n = 32).

	Critical values[1]				
	UH	SH	AHC	AHH	Combined
*(P < .05)	0.404	0.355	0.349	0.349	0.1832
**(P < .01)	0.515	0.456	0.449	0.449	0.2394

[1]Snedecor, 1946.

Table 18. Mean Weight, Growth Hormone Concentration and Total Growth Hormone Content of Anterior Pituitary Glands from Initial Slaughter Steers[a]

Cattle[b] type	No. of animals	AP wt,[c] g	GH concentration, µg/mg	Total GH content, µg
UH	3	0.669	24.3	16,135.0
SH	3	0.757	16.4	12,441.0
AHC	3	0.778	23.4	18,293.0
AHH	4	0.854	18.8	15,773.0

[a]Anterior pituitary glands from trial 1 (1975-76) missing due to freezer malfunction.

[b]UH = Unselected Hereford; SH = Selected Hereford; AHC = Angus x Hereford x Charolais; AHH = Angus x Hereford x Holstein.

[c]Weight of anterior pituitary gland.

of growth hormone/gland and the least amount of total growth hormone/gland (16.4 μg/mg and 12,441.0 μg, respectively). The Charolais crossbred steers from the initial slaughter group had the greatest amount of total growth hormone/gland (18,293.0 μg).

The effects of ration and cattle type on the total growth hormone content, growth hormone concentration and weight of the adenohypophyses from terminal slaughter steers are presented in Table 19. There was a significant effect of ration on growth hormone concentration and total growth hormone content ($P < .040$ and $P < .012$, respectively). Cattle type had a significant effect on the anterior pituitary weight ($P < .001$) and the total growth hormone content ($P < .004$).

Steers fed HS tended to have lighter anterior pituitary weights, significantly higher adenohypophyseal growth hormone concentrations and significantly more total growth hormone within the anterior pituitary than their counterparts fed HG. High silage fed steers had 18% more growth hormone in the anterior pituitary (15,274.0 vs 12,469.0 μg) than the HG fed steers.

Holstein crossbred steers had significantly ($P < .01$) heavier adenohypophyses than both the UH and SH steers (2.18 vs 1.52 and 1.36 g, respectively). In addition, the AHH steers had significantly ($P < .01$ and $P < .05$) more total growth hormone in the anterior pituitary than did the UH and SH steers, respectively. The AHC steers also had significantly ($P < .05$) more total growth hormone than the UH steers (14,780.0 vs 10,817.0 μg). No significant interactions between ration

Table 19. Effects of Ration and Cattle Type on the Total Growth Hormone Content, Growth Hormone Concentration and Weight of Anterior Pituitary Glands from Finished Steers (Terminal Slaughter) Fed to a Constant Compositional Endpoint[a]

Item	Ration[b]		Cattle type[c]				EMS[f]
	HG	HS	UH	SH	AHC	AHH	
Total GH content (μg)	12,469.0^{D}	15,274.0^{E}	10,817.0^{dD}	12,981.0^{DF}	14,780.0^{EF}	16,907.0^{eE}	5,940,931.0
GH concentration (μg/mg)	7.5^{D}	9.4^{E}	7.2	9.8	8.7	8.0	4.4
Weight (g)	1.79	1.63	1.52^{d}	1.36^{d}	1.78^{de}	2.18^{e}	0.1

[a]Least square means; gland data from trial 1 are missing.

[b]HG = high grain (12 animals/mean); HS = high silage (12 animals/mean).

[c]UH = Unselected Hereford; SH = Selected Hereford; AHC = Angus x Hereford x Charolais; AHH = Angus x Hereford x Holstein; six animals/mean for each cattle type.

[de]Means in rows differing in superscripts differ significantly, $P < .01$; [DEF]$P < .05$.

[f]EMS = error mean square.

and cattle type were observed for the anterior pituitary weight or the concentration. However, a significant interaction ($P < .029$) between main effects was observed for the total growth hormone content of the anterior pituitary.

In comparing the anterior pituitary data from the initial slaughter steers (Table 18) to the data from the terminal slaughter steers (Table 19), there was a substantial increase among cattle types in the weight of the anterior pituitary and a large reduction in the concentration of growth hormone in the adenohypophysis. Total growth hormone in the anterior pituitary tended to decrease from the initial to the terminal slaughter steers in the UH and AHC groups. However, the opposite was observed in the SH and AHH groups.

DISCUSSION

Feedlot Performance

Averaged across cattle types, high grain (HG) fed steers gained 26% faster than high silage (HS) fed steers. Hatfield et al. (1971) observed that average daily gains were 33% greater for cattle fed a 67% high moisture corn ration vs those fed a high silage ration. Similar results in other studies which had HG and HS comparisons were reported (Minish et al., 1966; Minish et al., 1967; Gill et al., 1976).

Fox and Black (1977) devised a performance simulation model in which a linear relationship between rate of gain and frame size was observed. Results of the present study were in accordance with the model of Fox and Black (1977). Average daily gains increased with increasing frame size for all cattle types except the Holstein crossbred steers. Crickenberger (1977) and Harpster (1978) reported that Holstein and Holstein crossbred steers gained less than expected based on their frame size. The preceding data did not conform to those of Dean et al. (1976) and Wyatt et al. (1977) who reported that average daily gains increased as the percentage of Holstein breeding increased.

Numerous investigators have reported that when cattle were fed to similar compositional endpoints, feed efficiency was independent of frame size for British and British x Exotic crossbred cattle (Klosterman et al., 1968; Fox and Black, 1977; Smith et al., 1977). However,

the smaller-framed steers in the present study tended to be more efficient than the larger-framed cattle. Similar observations have been reported by Ferrell et al. (1978).

The feed/gain data of AHH steers in the present trial revealed a decreased efficiency than for the UH, SH and AHC steers. Similar findings have been reported by Dean et al. (1976) and Wyatt et al. (1977). The poorer efficiency of the AHH steers is most likely attributed to a higher maintenance requirement. Ayala (1974) concluded that Holsteins required more energy for maintenance per unit of metabolic weight than Angus. Moreover, Holsteins were found to have higher feed requirements above maintenance per unit of gain than Herefords (Garrett, 1971).

Semitendinosus Muscle Data

Proximate Analysis

Relatively little data are available on the chemical composition of individual muscles that constitute a large proportion of the total muscle mass. However, data are plentiful on the protein, fat and water content of muscle tissue during growth and development of an animal. Numerous data have revealed that the proportions of lipid and protein increased and the percentage of water decreased with increasing animal age (Dickerson, 1960; Dickerson and Widdowson, 1960; Sink and Judge, 1971; LaFlamme et al., 1973; Giovannetti and Stothers, 1975). These data are corroborated by those of the present study in which the moisture percentage decreased, fat percentage increased and protein percentage increased slightly with advancing age.

Garrett and Hinman (1971) analyzed five freshly trimmed muscles from the beef carcass for ether extract and water content. They observed that the moisture percentages of U.S. Choice grade carcasses were 71.7, 69.9, 72.3, 72.6 and 72.7% for the infraspinatus, serratus ventralis, longissimus, gluteus medius and semimembranosus with adductor femoris, respectively. The fat percentages were 8.7, 11.2, 5.4, 5.0 and 4.6% for the respective muscles. Semitendinosus (ST) data from the current study, when averaged across cattle types, revealed that the moisture and fat percentages were 72.5 and 5.4%, respectively. These data are comparable to the semimembranosus with adductor femoris data of Garrett and Hinman (1971), the semimembranosus data of Doty and Pierce (1961) and Breidenstein et al. (1968) and the longissimus data of Tuma et al. (1963). The Garrett and Hinman (1971) study, however, reported no information concerning the breed or nutritional regimes of the beef carcasses utilized. Results of the present study have shown that finished steers fed HS had ST muscles that averaged 1.1% lower in ether extract and 1.2% higher in moisture than ST muscles from finished steers fed HG. Terminal slaughter steers within each cattle type were killed at an approximate constant compositional endpoint of 28 to 32% carcass fat. Since fat is the most variable of the carcass tissues (Elsley et al., 1964; Berg and Butterfield, 1976) and, consequently, very difficult to predict, finished steers fed HS, when averaged across cattle types, had 28.2% carcass fat vs 34.5% carcass fat for their counterparts fed HG (Harpster, 1978). All steers were adjusted to similar carcass weight

within a type. As a result, steers fed HG had a higher percentage of both carcass fat and ST fat and a lower moisture percentage in the ST than steers fed HS. These data of the ST agree with those of Leander et al. (1978) who also reported a decline in the moisture percentage and an increase in the ether extract percentage of the ST from grain-fed cattle vs those that were grass-fed. The protein percentage in the present study, as well as in the study performed by Leander et al. (1978), did not change between rations.

Although no significant differences in the proximate analysis of the ST from finished steers were observed between rations or among cattle types, it is highly possible that the small number of available animals considerably decreased the sensitivity of the statistical analyses. Consequently, this may have prevented the observed differences from being significantly different.

Nucleic Acids and Muscle Protein Fractions

Research data dealing with nucleic acid concentrations and muscle protein fractions in meat animals are not plentiful. Since muscle cells are multinucleated, the use of DNA as an index of cell number is logically subject to question (Bergen et al., 1975). The DNA analysis of muscle tissue indiscriminately includes blood vessels, connective tissues and nerves. Nevertheless, DNA concentrations of muscle tissue remain one of the best available measures of cellularity. Also, the use of protein/DNA or cell protein is likewise accepted as an indicator of cell size (Robinson, 1971; Cheek et al., 1971). Enesco

and Leblond (1962) observed that 55% of the nuclei within a muscle in a three-month-old rat were in muscle cells and the proportion increased with age. However, they later showed that a major proportion of the increase in muscle DNA originated from an increase in the number of nuclei within the muscle fiber itself. Satellite cells have contributed to the postnatal increase in skeletal muscle nuclei (Enesco and Leblond, 1962).

