THE RELATIONSHIP OF SOME SERUM HORMONES TO VARIOUS GROWTH AND CARCASS CHARACTERISTICS OF CATTLE

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ABSTRACT

THE RELATIONSHIP OF SOME SERUM HORMONES TO

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by John Steven Grigsby

The relationships of serum growth hormone (GH) and insulin to some growth and carcass characteristics were studied using 16 Hereford bulls, 17 Angus steers, 40 Holstein heifers, 19 Holstein steers and 13 Holstein bulls. The Hereford bulls were divided into two groups based on selection for either tenderness or leanness. Of the 40 Holstein heifers studied, 20 were fed 0.9 kg of grain daily and 20 were fed 4.5 kg of grain daily. Ten heifers from each nutritional level received 0.45 mg of MGA daily from 2.5 months of age. The Holstein steers were fed either 35% DM silage or 46% DM silage. Insulin and GH were quantitated by double antibody radioimmunoassays. Compositional data were obtained on wholesale rounds of Holstein heifers and 9-10-11 rib sections of Hereford bulls and Angus and Holstein steers. Tenderness was objectively determined using the Warner-Bratzler shear device and subjective measures of juiciness and overall acceptability were determined by taste panels.

Angus steers had significantly ($P \leq .05$) lower daily gains and Holstein steers had significantly higher ($P \leq .05$) daily gains during the bleeding period (ADGB) compared to the other breed and sex groups. Steers fed 35% DM corn silage had higher daily gains than those fed 46% DM silage. High nutrition as well as MGA treatment increased daily gains among Holstein heifers. Longissimus muscle areas (LMA) were not significantly different among the breed and sex groups but Angus steers had greater ($P \leq .05$) 12th rib fat thicknesses (FT-TH). Steaks from Holstein heifers were significantly ($P \leq .05$) more tender than steaks from the

other breed and sex groups. Taste panelists rated steaks of Angus steers more ($P \leq .05$) acceptable (OAA) than those of Holstein steers. Holstein steers had more lean and significantly more bone ($P \leq .05$) while Angus steers had significantly ($P \leq .05$) more separable fat than the other breed and sex groups. Among Hereford bulls, Angus steers and Holstein steers, FT-TH was negatively related ($r = -.71$, $P \leq .01$) to ADG and W-B shear values ($r = -.28$, $P \leq .05$) but positively related to OAA ($r = 0.36$, $P \leq .01$). High levels of grain increased the fat component ($P \leq .05$) and MGA treatment depressed ($P \leq .05$) bone and W-B shear values among the Holstein heifers.

Holstein steers had significantly ($P \leq .05$) higher average serum insulin (IN-A) while Holstein heifers had lower ($P \leq .05$) average serum GH concentrations (GH-A) than the other breed and sex groups. Bulls had lower ($P \leq .05$) GH-A levels than steers. Thirty five percent DM Pro-Sil (molasses, anhydrous ammonia and trace minerals) depressed serum GH compared to the 46% DM corn silage diet. Nutritional level among Holstein heifers had no influence upon plasma hormones but MGA treatment significantly increased IN-A. There were few significant correlations between either GH-A or IN-A and growth and carcass characteristics among individual breed and sex groups. In addition, hormone relationships with growth and carcass characteristics were generally inconsistent between breed and sex groups. However, when data of the individual groups were pooled, serum insulin was significantly and positively related to final weight ($r = 0.26$, $P \leq .01$), ADGB ($r = 0.37$, $P \leq .01$) and carcass weight ($r = 0.38$, $P \leq .01$). IN-A was not significantly related to any measures

of tenderness but generally was positively related to measures of lean and bone and negatively related to indices of fat. In contrast, GH-A was negatively related to daily gains ($r = -.32$, $P \leq .01$, total feeding period ADG; $r = -.15$, $P > .05$, ADGB) and Warner-Bratzler shear values ($r = -.69$, $P \leq .01$).

Standard errors of hormone values within breed and sex groups were high and the relationships with growth and carcass variables were often inconsistent between individual groups. However, serum hormones were significantly related to a number of economically important growth and carcass characteristics. Insulin, compared to GH, was more highly related to a greater number of growth and carcass characteristics of feedlot cattle.