Results in the present study revealed that the DNA and RNA concentrations across cattle types were lower in terminal slaughter steers than in initial slaughter steers. These data are in agreement with those of Robinson (1969), Tsai et al. (1973), Powell and Aberle (1975) and Harbison et al. (1976) in swine and LaFlamme et al. (1973) and Johns and Bergen (1976) in ruminants. Conversely, total DNA and total RNA contents of the ST muscle from each cattle type were higher in terminal slaughter steers than in initial slaughter steers. This finding has been well-documented by numerous investigators (Robinson, 1969; LaFlamme et al., 1973; Tsai et al., 1973; Powell and Aberle, 1975; Johns and Bergen, 1976; Harbison et al., 1976). Differences in total DNA, total RNA and total protein among cattle types appear to be mainly a reflection of the differences in ST weights. The larger-framed Charolais and Holstein crossbred steers had significantly heavier ST muscles than the smaller-framed UH steers. These data are in accordance with those of Kline et al. (1969) and Garcia-de-Siles et al. (1977).

The finished UH steers in this study possessed ST muscles with the lowest RNA concentration (0.79 mg/g), the lowest RNA/DNA ratio (2.9) and the highest protein/DNA ratio (774.9). Since RNA is a good measure of the available machinery capable of synthesizing protein (Wannamacher, 1972), Winick and Noble (1965), Munro (1969) and Powell and Aberle (1975) reported that a low RNA/DNA ratio was indicative of a low protein synthesizing capacity. The use of assessing differences in protein deposition, however, using nucleic acids as critical markers in finished steers, may not be valid because during the finishing phase, there is little net protein deposition. Utilizing the same cattle, Harpster (1978) observed that the average carcass protein gained per day between rations was similar among SH, AHC and AHH steers (105.9, 106.1 and 104.7 g/day, respectively). The UH steers gained the least amount of protein (87.0 g/day). Consequently, the data from the present study seem to illustrate that the finished UH steers had less available protein synthetic machinery in the ST muscle cell and, therefore, synthesized less protein per unit muscle weight. However, the finished UH steers did have the physiological cell size (protein/DNA) in their favor but the available protein synthetic machinery seemed to be the limiting factor.

Averaged across steer types, the HS fed steers had significantly heavier ST weights and significantly more total protein per ST than steers fed HG (Table 10). Additionally, the HS fed steers had more total RNA per ST, a higher RNA/DNA ratio and a significantly higher protein/DNA ratio (Table 9). Wannamacher and McCoy (1966) and Howarth

(1972) reported that protein synthesis was highly correlated with cellular RNA content. Furthermore, Waterlow and Stephen (1966), Young and Alexis (1968) and Young et al. (1971) observed that diets low in crude protein would decrease protein synthesis at the cellular level. Subsequent studies by Gilbreath and Trout (1973) and Trenkle (1974) showed that decreased protein synthesis was caused by a reduction in RNA content. Johns (1974) observed a decrease in total RNA, total DNA and the protein synthetic rate in lambs fed a low protein ration. As a result, there was a decrease in the weight of the gastrocnemius muscle. Additionally, lambs growing at different rates due to dietary protein density did not differ in DNA and RNA concentrations but differed in total RNA/DNA ratios.

Results of the present study did show variations in ST weight, total protein/ST, total RNA/ST, RNA/DNA ratio and protein/DNA ratio in finished steers fed HS vs those fed HG. These differences between rations could have resulted from inadequate crude protein levels in HG fed steers or possibly these steers were utilizing excess energy and/or protein for depot fat. Harpster (1978), however, reported that the intake of daily crude protein was significantly higher (0.974 vs 0.890 kg) in HG vs HS steers. Furthermore, he noted that the HG fed steers consumed significantly more metabolizable energy per day than the counterparts fed HS.

RNA/DNA ratios among cattle types tended to be lower for the terminal slaughter steers than those slaughtered initially. The protein/DNA ratios, on the other hand, were higher for the heavier

slaughter groups. These data are corroborated by data of Robinson (1969) and Powell and Aberle (1975) in swine and Lipsey et al. (1977) in cattle.

The muscle protein fractionation data, expressed as a percentage of the total protein nitrogen, for the initial slaughter cattle were 54, 19, 15 and 12% for the myofibrillar, sarcoplasmic, stroma and non-protein nitrogen fractions, respectively. These data consistently agree with the bovine data of Hill (1962) and Aberle (1965). Hill (1962) reported values of 55% for myofibrillar nitrogen, 19% for sarcoplasmic nitrogen, 14% for stroma nitrogen and 13% for non-protein nitrogen on a fat-free, fresh tissue basis.

A review of the literature indicated an increase in the total content of the intracellular proteins, myofibrillar and sarcoplasmic, with growth (LaFlamme et al., 1973; Gordon et al., 1966). However, Young (1970) reported that both the myofibrillar and sarcoplasmic proteins differed according to the stage of development with the rate of change related to the time of functional development of the muscle. Gordon et al. (1966) observed that the increase in myofibrillar protein predominated during the period of rapid protein accumulation. Although this was a comparative study between initial and terminal slaughter animals, there was no intention of seeking when the period of rapid protein accumulation occurred. Results of this study indicated, however, that total protein as well as each muscle protein fraction increased among cattle types. There tended to be an inverse relationship between cattle frame size and the percentage increase in

both the total protein content and weight of the ST muscles from terminal slaughter vs initial slaughter steers. The percentage increase in total protein was 161, 140, 125 and 106% for the UH, SH, AHC and AHH steers, respectively, for an average of 133%. These total protein data are in total agreement with LaFlamme et al. (1973). Additionally, there was a 148, 140, 118 and 92% increase in the ST weight in each of the respective steer types. The increase in the percentage of total protein was higher for the smaller-framed UH and SH steers because the percentage increase in ST weight was greater for the UH and SH vs AHC and AHH steers. No significant differences ($P > .05$) were observed in the protein percentage of the ST muscles across cattle types (Tables 7 and 8).

Data of LaFlamme et al. (1973) revealed that sarcoplasmic and myofibrillar proteins in finished cattle increased 125 and 150%, respectively, from initial concentrations. However, data from the present study indicated that the average percentage increase in sarcoplasmic and myofibrillar proteins across cattle types was 191 and 124%, respectively. A plausible explanation for the contradictory results may be attributable to the experimental procedure in extracting the muscle protein fractions. The quantity of muscle tissue (5 g) may have been too small for the large centrifuge bottle (250 ml) used during the extraction process. The structure of the bottle was such that it was practically impossible to decant the supernatants without losing a small quantity of muscle tissue.

The small-framed UH steers consistently had lower protein contents in the various muscle protein fractions (Tables 7 and 12). Table 7 revealed that the finished UH steers had significantly lower sarcoplasmic, myofibrillar and stroma protein (28, 29 and 29%, respectively) than the Charolais crossbred steers. However, since each fraction concentration was similar across cattle types, the difference in the protein content of each muscle protein fraction was a function of the ST weight. These data seem to agree with the feedlot performance data of Harpster (1978), who observed that increased frame size was the main effect of selecting for yearling weight. This phenomenon of how selection for body size influenced muscle weight had been illustrated in rodents by numerous investigators (Luff and Goldspink, 1967; Byrne et al., 1973; Hanrahan et al., 1973). Although ration had a significant effect on the total protein content of the ST muscle, as previously discussed, there was no effect of ration on the various muscle protein fractions.

SDS-Polyacrylamide Gel Electrophoresis

Little differences in the characterization and isolation of the myofibrillar proteins from ST samples of each cattle type were detected. However, upon close examination of Figure 3, one should be able to notice variations in the intensity of the protein bands. This resulted from differences in the amount of myofibrillar protein loaded onto the gels. As can be expected, the group 1 gel had the most myofibrillar protein (135 μg) loaded on it, whereas the group 4 gel had the least (34 μg). Gels representing groups 2 and 3 were intermediate

in protein concentration (74 and 70 μg, respectively). The protein bands of each gel were identified according to Porzio and Pearson (1977).

The avoidance in band distortion can be prevented by extreme care in the gel casting process. However, not enough care was taken in casting gels 1 and 3; consequently, some distortion in the myosin and actin bands has occurred.

Blood Data

Serum Growth Hormone

Data from the present study have indicated that ration had no effect on serum growth hormone concentrations of steers on days 1, 29, 57 and 113 of the feedlot trial. Earlier findings of Trenkle (1971b), McAtee and Trenkle (1971) and Rabolli and Martin (1977) have shown the same effect. However, on day 169 of the feedlot trial, the HS fed steers had significantly higher ($P < .01$) levels of serum growth hormone than did HG fed steers. Although Trenkle (1971b) reported that ration energy level had no effect on plasma growth hormone concentrations, he did observe, however, in one particular experiment that the plasma growth hormone levels were lower in sheep fed high concentrate *vs* high roughage rations.

Trenkle (1971b) and Hove and Blom (1973) have observed erratic alterations in bovine growth hormone levels and have associated these changes with the ration energy content. They also reported that serum growth hormone levels were elevated if animals were fed a high roughage

or low energy diet. These data agree with this study which noted higher serum growth hormone levels in HS vs HG fed steers. In the present study, steers within each cattle type consuming a HG ration (Figure 5) had more chronic changes between blood sampling periods in serum levels of growth hormone than did their counterparts fed HS (Figure 4). These observed differences were probably not reflections of the variation in managerial or environmental conditions because all animals were handled similarly prior to bleeding and exposed to the same climatic conditions. Hart et al. (1975) did, however, report chronic changes in the plasma growth hormone concentrations of lactating beef and dairy cows that were identically matched for age, stage of lactation and diet. They have suggested that the elevated levels of growth hormone in dairy cows may have been the result of alterations in the rate of hormone inactivation or in the rate of secretion, imposed possibly by lactation or by genetic differences between the two breeds of cows. They later concluded that the higher circulating levels of growth hormone in dairy cows were associated with their greater energy requirement.

The serum growth hormone patterns of this study clearly indicate the relative importance of sampling blood several times per day rather than single daily samples to assure repeatability. Single blood samples yield little useful information regarding accurate interpretation of hormonal data because such factors as diurnal variation or stress can markedly affect serum concentrations of certain hormones.

Contradictory data have appeared in the literature concerning the effect of stress on serum growth hormone concentrations. Eaton et al. (1968) have observed a 19-fold increase in the serum growth hormone levels of cattle due to stress in the bleeding chute. Conversely, Tucker (1971) has reported that repeated and rapid jugular venipuncture had no effect on the growth hormone concentrations of cattle. In the present study, the variation of growth hormone levels of each cattle type were probably not related to stress at the time of blood collection because all steers were handled gently, quietly and bled rapidly.

Very little data have been reported on the effect of beef breed on serum growth hormone concentrations. Data from the present study have indicated no consistent, significant differences in the serum growth hormone levels among the four cattle types. Similar results have been reported by Grigsby (1973), Trenkle (1971a) and Dev and Lasley (1969). The serum growth hormone values reported within this study were considerably lower than the growth hormone levels reported by Grigsby (1973) but were in agreement with those of Leining (1978), Eaton et al. (1968) and Trenkle (1971a).

Correlation data between average daily gains and serum growth hormone concentrations in this study have revealed that neither variable is highly correlated to the other. The negative relationship of serum growth hormone with average daily gain is corroborated by the work of Siers and Hazel (1970), Trenkle (1970), Siers and Swiger (1971), Purchas et al. (1971), Hafs et al. (1971), Bidner et al. (1973), Joakimsen and Blom (1976) and Trenkle (1977).

Plausible explanations have been suggested by Gershberg (1957), Trenkle and Irwin (1970), Siers and Hazel (1970) and Hafs et al. (1971) for the low correlation between growth rate and serum growth hormone. The latter two investigators suggested that slow-growing animals utilized growth hormone at a lesser rate than rapid-growing animals. The former pair of investigators postulated that with increasing age, the target tissues became less responsive to low physiological levels of growth hormone. However, recent reports by Phillips and Orawski (1977), Daughaday et al. (1975) and Tanner (1972) have stressed the importance of somatomedin for body growth. A strong possibility exists that insulin and growth hormone per se may not be directly responsible for developmental growth but both may act synergistically in the presence of somatomedin to influence body growth. However, additional research in this area is needed.

Serum Insulin

Serum concentrations of insulin, unlike growth hormone, were significantly influenced by ration in the present study. Although serum insulin levels were similar between rations on day 1 of the feedlot trial, there was a significant difference in the serum insulin concentrations on days 29, 57, 113 and 169 between HG vs HS fed steers (Table 15). These findings are in agreement with earlier reports by Trenkle (1966; 1970).

Regardless of cattle type, serum concentrations tended to increase with length of time in the feedlot. Each cattle type had higher insulin levels on day 169 than on day 1. These data are

corroborated by Trenkle (1970), Stern et al. (1971), McGuffey (1975) and Johns and Bergen (1976).

Like growth hormone, there is limited data on the effect of cattle type on serum insulin levels. Earlier data reported that serum insulin concentrations were significantly higher ($P < .05$) in Holstein steers than in any other breed (Grigsby, 1973). Results from the present data indicated that the Holstein crossbred steers had consistently higher concentrations of serum insulin than the UH, SH or AHC cattle types. However, the only significant differences were observed on day 1 of the feedlot trial. This significant observation on day 1 may have been due to greater feed intakes by the heavier AHH steers to sustain growth and maintenance. Hove and Blom (1973) reported that insulin concentrations in cattle varied with feed intake. Additionally, Bassett et al. (1971) observed that the amount of organic matter digested in the lower gut was positively correlated with plasma insulin.

Contrary to the correlation data between serum growth hormone concentrations and average daily gains, serum insulin tended to be significantly correlated to feedlot gains among the pooled cattle types (except on day 29). Similar data have also revealed positive correlations between average daily gains and serum insulin (Trenkle, 1970; Grigsby, 1973; Johns, 1974). It would appear that serum insulin and not growth hormone would be a fairly accurate measure of growth as related to average daily gain.

Correlation coefficients between serum concentrations of insulin and growth hormone in the present study were low and, in most cases, nonsignificant. Grigsby (1973) reported similar observations. Several investigators have stated that growth hormone and insulin were important growth-promoting hormones that acted synergistically to insure an ample supply of energy to body tissues or to store excess energy (Rabinowitz and Zierler, 1963; Weil, 1965; Althen, 1975). Although blood glucose was not determined in this study, it has been shown to be positively correlated to serum insulin (Davis et al., 1970; Siers and Trenkle, 1973) and negatively related to serum growth hormone (Machlin et al., 1968, 1970; Siers and Trenkle, 1973).

Anterior Pituitary Data

Results of the anterior pituitary data revealed that as steers increased in size or body weight across cattle types (initial vs terminal slaughter), there was a corresponding increase in the weight of the anterior pituitary. Trenkle (1977) and Purchas et al. (1970) reported the identical observation in cattle. Additionally, the initial slaughter steers across cattle types had relatively higher glandular concentrations of growth hormone (μg/mg) than the terminal slaughter steers. These data are in agreement with those obtained by Purchas et al. (1970), Curl et al. (1968) and Armstrong and Hansel (1956) and indicate that the steer calf has a relatively high concentration of adenohypophyseal growth hormone that declines with age. However, Johns and Bergen (1976) and Curl et al. (1968) have indicated that growth

hormone content of the anterior pituitary increased as gland size increased. These data are not in accordance with the present study that showed a leveling off of total pituitary growth hormone from 215.2 to 519.4 kg of live weight for the initial and terminal slaughter steers, respectively. Trenkle (1977) also reported that the growth hormone content of the adenohypophysis tended to level off as body weight more than doubled (231 vs 505 kg). He did, however, report that total pituitary growth hormone increased as body weight increased from 112 to 231 kg. Purchas et al. (1970) earlier found that pituitary growth hormone content increased at an increasing rate from birth to three months of age but decreased thereafter to five months of age at an increasing rate. Total growth hormone content remained relatively stable from five to twelve months of age.

Results from the present study also indicated that cattle type has no effect on the pituitary growth hormone concentration. These data are in accordance with those of Trenkle (1977). Cattle type did have a significant effect on anterior pituitary weight. The larger-framed AHH steers had significantly heavier ($P < .01$) glands than the smaller-framed UH and SH steers. This may have been attributable to differences in cattle size and/or weight. Purchas et al. (1970) and Trenkle (1977) also showed that pituitary growth paralleled body growth and that heavier cattle had heavier adenohypophyses than lighter cattle. Similar observations were noted in female rats (Ojeda and Jameson, 1977).

Although sparse data have been reported on the effect of ration on the anterior pituitary weight, results of this study revealed that there were no significant differences in the anterior pituitary weights (1.79 vs 1.63 g) of HG vs HS fed steers, respectively. Little difference in terminal slaughter weights were observed in the HG vs HS fed steers (517.4 vs 521.4 kg, respectively). Johns and Bergen (1976), however, reported a significant effect ($P < .05$) of a low vs high protein diet on pituitary weight. On the other hand, crude protein levels were not limiting in the present study.

The effect of ration was significant ($P < .05$) on the growth hormone concentration and total content of the anterior pituitary. High silage fed steers had a higher pituitary concentration (20%) and total content (18%) than HG fed steers. Since high roughage or low concentrate diets elevate serum growth hormone concentrations (Trenkle, 1971b; Hove and Blom, 1973), these types of rations may enhance the synthesis of growth hormone and its release from the anterior pituitary. However, additional research is needed to better elucidate the effects of high roughage rations on pituitary and plasma growth hormone. Research must also be undertaken to understand the mechanisms involved in slow-growing animals that have high circulating levels of growth hormone.

Although cattle type had a significant effect on total pituitary growth hormone, the total content was confounded by a significant interaction between the main effects of ration energy level and cattle type. However, the least square means revealed that the larger-framed AHC and

AHH steers tended to have significantly more total growth hormone than did the smaller-framed UH and SH steers. This same significant trend was also observed in the anterior pituitary weight and this may have influenced the significance of the total growth hormone content among cattle types.

CONCLUSIONS

1. Average daily gains were 26% greater for steers fed HG than the HS rations.
2. Average daily gains increased with increasing frame size for all cattle types except the Holstein crossbred steers.
3. The ST muscles from terminal slaughter steers had (percentage basis) less moisture, higher intramuscular lipid and slightly more protein than ST muscles from initial slaughter steers.
4. Steers fed HG had ST muscles that were lower in moisture and higher in intramuscular lipid than those muscles from HS fed steers.
5. Muscle DNA and RNA concentrations across cattle types were lower from terminal slaughter steers than from initial slaughter steers. However, total muscle DNA and RNA contents were higher from the terminal slaughter steers.
6. Steers fed HG had significantly lighter ST weights and significantly lower protein/DNA ratios than steers fed HS. Additionally, HG fed steers had lower total RNA/ST, lower total protein/ST and lower RNA/DNA ratios than steers fed HS.
7. The larger-framed Charolais and Holstein crossbred steers had heavier ST muscles, more total protein/ST and more total DNA/muscle than the smaller-framed UH and SH steers. The absolute weights of the ST muscle increased with increasing frame size.