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INTRODUCTION

The demand for red meat in the United States is rapidly increasing as evidenced by an approximate twenty two percent increase in per capita consumption of beef, pork, veal and lamb during the ten year period from 1961 to 1971. Although the efficiency of animal protein production is low compared to plant proteins, livestock producers have generally been able to meet the demand for their product. The ability of livestock producers to increase production efficiency while concomitantly increasing gross meat production is partially attributed to the use of various growth stimulants and antibiotics. Recently, both oral and implant administration of diethylstilbestrol (DES) have been prohibited. The ban on DES administration and the seemingly inevitable removal of other growth stimulants and antibiotics from livestock pharmaceutical markets may be attributed to their implication as possible human health hazards and pose a threat to present animal production efficiency. These facts indicate the tremendous need for animal scientists to understand basic physiological factors controlling growth, composition and meat quality. If such knowledge were available, it could possibly be used as a tool for selection or manipulation of growth and development criteria and; thus, may ultimately provide the basis of controlling carcass composition and meat animal production efficiency.

It has been recognized for many years that hormones affect the physiological functions of animals. More recently, hormones have been shown to

specifically affect or alter estrus cyclicity, sexual behavior, pubertal onset, growth, development, body composition and meat quality in animals. Therefore, it seems apparent that the endocrine system plays an extremely important role in meat animal production and that a thorough understanding of its functions may help to ultimately control or alter economically important factors in the production of muscle for food.

The recent development of sensitive assay methods to quantitate endogenous hormone concentrations has provided animal scientists with a tool to study the relationships of circulating hormones to parameters important in meat production.

This study was initiated to determine the relationships of endogenous growth hormone and insulin with some growth, composition and meat quality characteristics among cattle differing in sex, breed and nutritional treatment.

LITERATURE REVIEW

Growth and Composition

Growth rate and carcass composition are economically important factors which contribute to the efficiency of production and ultimate usefulness of meat animals. These two factors are interrelated as Hedrick (1968) noted that during growth and development an animal changes in form and composition. The rate and efficiency of growth as well as the factors affecting proportion of muscle, fat and bone in the carcass are current concerns of the animal scientist. His efforts to understand the mechanisms controlling these characteristics may allow him to ultimately use such information in manipulating body growth and/or composition.

Attempts to define growth have been made by many workers who generally agree that growth is more than an increase in body size or weight. Brody (1945) defined growth as the production of new biochemical units brought about by cell division, cell enlargement or incorporation of materials from the environment, while McMeekan (1959) defined growth as an increase in weight until a mature size is reached. McMeekan (1959) also defined development as changes in body shape and/or conformation until the body structure and its various parts reach maturity. The inclusion of fat deposition into the definition of growth has met with controversy. Maynard and Loosli (1962) suggested that growth be defined as an increase in structural tissues and should be distinguished from fat deposition. In contrast, Pomeroy (1955) maintained that the distinction between growth and fattening was arbitrary and he could find no logical reason for excluding fat deposition from the growth process.

The major tissues of the animal body grow at relatively different rates postnatally (Hedrick, 1968). Hammond (1933) reported a triphasic growth pattern with maximum growth occurring sequentially, first bone deposition, then muscle and finally fat. Furthermore, body proportions change during development with certain anatomical locations developing faster than others. Wallace (1948) reported a gradient of increasing growth intensity in sheep with the lower limbs having maximum growth early and with late maximum growth occurring in the loin region. Luitingh (1962) studied the influence of fattening, age and nutrition on developmental changes in beef steers and observed the shoulder to be the slowest growing followed by the round, loin, plate, neck, brisket and finally by the fat depots, flank, cod and kidney fat.

According to Palsson (1955) each tissue has a sigmoidal growth curve, but the inflection or point of maximal growth occurs at different ages for each tissue. He attributed the different rates of growth to nutrient priority by various tissues. The order of priority for nutrients of tissues from highest to lowest would be nervous tissue, bone, muscle and fat, respectively. Conforming to this hypothesis, early nutrient restrictions would have a greater effect on fat than nervous tissue. However, as nutrients become increasingly available, a greater proportion of the available nutrients would be utilized by low priority tissues. The second inflection or point of growth rate deceleration of each tissue occurs at different ages and as each matures their demand for nutrients decrease correspondingly. Therefore, the observation that fat deposition occurs last among the major animal tissues can be explained by Palsson's hypothesis

of nutrient priority.