8. There tended to be an inverse relationship between cattle frame size and the percentage increase in both the total protein and the weight of the ST muscles from terminal slaughter *vs* initial slaughter steers.
9. The total content of the intracellular proteins increased substantially with growth across cattle types. Total protein/muscle also increased with advancing age.
10. Ration had no significant effect on the total protein in the various protein fractions. However, cattle type had a significant effect on the total protein in the sarcoplasmic, myofibrillar and stroma fractions but the relative differences were a function of the ST weight.
11. There were no differences in myofibrillar protein composition in ST muscles among the four cattle types.
12. Serum growth hormone concentrations in steers fed HG tended to fluctuate more erratically throughout the feedlot period than in steers fed HS.
13. Serum growth hormone levels throughout the trial were not consistently affected by cattle type or ration.
14. Serum levels of growth hormone tended to be higher in steers fed HS than HG.
15. The correlation between serum growth hormone and ADG was negative (or approaching zero), whereas serum insulin was positively correlated with ADG. Additionally, correlation coefficients between serum concentrations of insulin and growth hormone were, in most cases, negative and nonsignificant.

16. High grain fed steers had significantly higher serum insulin levels than their counterparts fed HS. Serum insulin concentrations also tended to increase with increasing time in the feedlot.
17. The absolute anterior pituitary weight increased with increasing frame size among cattle groups.
18. The initial slaughter steers across cattle types had higher anterior pituitary concentrations of growth hormone than the terminal slaughter steers. However, total pituitary growth hormone tended to level off from 215.2 to 519.4 kg of live weight for the initial and terminal slaughter steers, respectively.
19. Cattle type had no significant effect on the adenohypophyseal growth hormone concentration but did have an effect on the weight and total content of the anterior pituitary gland.
20. High silage fed steers had significantly higher anterior pituitary concentration and content of growth hormone than HG fed steers.

APPENDIX A

COMPOSITION OF REAGENTS

Table A.1 Borate Buffer

Using a 1,000 ml volumetric flask, dissolve 7.6274 g $Na_2B_4O_7 \cdot 10\ H_2O$ and 5.0 g sodium lauryl sulfate in 900 ml of deionized distilled water. Adjust pH to 8.0 and bring volume to 1 liter.

Table A.2 Orcinol Reagent

Make a stock solution of 0.1% $FeCl_2 \cdot 6\ H_2O$ in concentrated HCl. Before each use, dissolve 1 g orcinol in 100 ml of stock solution.

Table A.3 Diphenylamine Reagent

Dissolve 4 g diphenylamine reagent in 100 ml of glacial acetic acid.

Table A.4 Acetaldehyde Solution

Add .4 ml of acetaldehyde to a 250 ml volumetric flask. Dilute with deionized distilled water and bring volume to 250 ml. Store at 4°C.

Table A.5 Low Ionic Strength Buffer

Using a 1,000 ml volumetric flask, dissolve 2.16 g K_2HPO_4 and 0.326 g KH_2PO_4 in 980 ml of deionized distilled water. Adjust pH to 7.5 and bring volume to 1 liter.

Table A.6 High Ionic Strength Buffer

Using a 1,000 ml volumetric flask, dissolve 14.631 g K_2HPO_4, 2.178 g KH_2PO_4 and 182.6 g KI in 750 ml of deionized distilled water. Adjust pH to 7.5 and bring volume to 1 liter.

Table A.7 Composition of Reagents for Radioimmunoassays

A. Buffer A_1	
$NaH_2PO_4 \cdot 2\ H_2O$	6.2 g
Merthiolate (Thimersal)	0.25 g
Bovine Serum Albumin (BSA; Fraction V, Sterile, 35% solution serological, NBC, Cleveland, Ohio)	14.6 ml
Deionized distilled water	950 ml
Adjust pH to 7.5 with 5 N NaOH.	
Bring final volume to 1 liter.	
Store at 4°C (up to 3 months).	
B. 0.05 M PBS-1% BSA, pH 7.4	
NaCl	9.0 g
Dissolve with 1 liter of Buffer A_1.	
Store at 4°C (up to 3 months).	
C. Guinea Pig Anti-Bovine Insulin (GPABI) and Guinea Pig Anti-Bovine Growth Hormone (GPABGH); hereafter referred to as antibody 1.	
Antisera dilusted 1:400 with 0.05 M PBS-EDTA, pH 7.0. On day of use, dilute 1:400 antisera to required concentration using 1:400 NGPS as diluent.	
D. 0.05 M Disodium Ethylenediamine Tetraacetate (EDTA)-PBS, pH 7.0	
Disodium EDTA	18.612 g
Add 0.01 M PBS	950 ml
Adjust pH to 7.0 with 5 N NaOH.	
Dilute to 1 liter.	
Store at 4°C.	
E. 0.01 M Phosphate Buffered Saline (PBS), pH 7.0	
NaCl	143 g
Monobasic phosphate	120 ml
Dibasic phosphate	240 ml
Merthiolate (Thimersal)	1.75 g
Dissolve in deionized distilled water and transfer to large container.	
Dilute to 17.5 liters with deionized distilled water.	
Adjust pH to 7.0 with NaOH if necessary.	
Store at 4°C.	

Table A.7--Continued

F. Phosphate Buffered Saline-1% Bovine Serum Albumin (PBS-1% BSA).

BSA (Fraction V, Sterile, 35% solution serological, NBC, Cleveland, Ohio) . . . . . 50 ml
Add 1,750 ml PBS; mix.
Store at 4°C.

G. Monobasic Phosphate (0.5 M)

$NaH_2PO_4 \cdot H_2O$ . . . . . 69.05 g
Dissolve in deionized distilled water and dilute to 1 liter.

H. Dibasic Phosphate (0.5 M)

Na_2HPO_4 . . . . . 70.98 g
Dissolve in deionized distilled water, heat to dissolve and dilute to 1 liter.

I. 1:400 Normal Guinea Pig Serum (NGPS)

Obtain blood from guinea pigs not used for antibody production.
Clot the blood, harvest serum and store (-20°C).
Add 2.5 ml of serum to 1 liter volumetric flask and dilute to 1 liter with 0.05 M PBS-EDTA and store (-20°C).

J. Anti-Gamma Globulin

Use sheep anti-guinea pig gamma globulin (SAGPGG) obtained from sheep injected with guinea pig gamma globulin.
Dilute antisera 1 + 25 on day of use (one part SAGPGG serum plus 25 parts 0.05 M PBS-EDTA, pH 7.0.
Store at 4°C.

Table A.8 0.25 M Sucrose, 0.05 M Tris, 1 mM EDTA, pH 7.6

Dissolve 171.15 g of sucrose, 12.11 g of Trizma Base and .74 g of disodium ethylenediamine tetraacetate (EDTA) in a 2 liter volumetric flask. Bring volume to 2,000 ml and store at 4°C.

Table A.9 0.05 M Tris, 1 mM EDTA, pH

Dissolve 12.11 g of Trizma Base and .74 g of EDTA in a 2 liter volumetric flask. Store at 4°C.

Table A.10 100 mM KCL, 1% Triton X-100

Dissolve 14.912 g of KCL and 20 g of Triton X-100 in a 2 liter volumetric flask. Bring volume to 2,000 ml and store at 4°C.

Table A.11 100 mM KCL

Dissolve 14.912 g of KCL in a 2 liter volumetric flask. Dilute to volume and store at 4°C.

Table A.12 50 mM KCL, 1 mM Sodium Azide

Dissolve 3.728 g of KCL and .065 g of sodium azide in a 1 liter volumetric flask. Bring volume to 1 liter and store at 4°C.

Table A.13 Tracking Dye (1.0% SDS, 0.05 M Tris-HCL, pH 7.1, 20% Glycerol, 0.5% Mercaptoethanol and 0.01% Pyronin Y)

Add 40 ml of 2.5% SDS, 0.61 g of Trizma Base, 0.5 ml of 2-mercaptoethanol, 20 ml of glycerol and 0.01 g of Pyronin Y to a 100 ml volumetric flask. Bring to volume with deionized distilled water. Adjust pH to 7.2 with 6 N HCL. Store in plastic bottle at -20°C.

Table A.14 Chamber Buffer (0.1% SDS, 0.2 M Tris-Glycine, pH 8.8)

Add 100 ml of 2.0 M Tris-glycine, 40 ml of 2.5% SDS and 860 ml of deionized distilled water to a large container. Mix and store at room temperature.

Table A.15 Staining Solution (0.033% Coomassie Blue)

Add 0.1 g of Coomassie brilliant blue R250, 150 ml of methanol, 129 ml of distilled water and 21 ml of glacial acetic acid to a 500 ml beaker. Mix and store at room temperature.

Table A.16 Destaining Solution

Add 1,750 ml of distilled water, 150 ml glacial acetic acid and 100 ml methanol to a 3 liter container. Mix and store at room temperature.

APPENDIX B

RAW DATA

Table B.1 Individual Serum Insulin and Growth Hormone Data for Unselected Hereford Steers in Both Trials 1 (1975-76) and 2 (1976-77)

Pen no.	Steer no.	Ration[a]	Trial no.	Serum Insulin Data (ng/ml)					Serum Growth Hormone Data (ng/ml)				
				Day 1	Day 29	Day 57	Day 113	Day 169	Day 1	Day 29	Day 57	Day 113	Day 169
5	148	HS	1	1.372	0.891	--	2.169	1.683	10.844	5.496	6.963	8.301	34.691
	159			1.699	1.175	3.610	7.859	8.415	6.890	7.133	52.368	14.052	18.982
	166			0.824	1.263	1.813	2.325	2.019	17.318	8.663	73.335	24.765	35.884
	177			0.988	1.175	4.835	Removed	Removed	9.627	10.551	18.076	Removed	Removed
	178			0.795	1.857	--	6.972	2.808	27.924	36.364	21.177	2.361	18.425
	197			0.977	1.104	2.044	2.802	2.045	4.182	4.555	98.200	5.982	48.458
5	189	HS	2	1.490	2.078	1.544	3.324	2.208	23.104	5.894	2.496	22.743	12.682
	127			1.657	3.526	1.525	4.961	2.924	3.228	7.403	13.029	17.373	7.512
	165			1.489	4.724	3.096	4.154	4.013	4.010	7.059	2.636	13.827	34.184
	188			1.539	1.294	2.369	3.884	2.752	3.524	9.870	4.325	11.344	97.216
	177			0.895	3.037	3.700	8.432	2.728	5.992	2.560	5.905	16.004	2.588
	195			1.402	2.825	8.933	2.665	2.987	5.904	1.267	2.846	1.538	39.181
11	142	HG	1	1.584	2.032	2.699	13.610	13.499	6.558	4.241	2.223	19.119	10.704
	144			2.349	2.072	5.109	7.983	11.942	5.705	21.621	6.220	1.725	8.216
	165			0.942	2.844	8.871	7.530	--	2.610	12.572	2.887	1.975	--
	176			2.185	3.160	9.168	6.574	5.568	11.894	10.101	1.999	1.989	3.946
	181			0.825	1.770	3.277	12.111	10.824	4.812	8.927	2.237	1.513	12.984
	187			0.891	1.959	5.704	8.762	6.718	4.321	15.515	4.561	8.513	10.655
11	175	HG	2	1.585	10.189	5.101	10.634	4.000	3.874	4.755	11.836	23.240	22.367
	153			1.451	4.317	3.881	7.987	8.708	2.791	4.345	1.810	1.896	11.352
	184			1.782	3.347	6.970	7.258	7.910	25.077	8.105	24.495	55.376	41.877
	194			2.156	5.171	5.166	7.915	7.866	20.887	6.175	1.036	75.937	6.083
	167			2.084	3.756	6.799	14.694	5.667	2.781	5.686	4.798	10.551	4.397
	191			0.766	5.845	9.171	7.961	12.706	18.646	8.064	1.910	20.540	8.366

[a]HS = high silage; HG = high grain.

Table B.2 Individual Serum Insulin and Growth Hormone Data for Selected Hereford Steers in Both Trials 1 (1975-76) and 2 (1976-77)

Pen no.	Steer no.	Ration[a]	Trial no.	Serum Insulin Data (ng/ml)					Serum Growth Hormone Data (ng/ml)				
				Day 1	Day 29	Day 57	Day 113	Day 169	Day 1	Day 29	Day 57	Day 113	Day 169
6	111	HS	1	1.279	3.956	3.445	5.104	2.308	16.201	3.435	2.829	2.855	3.796
	113			2.282	1.457	1.093	1.844	1.772	38.287	12.279	1.785	3.918	4.844
	115			2.574	1.196	2.454	2.709	2.031	4.828	12.402	1.547	3.687	4.750
	118			1.798	2.554	--	3.994	2.941	4.219	7.835	3.590	2.761	22.440
	150			1.756	2.447	6.857	5.915	5.647	7.832	10.438	17.515	2.590	21.171
	188			2.962	1.120	2.374	2.378	3.942	7.488	22.372	2.248	2.731	35.049
	190			0.808	0.824	0.832	1.459	2.393	6.687	4.941	8.714	9.677	11.912
6	150	HS	2	1.198	3.282	2.848	2.469	2.394	4.489	12.674	5.538	23.619	14.959
	112			3.904	3.770	1.187	1.869	3.997	3.575	8.966	3.552	4.347	27.463
	121			0.970	2.120	2.561	3.141	1.706	4.747	6.676	3.234	1.937	2.870
	154			0.859	1.295	6.933	2.009	2.448	7.428	9.391	15.525	22.487	11.764
	173			1.120	2.033	5.736	3.550	3.450	3.471	8.084	2.012	10.659	27.323
	179			2.309	2.225	6.403	2.760	3.520	6.154	6.220	9.259	14.657	30.442
	123			1.413	2.220	2.019	3.543	2.297	7.617	13.449	11.277	57.958	30.514
	162			1.528	2.033	4.978	3.211	2.868	3.668	8.094	3.870	12.782	6.063
12	119	HG	1	1.077	1.914	4.491	4.107	7.564	4.433	7.768	3.355	7.399	19.827
	120			0.822	1.613	6.985	10.298	6.147	4.675	3.984	1.795	4.238	4.176
	139			1.137	1.883	3.886	6.920	5.849	9.989	20.722	2.967	10.722	16.431
	152			4.002	1.501	2.155	6.958	8.218	3.258	25.007	1.299	2.271	2.852
	155			1.573	3.160	14.708	16.449	6.436	6.276	6.221	1.268	2.947	6.792
	193			1.015	1.542	4.177	10.898	3.262	5.921	10.568	17.796	13.805	17.350
	195			0.832	3.182	2.454	8.263	3.123	5.496	15.344	1.749	20.243	11.749
	890			0.752	2.647	5.713	2.770	3.207	4.262	3.844	1.468	30.255	22.019
12	132	HG	2	1.975	4.606	3.434	6.929	13.272	4.085	4.098	5.756	1.632	3.300
	158			1.055	3.782	5.909	13.994	5.484	4.270	2.854	1.247	2.018	1.957
	192			1.060	16.570	6.356	10.550	9.457	3.918	2.598	1.695	2.119	2.315
	178			0.624	6.108	5.835	4.748	5.482	4.949	3.624	14.300	10.061	2.433
	164			1.226	2.842	3.522	13.170	8.376	9.330	3.641	2.279	5.619	1.838
	182			1.400	3.993	3.327	5.229	4.532	2.308	3.549	2.144	3.420	12.448
	130			0.854	2.738	3.131	3.313	1.688	7.263	7.869	2.223	21.823	39.600
	149			0.840	4.345	1.665	2.178	3.948	7.095	4.072	2.782	2.730	3.662

[a]HS = high silage; HG = high grain.

Table B.3 Individual Serum Insulin and Growth Hormone Data for Angus x Hereford x Charolais Steers in Both Trials 1 (1975-76) and 2 (1976-77)

Pen no.	Steer no.	Ration[a]	Trial no.	Serum Insulin Data (ng/ml)					Serum Growth Hormone Data (ng/ml)				
				Day 1	Day 29	Day 57	Day 113	Day 169	Day 1	Day 29	Day 57	Day 113	Day 169
9	122	HS	1	0.933	1.169	0.818	1.279	4.249	5.788	16.960	21.862	10.916	9.936
	133			0.988	1.057	0.882	1.770	1.311	19.187	14.046	8.012	10.371	4.661
	149			0.729	1.542	1.120	2.227	3.294	11.185	62.171	37.923	31.484	13.813
	161			1.415	1.415	1.857	2.789	1.774	4.875	9.205	3.155	9.028	4.663
	164			2.347	1.670	1.169	2.746	1.567	13.317	4.654	7.831	3.140	26.260
	175			0.977	2.757	0.916	2.106	1.361	37.893	29.171	23.714	3.750	99.935
	182			--	1.976	--	1.276	0.921	--	16.571	25.834	20.055	18.703
	186			1.120	2.173	2.870	3.910	2.460	8.959	13.727	2.312	5.271	3.159
9	135	HS	2	1.361	1.679	4.701	2.383	1.220	7.178	21.635	1.873	1.821	94.367
	166			1.673	2.857	4.958	2.568	2.716	9.594	6.956	30.790	65.103	11.282
	146			1.297	1.889	2.120	2.346	1.870	4.045	11.717	3.318	3.484	6.352
	131			1.703	3.789	8.331	2.002	1.176	4.857	11.087	5.892	12.154	77.149
	186			1.427	3.184	1.197	3.855	2.676	10.004	2.398	21.654	12.770	35.663
	168			1.386	2.361	4.377	2.749	0.922	18.168	8.934	6.786	38.463	39.281
	129			1.143	3.126	3.985	2.967	2.874	20.466	20.626	51.584	6.038	22.017
	183			1.594	5.021	--	3.844	1.824	5.704	7.857	--	71.844	93.220
13	117	HG	1	2.494	4.136	3.085	2.878	4.644	28.269	2.649	1.749	40.976	18.081
	124			1.004	4.044	1.336	2.788	6.409	7.564	5.944	5.307	75.206	8.183
	147			2.521	2.163	4.205	8.849	5.342	5.361	10.769	10.210	14.219	4.439
	163			1.855	6.316	5.522	11.036	9.289	9.903	12.547	36.179	15.051	28.294
	154			1.331	2.897	4.706	3.750	7.030	10.401	15.680	6.533	39.262	13.259
	158			0.891	2.647	9.192	6.560	21.176	20.613	59.733	51.755	19.559	13.217
	179			0.832	1.040	6.541	6.874	4.710	8.336	2.509	4.182	2.486	4.240
	189			2.841	3.702	6.781	3.783	9.250	3.493	2.636	1.424	3.175	2.862
13	110	HG	2	0.882	4.660	3.458	5.472	4.831	2.684	2.655	7.024	46.863	21.638
	104			1.285	5.919	2.482	3.597	6.129	3.646	3.943	2.279	2.148	4.164
	169			1.819	3.220	4.660	6.274	8.041	11.535	9.968	3.014	9.642	2.656
	145			1.343	2.315	5.279	4.351	5.259	10.100	4.564	37.683	1.943	39.238
	147			1.640	2.177	4.415	6.301	5.784	13.310	3.827	4.164	1.715	2.706
	128			1.360	2.901	5.776	8.798	9.710	14.729	3.608	2.327	4.027	2.002
	163			1.339	1.739	7.900	9.985	3.554	7.476	3.757	16.737	8.154	7.120
	125			1.287	--	3.679	7.741	9.978	15.278	--	18.179	5.073	16.975

[a]HS = high silage; HG = high grain.