Bone Growth and Development. McMeekan (1959) suggested that bone completes a major portion of its growth early in postnatal life. Other workers have determined that the skeleton is better developed at birth than either muscle or fat (Allen, 1966). However, not all bones mature at the same time. Palsson and Verges (1952) measured the effect of various nutritional levels on body component growth in lambs and they observed that sheep on a low level diet early in life and a high level later had the heaviest rib weights. In a reanalysis of the Palsson and Verges data, Fowler (1968) suggested that the increase in rib weight may be explained by the function of the rib as a rigid container for the thoracic and anterior abdominal organs rather than the ribs being late maturing bones. The low-high nutritionally treated sheep had heavier thoracic organs and foregut weights; and thus, it follows that these organs would require a larger container. This agrees with the theory proposed by Fowler (1965) that an animal tends to respond to environmental changes in such a way that the vital functional relationships between essential body components are preserved or modified to a form which gives the animal its best chance of survival and successful reproduction. Cuthbertson and Pomeroy (1962) reported similar data with pigs and showed that increased weight was associated with increased thickening and ossification of bone.

Weiss et al. (1971) reported that bone decreased from 32 to 13 percent as body weight increased from 1 to 137 kg in pigs, suggesting that bone and body growth were not proportional. Zinn (1967) reported that the

growth rate of bone decreased after 150 days on feed in both steers and heifers. Lambuth, Kemp and Glimp (1970) reported a higher percent bone among rapid gaining lambs (slaughtered at 36, 45 or 54 kg) as opposed to those which gained slowly.

Muscle Growth and Development. Muscle is the major body tissue by weight (Hedrick, 1968) and its development is intermediate to that of bone and fat. Everitt (1963) indicated that muscle mass was dependent primarily on number, length and cross sectional size of muscle fibers and the associated connective tissues and to a lesser degree on other muscle components. Growth of muscle tissue is characterized by three distinct phases: in the first phase hyperplasia predominates and is followed by a phase of concurrent hyperplasia and hypertrophy and the final phase consists predominantly of hypertrophy (Winick and Nobel, 1965). Robinson (1969) noted that all these developmental stages are dependent upon an adequate substrate supply and that since the musculature is one of the later developing tissues it may suffer as a result of substrate inadequacy during cellular hyperplasia. He conducted an experiment with pigs involving three nutritional regimes. These regimes included a normal diet throughout pregnancy and lactation, half of the normal diet throughout pregnancy but normal during lactation and the third level consisted of half the normal diet during both pregnancy and lactation. He reported that nutritional stress during pregnancy had the least effect on ultimate muscle development, although muscle hyperplasia ceased earlier than in control animals. Nutritional stress during both pregnancy and lactation markedly decreased hyperplasia. Winick and Nobel (1965) noted

that tissues are most susceptible to nutritional stress during active hyperplasia and that the effects of severe stress may be irreversible during this stage. The continuation of postnatal hyperplasia disagrees with McMeekan's (1940) suggestion that muscle fiber number is fixed at birth.

Most postnatal increase in muscle mass occurs by hypertrophy. Chrystall, Zobrisky and Bailey (1969) reported a 100 percent increase in muscle fiber diameter from birth to 25 days of age in pigs and only a 10 percent increase from 100 to 125 days. At 150 days, fiber diameter increase was 95 percent complete and after 150 days muscle growth was extremely slow. McMeekan (1940) observed that the coefficient of variation of the longissimus muscle fibers decreased from 44 percent at birth to 25 percent at 28 weeks of age, during which time fiber diameter increased eight fold. Chrystall et al. (1969) found that age and weight of pigs were significantly correlated ($P < .01$) with and accounted for 64 and 72 percent, respectively, of the variation in muscle fiber diameter. Furthermore, they reported correlation coefficients of 0.85 and 0.92 between longissimus muscle area and age and weight, respectively. In this same study protein content did not increase proportionally with the increase in muscle fiber diameter. Swatland and Cassens (1972) studied muscle enlargement in two lines of rats selected for rapid and slow postweaning weight gain and reported greater mean muscle fiber diameter among the rapid gaining rats than for those with slow gains. Brody (1945) noted that during rapid growth fiber diameter increased but the increment in fiber size became progressively less as growth slowed and mature size was attained.