Table B.4 Individual Serum Insulin and Growth Hormone Data for Angus x Hereford x Holstein Steers in Both Trials 1 (1975-76) and 2 (1976-77)

Pen no.	Steer no.	Ration[a]	Trial no.	Serum Insulin Data (ng/ml)					Serum Growth Hormone Data (ng/ml)				
				Day 1	Day 29	Day 57	Day 113	Day 169	Day 1	Day 29	Day 57	Day 113	Day 169
10	109	HS	1	2.385	1.870	2.534	1.819	3.009	13.099	53.809	42.039	8.141	43.743
	114			1.415	1.185	2.347	1.181	--	20.848	5.885	39.973	9.015	--
	116			5.110	1.855	6.697	7.032	8.891	18.944	8.835	27.245	12.928	10.540
	121			1.014	2.173	4.510	3.788	3.890	12.908	6.512	1.541	4.874	3.713
	130			1.743	1.040	2.605	2.127	2.092	18.782	20.397	39.544	52.344	35.691
	167			4.046	2.230	7.130	4.407	4.580	33.861	7.802	1.939	2.790	3.642
	169			4.071	1.727	3.205	2.144	6.146	17.673	22.464	5.333	--	47.761
	191			1.232	0.739	Removed	Removed	Removed	7.341	12.216	Removed	Removed	Removed
10	137	HS	2	2.427	2.310	3.441	2.651	3.352	6.506	4.831	8.497	3.894	9.335
	181			1.134	3.560	1.571	3.352	1.605	4.724	3.048	2.800	2.319	7.685
	102			2.757	1.693	8.911	3.907	1.283	9.983	30.795	25.619	14.588	18.323
	176			2.358	2.085	5.335	3.671	3.769	4.074	3.748	11.225	2.006	8.067
	117			1.807	4.187	2.714	3.549	3.994	19.221	6.967	17.358	2.361	3.604
	136			1.470	3.690	7.461	2.217	4.881	4.104	5.650	12.347	13.587	45.645
	109			1.355	2.986	4.700	6.920	3.331	5.652	4.169	1.781	1.698	11.948
	193			1.503	3.126	6.152	3.710	3.338	5.344	41.066	6.857	9.100	95.234
14	107	HG	1	2.292	3.865	2.547	6.801	1.697	21.915	7.267	2.807	7.990	51.593
	110			1.927	8.297	9.804	5.345	7.238	17.493	29.483	13.446	20.953	23.105
	132			1.049	5.620	5.665	12.672	16.274	5.354	18.385	9.220	21.709	18.353
	134			1.943	5.488	7.703	--	2.741	40.050	15.748	48.344	--	4.012
	135			2.032	1.513	4.064	6.031	2.314	15.833	11.095	15.272	10.642	8.862
	146			3.477	7.008	17.072	21.748	21.220	5.729	3.688	10.144	8.443	5.280
	158			2.494	8.445	5.994	4.030	3.766	4.519	5.849	14.868	13.747	33.604
	171			1.104	10.920	16.522	18.405	15.220	3.917	2.915	1.421	3.490	8.839
14	124	HG	2	1.950	5.101	3.715	6.594	9.071	13.880	26.862	86.275	13.143	4.610
	119			1.556	--	6.296	8.695	5.664	11.250	--	11.433	18.181	11.618
	103			2.109	5.031	3.228	5.675	6.704	12.519	4.366	27.064	4.350	4.232
	170			1.575	3.579	9.285	8.997	17.586	9.509	5.633	4.090	14.552	5.714
	114			1.175	4.289	2.667	2.477	2.457	19.893	5.878	4.323	12.018	37.782
	141			2.412	--	1.844	11.962	8.682	13.389	--	15.825	2.954	15.497
	151			2.002	2.997	4.984	7.219	6.257	17.878	7.048	15.196	2.368	6.725
	138			0.921	--	3.395	4.834	4.857	13.790	--	1.110	2.487	12.230

[a]HS = high silage; HG = high grain.

Table B.5 Semitendinosus Muscle Data from Initial Slaughter Steers in Both Trials 1 (1975-76) and 2 (1976-77)

Steer no.	Brg.[a] group	Live wt, kg	Trial no.	ST wt,[b] g	Proximate analysis, %, Wet basis				Nucleic acids, Mg/g wet muscle		Protein fractionation[c], Percentage of total protein nitrogen			
					Moisture	Protein	Fat	Ash	DNA	RNA	Sarco-plasmic	Fibrillar	NPN	Stroma
162	1	150.0	1	592	77.43	19.17	1.37	2.03	0.3698	1.5410	--	--	--	--
128		184.1	1	825	74.39	20.73	1.99	2.89	0.2956	1.3208	--	--	--	--
191		195.5	2	839	76.62	21.01	1.55	0.82	0.3891	1.2180	16.94	52.48	12.03	18.55
199		170.5	2	663	76.14	20.83	1.95	1.08	0.3862	1.2105	19.33	53.51	12.24	14.92
205		115.9	2	434	77.18	19.69	1.21	1.92	0.4933	1.5515	17.36	57.72	13.07	11.84
170	2	236.4	1	849	71.96	21.81	4.10	2.13	0.4101	1.7300	--	--	--	--
180		218.2	1	783	75.77	20.68	1.18	2.37	0.3303	1.2760	--	--	--	--
194		181.8	1	730	74.91	19.67	2.33	3.09	0.3683	1.4230	--	--	--	--
193		227.3	2	901	75.77	20.73	1.85	1.65	0.3474	1.1155	20.53	55.44	12.81	11.22
190		213.6	2	949	77.04	20.23	1.74	0.99	0.3407	0.8233	20.71	47.87	12.21	19.21
202		150.0	2	603	77.75	20.16	0.85	1.24	0.3576	1.5390	17.48	53.51	12.93	16.09
108	3	238.6	1	1,021	74.90	21.54	0.90	2.66	0.3337	1.5540	--	--	--	--
138		234.1	1	748	72.26	22.10	2.94	2.70	0.3930	1.1430	--	--	--	--
105		290.9	1	1,465	75.42	21.24	1.03	2.31	0.3052	0.9414	--	--	--	--
192		261.4	2	1,116	77.27	19.75	1.20	1.78	0.3234	1.1350	19.06	55.26	12.82	12.86
126		250.0	2	1,229	77.16	19.66	2.18	1.00	0.3793	1.2590	17.50	53.49	12.40	16.61
195		227.3	2	873	75.85	20.83	1.84	1.48	0.4836	1.1940	19.16	52.70	12.49	15.64
101	4	270.5	1	935	74.30	21.21	2.46	2.03	0.4019	1.5090	--	--	--	--
185		275.0	1	1,220	73.94	21.16	2.02	2.88	0.3292	1.5760	--	--	--	--
187		261.4	2	1,219	77.38	20.15	1.57	0.90	0.3901	0.7639	20.78	54.02	10.46	14.73
198		225.0	2	918	76.03	20.39	2.01	1.57	0.3660	1.3010	20.37	54.69	11.62	13.32
185		215.9	2	729	77.27	20.44	1.18	1.11	0.4330	0.9125	18.58	53.14	12.47	15.81
188		284.1	2	1,362	75.85	20.57	1.80	1.78	0.3269	1.3100	20.30	57.28	12.44	9.99

[a] 1 = Unselected Hereford; 2 = Selected Hereford; 3 = Angus x Hereford x Charolais; 4 = Angus x Hereford x Holstein.

[b] Weight of semitendinosus muscle.

[c] Missing values due to freezer malfunction.

Table B.6 Semitendinosus Muscle Data from Finished Steers (Terminal Slaughter) in Both Trials 1 (1975-76) and 2 (1976-77)