Yeats (1964) observed a decrease in cross sectional area of bovine longissimus muscle following starvation and attributed it to muscle fiber atrophy and loss of intramuscular fat. He suggested that the connective tissues were relatively unaffected. Subsequent regain of live weight resulted in recovery of whole muscle dimensions and muscle fiber diameters. Thus, it would appear that inadequate nutrition irreversibly affects hyperplasia while the effect on hypertrophy is reversible.

Fat Growth and Development. According to Palsson (1955), fat has the lowest nutrient priority during early development and therefore, it is the latest of the three major tissues (bone, muscle and fat) to develop. During growth and development, fat mass increases by hyperplasia and hypertrophy. Kirsch (1969) reported that the early growth of fat depots in rats was accompanied by progressive enlargement of fat cells as well as by an increase in cell number. However, during late growth, the increase in fat mass was due entirely to hypertrophy. Hood (1972) showed that during early growth mass of adipose tissue increased by hypertrophy and hyperplasia in bovine and porcine animals. However, in pigs beyond 20 weeks of age, adipose tissue growth occurred exclusively by hypertrophy. He noted that in young animals, growth rate was positively correlated with adipocyte number but unrelated to fat cell number in more mature animals. By 14 months of age, hyperplasia was completed in all bovine fat depots except the late developing intramuscular adipose tissue.

Hood (1972) fasted 109 kg pigs to produce weight losses of 32 kg and reported a significant decrease in fat cell size among both lean and fat strains. In the lean pigs, cell number remained unchanged while in the

fat pigs, hyperplasia continued during the fasting period. He suggested that the fat strain had not yet attained maximum cell numbers prior to the onset of fast; and therefore, hyperplasia continued until the ultimate adult cell numbers had developed.

Hedrick (1968) indicated that rates of fat deposition in different parts of the body vary widely. The sequence of fat deposition showed that the initial deposit occurs around the viscera and kidney followed by intermuscular fat, subcutaneous fat and finally intramuscular fat.

Nutritional Effects. Nutrition contributes significantly to animal growth and body composition. During gestation, fetal nutrient supply is completely dependent upon the maternal supply of nutrients. Robinson (1969) restricted sows to low levels of nutrition during gestation which resulted in an early cessation of myohyperplasia in their offspring. He stated that nutritional stress imposed at birth caused permanent stunting in tissues in a manner directly proportional to their order of development and related to the degree of active hyperplasia proceeding at the time of stress. Ahlschwede and Robison (1971) studied the influence of pre- and postnatal environments on growth and backfat in pigs. Prenatal effects contributed approximately 17 percent of the variance in postweaning growth and backfat while postnatal effects only accounted for 11 percent of the variance. Robinson (1969) reported that growth generally, and in muscle specifically, was much slower in pigs from sows on restricted feeding during pregnancy and lactation compared to those from sows on normal diets during lactation. In addition, pigs subjected to nutritional stress during active hyperplasia reached the same body weight but had a greater proportion of fat than animals on normal diets.

Berg and Butterfield (1968) indicated that the major tissues of the bovine carcass show differential growth during development and that nutritional levels affected tissue growth. They noted that semistarvation depleted fat and decreased muscle deposition with only minor bone depletion. Realimentation restored the normal muscle and bone ratios and the proportion of fat was related to the plane of nutrition and length of the compensatory feeding period. Carr, Allen and Phar (1971) could not show any depletion of intramuscular fat in the longissimus muscle of fasted steers. Moulton, Trowbridge and Haigh (1922) observed greater percentages of muscle and higher muscle to bone ratios as the level of nutrition increased in steers. Kelly et al. (1968) reported increased fat percentages and decreased bone in cattle as the amount of grain in the diet was increased. Henrickson, Pope and Hendrickson (1965) observed that cattle on a high nutritional level during the last half of the total feedlot gain had 2.8 percent less muscle, 4 percent more fat and 0.8 percent less bone than those fed a moderate level. On a constant weight basis cattle on the moderate nutritional level during the last half of their feedlot gain required 65 days longer to reach slaughter weight. They concluded that the major differences in carcass composition resulted from the level of nutrition during the last half of the feedlot gain period. Similar results were reported by Waldman, Tyler and Brungardt (1971).