Steer no.	Brg.[a] group	Ration[b]	Live wt, kg	Days on feed	Trial no.	ST wt,[c] g	Proximate analysis, %, Wet basis: Moisture	Protein	Fat	Ash	Nucleic acids, Mg/g wet muscle: DNA	RNA	Protein fractionation[d], % of total protein nitrogen: Sarcoplasmic	Fibrillar	NPN	Stroma
165	1	HG	377.3	204	1	1,322	69.46	21.54	7.58	1.42	0.2754	0.8315	--	--	--	--
166		HS	397.7	253	1	2,021	72.39	20.57	4.91	2.13	0.2738	0.8540	--	--	--	--
197		HS	397.7	337	1	1,428	71.96	20.37	6.71	0.96	0.3328	0.7743	19.79	51.46	12.16	16.59
194		HG	486.4	222	2	1,758	73.86	20.61	4.95	0.58	0.2844	0.6973	22.44	52.26	11.95	13.36
184		HG	463.6	222	2	1,624	72.15	21.34	5.48	1.03	0.3347	0.9406	20.90	51.40	12.02	15.68
188		HS	427.3	271	2	1,710	74.82	21.50	3.33	0.88	0.2916	0.7184	21.36	51.44	12.19	15.01
191		HG	445.5	271	2	1,577	72.82	21.77	4.97	0.44	0.3342	0.6812	21.94	50.39	11.27	16.40
195		HS	472.7	327	2	1,901	73.19	22.01	4.66	0.14	0.2001	0.7982	21.21	49.06	10.75	18.98
127		HS	461.4	327	2	1,655	73.15	22.28	4.41	0.16	0.2161	0.8123	29.27	49.80	2.95	17.99
155	2	HG	493.2	204	1	1,927	71.09	21.34	5.27	2.30	0.3141	1.0140	--	--	--	--
111		HS	481.8	253	1	1,983	72.43	21.11	4.37	2.09	0.3152	1.1390	--	--	--	--
113		HS	459.1	337	1	1,635	73.69	21.31	3.79	1.21	0.3228	0.7983	18.56	51.27	14.64	15.33
158		HG	515.9	222	2	1,669	71.34	20.93	6.58	1.15	0.3068	1.0480	24.17	52.42	8.57	14.84
178		HG	509.1	222	2	2,261	73.69	21.28	3.64	1.39	0.3161	1.0450	24.55	53.47	8.14	13.83
149		HG	472.7	271	2	1,686	74.69	20.61	4.34	0.36	0.3240	0.5580	21.91	50.00	11.72	16.37
154		HS	468.2	271	2	1,979	75.56	21.32	2.54	0.58	0.1912	0.7680	28.80	53.61	5.23	12.36
162		HS	486.4	327	2	2,019	75.17	21.41	3.41	0.01	0.2235	0.7645	23.15	52.52	8.03	16.30
112		HS	536.4	327	2	2,094	66.78	18.83	14.26	0.13	0.2400	1.2277	27.22	45.42	7.16	20.21
189	3	HG	484.1	204	1	1,966	69.28	20.86	8.40	1.46	0.3591	0.9015	--	--	--	--
161		HS	531.8	253	1	2,726	73.63	21.71	3.04	1.62	0.3177	1.2960	--	--	--	--
182		HS	522.7	337	1	2,361	73.48	20.67	4.67	1.18	0.3079	0.7203	21.52	49.59	12.54	16.35
145		HG	543.2	222	2	2,750	73.22	21.02	4.38	1.38	0.3307	1.1180	21.10	51.70	12.42	14.78
147		HG	545.5	222	2	1,937	71.78	21.04	5.94	1.24	0.3336	1.0030	24.89	48.06	8.67	18.38
168		HS	550.0	271	2	2,239	74.81	21.70	3.06	0.44	0.3158	0.5898	20.21	52.14	12.21	15.43
125		HG	572.7	271	2	2,202	74.76	20.38	4.34	0.53	0.3169	0.7040	20.62	51.00	11.99	16.38
183		HS	550.0	327	2	2,562	73.13	21.98	4.64	0.25	0.2540	1.0541	21.87	49.96	9.38	18.78
129		HS	559.1	327	2	2,510	70.98	21.50	7.05	0.48	0.2154	1.1354	25.72	52.68	6.53	15.07
171	4	HG	534.1	204	1	1,677	65.35	19.45	13.34	1.86	0.2606	0.8898	--	--	--	--
109		HS	515.9	253	1	1,862	71.45	21.95	4.73	1.87	0.2714	0.9420	--	--	--	--
114		HS	525.0	337	1	2,446	74.41	20.40	3.56	1.63	0.2959	0.7529	23.89	49.31	12.81	13.98
170		HG	550.0	222	2	2,085	70.72	20.25	8.17	0.86	0.3300	0.8377	24.65	50.63	8.50	16.22
103		HG	536.4	222	2	1,817	72.23	21.22	5.19	1.36	0.3713	1.2915	25.08	52.03	8.13	14.77
136		HS	563.6	271	2	1,834	73.61	21.80	4.18	0.41	0.4162	0.6930	19.99	50.07	11.99	17.95
138		HG	568.2	271	2	2,180	74.33	22.09	2.88	0.70	0.3583	0.6657	22.07	53.62	11.77	12.54
193		HS	584.1	327	2	2,208	73.35	20.71	5.94	0.00	0.2512	0.7984	23.30	51.19	7.87	17.64
117		HS	597.7	327	2	2,401	73.30	21.89	4.21	0.59	0.2256	1.0655	30.36	40.45	2.30	26.89

[a] 1 = Unselected Hereford; 2 = Selected Hereford; 3 = Angus x Hereford x Charolais; 4 = Angus x Hereford x Holstein.

[b] HS = high silage; HG = high grain.

[c] Weight of semitendinosus muscle.

[d] Missing values due to freezer malfunction.

Table B.7 Individual Performance Data for Unselected Hereford Steers in Both Trials 1 (1975-76) and 2 (1976-77)

Pen no.	Steer no.	Ration[a]	Trial no.	Day 1 Initial live wt, kg	Day 29 Live wt, kg	Day 29 ADG, kg	Day 57 Live wt, kg	Day 57 ADG, kg	Day 113 Live wt, kg	Day 113 ADG, kg	Day 169 Live wt, kg	Day 169 ADG, kg
5	148	HS	1	143.2	163.6	0.73	177.3	0.49	243.2	1.22	297.7	1.02
	159			213.6	227.3	0.49	247.7	0.73	329.5	1.54	397.7	1.18
	166			197.7	218.2	0.73	234.1	0.57	295.5	1.22	336.4	0.78
	177			168.2	195.5	0.97	225.0	1.05	Removed	--	Removed	--
	178			168.2	195.5	0.97	220.5	0.89	302.3	1.62	365.9	1.10
	197			129.5	154.5	0.89	175.0	0.73	234.1	1.05	284.1	0.94
5	189	HS	2	211.4	238.6	0.97	275.0	1.40	329.5	0.73	381.8	0.40
	127			186.4	211.4	0.89	240.9	1.14	281.8	0.25	336.4	0.81
	165			181.8	197.7	0.57	213.6	0.61	272.7	0.81	343.2	0.73
	188			172.7	188.6	0.57	215.9	1.05	265.9	0.97	336.4	1.14
	177			165.9	188.6	0.81	220.5	1.22	270.5	0.65	343.2	1.05
	195			154.5	170.5	0.57	200.0	1.14	263.6	0.97	331.8	1.14
11	142	HG	1	154.5	186.4	1.14	218.2	1.14	286.4	0.89	352.3	1.25
	144			225.0	268.2	1.54	304.5	1.30	379.5	0.89	454.5	1.33
	165			163.6	195.5	1.14	229.5	1.22	288.6	0.97	354.5	1.18
	176			179.5	227.3	1.70	268.2	1.46	354.5	1.38	456.8	1.96
	181			109.1	143.2	1.22	156.8	0.49	245.5	1.30	336.4	1.41
	187			138.6	172.7	1.22	211.4	1.38	270.5	1.05	313.6	0.63
11	175	HG	2	225.0	250.0	0.89	293.2	1.66	363.6	1.22	463.6	1.62
	153			184.1	204.5	0.73	240.9	1.40	311.4	1.05	397.7	1.62
	184			181.8	213.6	1.14	247.7	1.31	325.0	1.30	411.4	1.62
	194			175.0	218.2	1.54	261.4	1.66	336.4	1.14	431.8	1.22
	167			161.4	186.4	0.89	227.3	1.57	290.9	1.14	372.7	1.14
	191			134.1	163.6	1.05	195.5	1.22	250.0	0.73	329.5	1.05

[a]HS = high silage; HG = high grain.

Table B.8 Individual Performance Data for Selected Hereford Steers in Both Trials 1 (1975-76) and 2 (1976-77)

Pen no.	Steer no.	Ration[a]	Trial no.	Day 1 Initial live wt, kg	Day 29 Live wt, kg	Day 29 ADG, kg	Day 57 Live wt, kg	Day 57 ADG, kg	Day 113 Live wt, kg	Day 113 ADG, kg	Day 169 Live wt, kg	Day 169 ADG, kg
6	111	HS	1	240.9	279.5	1.38	315.9	1.30	363.6	0.40	404.5	0.78
	113			268.2	200.0	0.81	225.0	0.89	268.2	0.73	315.9	0.78
	115			243.2	272.7	1.05	304.5	1.14	359.1	0.73	404.5	0.94
	118			218.2	263.6	1.62	297.7	1.22	359.1	0.97	411.4	0.86
	150			197.7	229.5	1.14	259.1	1.05	318.2	0.89	368.2	1.02
	188			197.7	231.8	1.22	259.1	0.97	318.2	0.97	377.3	0.94
	190			193.2	225.0	1.14	256.8	1.14	327.3	1.22	388.6	1.10
6	150	HS	2	245.5	243.2	-0.08	268.2	0.96	347.7	1.05	425.0	1.30
	112			220.5	243.2	0.81	270.5	1.05	327.3	0.89	393.2	0.81
	121			204.5	231.8	0.97	263.6	1.22	322.7	0.65	381.8	0.57
	154			204.5	227.3	0.81	254.5	1.05	311.4	0.81	384.1	0.97
	173			184.1	211.4	0.97	236.4	0.96	302.3	1.14	377.3	1.22
	179			181.8	211.4	1.05	240.9	1.14	306.8	0.73	379.5	0.81
	123			175.0	204.5	1.05	227.3	0.87	290.9	1.14	350.0	1.22
	162			145.5	179.5	1.22	204.5	0.96	259.1	0.81	322.7	0.81
12	119	HG	1	229.5	268.2	1.38	300.0	1.14	393.2	1.62	481.8	1.33
	120			175.0	225.0	1.79	281.8	2.03	395.5	1.87	481.8	1.57
	139			256.8	263.6	0.25	320.5	2.03	413.6	1.70	463.6	0.63
	152			195.5	220.5	0.89	250.0	1.05	329.5	1.38	393.2	1.02
	155			204.5	236.4	1.14	295.5	2.11	379.5	1.14	454.5	1.49
	193			170.5	206.8	1.30	247.7	1.46	327.3	1.38	397.7	1.18
	195			154.5	188.6	1.22	227.3	1.38	302.3	1.38	370.5	1.02
	890			204.5	236.4	1.14	290.9	1.95	377.3	1.54	443.2	1.57
12	132	HG	2	306.8	350.0	1.54	390.9	1.57	481.8	1.14	577.3	1.62
	158			220.5	254.5	1.22	286.4	1.22	372.7	1.38	454.5	1.46
	192			204.5	238.6	1.22	277.3	1.49	356.8	1.38	463.6	1.87
	178			200.0	231.8	1.14	265.9	1.31	345.5	1.14	411.4	1.05
	164			197.7	229.5	1.14	263.6	1.31	347.7	1.46	434.1	1.54
	182			175.0	215.9	1.46	245.5	1.14	322.7	1.30	409.1	1.46
	130			147.7	177.3	1.05	202.3	0.96	261.4	0.97	336.4	1.62
	149			140.9	179.5	1.38	209.1	1.14	279.5	0.89	368.2	1.54

[a]HS = high silage; HG = high grain.

Table B.9 Individual Performance Data for Angus x Hereford x Charolais Steers in Both Trials 1 (1975-76) and 2 (1976-77)

Pen no.	Steer no.	Ration[a]	Trial no.	Day 1 Initial live wt, kg	Day 29 Live wt, kg	Day 29 ADG, kg	Day 57 Live wt, kg	Day 57 ADG, kg	Day 113 Live wt, kg	Day 113 ADG, kg	Day 169 Live wt, kg	Day 169 ADG, kg
9	122	HS	1	240.9	268.2	0.97	306.8	1.38	393.2	1.05	456.8	1.02
	133			297.7	329.5	1.14	350.0	0.73	406.8	0.57	454.5	1.10
	149			215.9	259.1	1.54	288.6	1.05	370.5	0.81	440.9	1.49
	161			247.7	290.9	1.54	327.3	1.30	370.5	0.57	436.4	1.41
	164			286.4	318.2	1.14	336.4	0.65	422.7	0.73	431.8	-0.24
	175			195.5	229.5	1.22	268.2	1.38	329.5	0.49	381.8	0.94
	182			229.5	252.3	0.81	272.7	0.73	336.4	0.49	384.1	0.63
	186			200.0	240.9	1.46	268.2	0.97	340.9	1.05	400.0	1.02
9	135	HS	2	277.3	309.1	1.14	340.9	1.22	422.7	1.46	490.9	1.14
	166			256.8	295.5	1.38	322.7	1.05	404.5	0.81	484.1	1.14
	146			254.5	290.9	1.30	309.1	0.70	384.1	0.81	452.3	1.05
	131			245.5	293.2	1.70	320.5	1.05	393.2	0.73	447.7	1.22
	136			229.5	265.9	1.30	288.6	0.87	359.1	1.05	429.5	0.97
	168			225.0	268.2	1.54	302.3	1.31	379.5	1.22	438.6	1.30
	129			222.7	263.6	1.46	290.9	1.05	354.5	0.81	415.9	0.89
	183			222.7	252.3	1.05	272.7	0.79	345.5	1.14	400.0	1.22
13	117	HG	1	250.0	265.9	0.57	309.1	1.54	377.3	0.97	447.7	1.65
	124			252.3	275.0	0.81	309.1	1.22	379.5	1.22	468.2	1.72
	147			302.3	334.1	1.14	388.6	1.95	465.9	0.81	536.4	1.25
	153			286.4	338.6	1.87	404.5	2.35	490.9	1.30	602.3	2.43
	154			213.6	265.9	1.87	318.2	1.87	370.5	0.57	452.3	1.49
	168			252.3	284.1	1.14	331.8	1.70	409.1	1.05	493.2	2.12
	179			188.6	218.2	1.05	268.2	1.79	361.4	1.54	427.3	1.49
	189			227.3	263.6	1.30	320.5	2.03	379.5	1.14	450.0	1.80
13	110	HG	2	284.1	313.6	1.05	331.8	0.70	425.0	1.05	520.5	1.62
	104			259.1	295.5	1.30	345.5	1.92	438.6	1.22	513.6	0.89
	169			247.7	288.6	1.46	322.7	1.31	390.9	0.73	477.3	1.54
	145			240.9	275.0	1.22	336.4	2.36	425.0	1.22	497.7	1.22
	147			227.3	261.4	1.22	311.4	1.92	402.3	1.46	495.5	1.70
	128			227.3	263.6	1.30	313.6	1.92	404.5	0.89	486.4	0.81
	163			225.0	256.8	1.14	304.5	1.84	384.1	1.05	461.4	1.14
	125			218.2	238.6	0.73	277.3	1.49	363.6	1.05	440.9	1.30

[a]HS = high silage; HG = high grain.

Table B.10 Individual Performance Data for Angus x Hereford x Holstein Steers in Both Trials 1 (1975-76) and 2 (1976-77)

Pen no.	Steer no.	Ration[a]	Trial no.	Day 1 Initial live wt, kg	Day 29 Live wt, kg	Day 29 ADG, kg	Day 57 Live wt, kg	Day 57 ADG, kg	Day 113 Live wt, kg	Day 113 ADG, kg	Day 169 Live wt, kg	Day 169 ADG, kg
10	109	HS	1	256.8	295.5	1.38	331.8	1.30	390.9	0.57	452.3	1.02
	114			254.5	297.7	1.54	329.5	1.14	377.3	0.49	422.7	1.02
	116			286.4	331.8	1.62	379.5	1.70	452.3	1.14	513.6	0.86
	121			275.0	309.1	1.22	350.0	1.46	425.0	1.05	472.7	1.18
	130			268.2	311.4	1.54	350.0	1.38	409.1	0.89	454.5	1.41
	167			263.6	304.5	1.46	345.5	1.46	402.3	0.73	463.6	1.10
	169			238.6	277.3	1.38	306.8	1.05	365.9	0.65	420.5	1.18
	191			197.7	222.7	0.89	247.7	0.89	Removed	--	Removed	--
10	137	HS	2	277.3	302.3	0.89	329.5	1.05	402.3	1.14	468.2	0.89
	181			265.9	295.5	1.05	320.5	0.96	379.5	0.65	459.1	1.22
	102			263.6	313.6	1.77	347.7	1.31	415.9	0.89	472.7	0.32
	176			259.1	300.0	1.46	318.2	0.70	397.7	1.14	477.3	1.22
	117			250.0	295.5	1.62	322.7	1.05	384.1	0.65	447.7	0.73
	136			243.2	275.0	1.14	311.4	1.40	397.7	1.22	470.5	1.05
	109			231.8	288.6	2.03	311.4	0.87	375.0	0.97	438.6	1.05
	193			220.5	247.7	0.97	268.2	0.79	340.9	0.89	427.3	1.30
14	107	HG	1	206.8	238.6	1.14	265.9	0.97	327.3	1.05	372.7	1.02
	110			265.9	272.7	0.25	340.9	2.44	413.6	0.97	479.5	1.18
	132			279.5	329.5	1.79	363.6	1.22	459.1	1.79	520.5	1.02
	134			252.3	288.6	1.30	327.3	1.38	395.5	1.22	450.0	1.49
	135			286.4	329.5	1.54	372.7	1.54	461.4	1.14	534.1	1.49
	146			284.1	329.5	1.62	377.3	1.70	465.9	0.73	550.0	1.57
	158			272.7	318.2	1.62	370.5	1.87	454.5	1.38	522.7	1.10
	171			247.7	295.5	1.70	334.1	1.38	413.6	0.57	504.5	1.57
14	124	HG	2	275.0	297.7	0.81	336.4	1.49	409.1	0.89	497.7	1.70
	119			272.7	306.8	1.22	352.3	1.75	443.2	1.38	529.5	1.70
	103			261.4	302.3	1.46	336.4	1.31	397.7	0.97	472.7	1.14
	170			256.8	281.8	0.89	329.5	1.84	406.8	1.05	493.2	1.70
	114			250.0	284.1	1.22	329.5	1.75	415.9	1.38	475.0	0.97
	141			247.7	275.0	0.97	313.6	1.49	356.8	0.25	438.6	0.97
	151			227.3	247.7	0.73	302.3	2.10	368.2	0.65	465.9	1.62
	138			211.4	238.6	0.97	288.6	1.92	359.1	1.22	445.5	1.46

[a]HS = high silage; HG = high grain.

Table B.11 Anterior Pituitary Data from Initial Slaughter Steers in Trial 2 (1976-77)[a]

Steer no.	Brg. group[b]	Live wt, kg	AP wt,[c] g	AP conc,[d] µg/ml
191	1	195.5	0.808	681.79
199		170.5	0.653	744.25
205		115.9	0.547	759.04
193	2	227.3	0.717	424.28
190		213.6	0.765	692.06
202		150.0	0.790	362.22
192	3	261.4	0.832	592.75
126		250.0	0.852	828.88
195		227.3	0.650	687.71
187	4	261.4	0.987	500.55
198		225.0	0.765	641.63
185		215.9	0.735	640.12
188		284.1	0.927	471.52

[a]Anterior pituitaries from trial 1 (1975-76) missing due to freezer malfunction.

[b]1 = Unselected Hereford; 2 = Selected Hereford; 3 = Angus x Hereford x Charolais; 4 = Angus x Hereford x Holstein.

[c]Weight of anterior pituitary.

[d]Anterior pituitary concentration of the 30 mg/ml extract.

Table B.12 Anterior Pituitary Data from Finished Steers (Terminal Slaughter) in Trial 2 (1976-77)[a]

Steer no.	Brg. group[b]	Ration[c]	Live wt, kg	AP wt,[d] g	AP conc,[e] µg/ml
194	1	HG	486.4	1.758	158.04
184		HG	463.6	1.640	189.66
188		HS	427.3	1.150	255.79
191		HG	445.5	1.497	214.17
195		HS	472.7	1.595	247.55
127		HS	461.4	1.461	238.49
158	2	HG	515.9	1.130	425.78
178		HG	509.1	1.310	307.52
149		HG	472.7	1.400	245.52
154		HS	468.2	1.443	209.32
162		HS	486.4	1.321	379.36
112		HS	536.4	1.565	195.31
145	3	HG	543.2	1.760	249.45
147		HG	545.5	2.500	156.48
168		HS	550.0	1.808	313.35
125		HG	572.7	1.763	205.10
183		HS	550.0	1.472	261.26
129		HS	559.1	1.387	372.28
170	4	HG	550.0	1.740	213.90
103		HG	536.4	2.850	148.08
136		HS	563.6	2.360	215.81
138		HG	568.2	2.133	171.06
193		HS	584.1	2.100	325.06
117		HS	597.7	1.924	359.69

[a]Anterior pituitaries from trial 1 (1975-76) missing due to freezer malfunction.

[b]1 = Unselected Hereford; 2 = Selected Hereford; 3 = Angus x Hereford x Charolais; 4 = Angus x Hereford x Holstein.

[c]HS = high silage; HG = high grain.

[d]Weight of anterior pituitary.

[e]Anterior pituitary concentration of the 30 mg/ml extract.

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