The type of diet has also been shown to affect composition. Garrigus et al. (1967) and Johnson et al. (1967) observed increased carcass fat when a corn concentrate diet was fed, especially in the later stages of the feeding period, as compared to hay or corn silage diets. Davey and Morgan

(1969) fed either a 12 percent or 20 percent protein diet to pigs selected for high or low backfat. On a 20 percent protein diet, the lean strain pigs had 36 percent more muscle and the fat strain had 11 percent more muscle than those fed 12 percent protein. In both lines of pigs, the 20 percent protein diet significantly increased average daily gain but had no effect on longissimus muscle area. During the feeding period of beef, (up to 341 kg) muscle weight increases rapidly while bone and fat deposition is slow. From 341 kg to 590 kg bone and muscle weights increase proportionally (Waldman, Tyler and Brungardt, 1971) but fat deposition accelerates between 227 kg and 341 kg. Zinn (1967), Stringer et al. (1968) and Hedrick, Thompson and Krause (1969) reported increased fat and decreased retail yields as the length of the feeding period increased. The longissimus muscle area increased but the increase was not proportional to carcass weight. In addition, rate of gain tended to decrease as time on feed increased (Stringer et al., 1968).

Anderson, Fausch and Gesler (1965) fed pigs on three nutritional regimes as follows: 1) ad libitum, 2) one--two hour period per day and 3) two--one hour feeding periods per day. Pigs allowed to eat twice daily for one hour had less backfat, lower ham weights and smaller longissimus muscles than ad libitum fed pigs of similar weights. However, pigs fed twice daily (two--one hour feedings) had more backfat than those fed only once daily. Ad libitum fed pigs consumed 27 percent more feed but gained 27 percent more weight than pigs allowed to eat only once daily for two hours.

Sex Effects. Sex, whether determined at conception or as a result of castration after birth, influences beef carcass composition (Hedrick, 1968). It is generally accepted that, at given weights, bulls have less fat than steers and steers have less fat than heifers. Hankins and Howe (1946) studied the composition of 84 steers and 36 heifers and reported 23.8 and 29.2 percent fat, 58.3 and 55.8 percent muscle and 18.0 and 18.1 percent bone for steer and heifer carcasses, respectively. Bailey, Probert and Bohman (1966), Pearson (1966), Hedrick (1968), Arthaud et al. (1969), Hedrick et al. (1969) and Field (1971) all reported higher percents of muscle or percents of retail cuts and lower percents of fat from bull than steer carcasses. Hedrick (1968) reported that steers have greater fat thickness and more total separable fat but less kidney fat than bulls. Hedrick et al. (1969) reported that bulls exceeded steers and heifers in rate of gain but that steers and heifers had similar gain rates. In addition, bulls had larger longissimus muscles and less fat than steers or heifers while steers and heifers were remarkedly similar. Steers had higher percents of retail cuts than heifers. Bailey et al. (1966) reported 31 and 40 percent fat, 52 and 44 percent muscle and 17 and 16 percent bone for bulls and steers, respectively. They also noted that bulls and steers were similar in preweaning growth rate but that bulls grew more rapidly and were more efficient in the feedlot than steers. On a weight constant basis, bulls yielded 13.2 kg more retail products than steers and on an age constant basis bulls yielded 26.8 kg more boneless retail cuts than steers (Arthaud et al., 1969).

Champagne et al. (1969) castrated bulls at birth, 2 months, 7 months and 9 months of age and they observed less fat over the longissimus muscle

of bulls compared to steers, except for those castrated at 9 months. Differences in fat among the 2 and 7 month castrates were not significant. The longissimus muscles of castrates were not significantly different from each other but tended to increase with increasing age at castration. Bull carcasses had significantly more longissimus muscle area and yielded more boneless retail cuts than any group of castrates.

Breed Effects. In general, most studies involving breed effects on carcass composition have compared beef and dairy cattle or crosses of various breeds. Carrol, Rollins and Ittner (1955) and Riggs and Maddox (1955) compared Hereford and Hereford X Brahman steers and reported that Herefords had less bone, more fat and less muscle than crossbred steer carcasses. Carpenter et al. (1961) reported a decrease in percent fat and an increase in percent muscle as the percent Brahman breeding increased in Brahman X Shorthorn steers. Cole et al. (1964) compared carcasses from Hereford and Angus, Brahman and Santa Gertrudis and Holstein and Jersey steers. British breeds yielded more fat and less separable muscle than Zebu or dairy breeds while percent separable bone was greatest in cattle of dairy breeding. Angus carcasses had the lowest percent separable muscle and bone but the highest percent separable fat while Holstein carcasses had the lowest percent fat and highest percent muscle and bone. Branaman et al. (1962) compared carcasses from Hereford and Holstein steers and found no significant differences in percent separable muscle or fat, but dairy steers had significantly higher percentages of bone. Hedrick (1968) reported that dairy breeds have higher percentages of kidney and pelvic fat but lower subcutaneous fat percents than beef breeds. Klosterman,

Cahill and Kunkle (1961) and Cahill, Klosterman and Ockerman (1962) reported higher percentages of trimmed retail cuts and lower fat percents in Charolais carcasses compared to Herefords.

Not only do breeds differ in composition, but strain differences within breeds are also apparent. For example, Davey and Morgan (1969) selected pigs for high and low fat and were able to produce pigs in the fat line with 9.1 percent less muscle, 11.7 percent more fat and 2.5 percent less bone than the lean line.

Age and Size Effects. The growth and development of various tissues reflects the effect of age on composition. In a review, Hedrick (1968) indicated that, generally, rapid muscle and bone growth at an early age was followed by an acceleration of fat deposition with a concomitant slowing of muscle and bone growth. In addition, he reported that growth rate was more rapid early in life and became slower as maturity was approached. However, he noted that most available data indicated that weight and stage of fattening had a greater effect on composition of the carcass than age per se. Weiss et al. (1971) slaughtered pigs at various live weights. In general, the percentages of muscle and bone decreased and separable fat increased significantly as slaughter weight increased. They also reported a significant weight-by-body section interaction which conformed to the growth pattern of rapid muscle deposition early in life followed by an increase in fat deposition with advancing maturity. They observed a progressive anterior to posterior pattern of muscle development. Hiner (1971) compared pigs at various weights and reported that the three major carcass components increased as weight increased. However, the

ratio of lean to fat decreased as live weight increased from 56.7 kg to 124.7 kg. Lambuth, Kemp and Glimp (1970) and Shelton and Carpenter (1972) compared lambs at several live weight intervals and found that percent boneless retail cuts and bone decreased while percent fat increased as weight increased. Hedrick et al. (1969) compared animals at 400 and 500 kg live weight and reported that extended feeding resulted in decreased retail cut and bone percentages and increased fat trim in bulls, steers and heifers. Waldman et al. (1971) found that the increases in fat and bone were small compared to muscle during early growth in steers. At approximately 227 kg, fat deposition accelerated and paralleled muscle deposition through 590 kg live weight. Allen et al. (1968) studied the effect of carcass weight on composition in eighty steer carcasses divided into two weight groups (227 to 250 kg and 318 to 340 kg). The light weight group yielded 51.7 percent muscle, 35.5 percent fat and 11.4 percent bone compared to 50.8, 37.0 and 11.2 percent muscle, fat and bone, respectively, in the heavy weight group. Carcass weight significantly ($P < .01$) affected the weight of separable muscle, fat and bone but the percentages of separable components were not significantly different between the two weight groups. The light weight carcasses yielded higher percentages of retail cuts than the heavy weight groups.

Assessment of Growth and Composition

Available methods of measuring growth and composition were reviewed by Hedrick (1968) and Pearson (1968). They indicated that only a relatively small percentage of actual variation in retail cuts could be accounted for

by subjective estimates on live animals and that precise objective measures are needed for selection of carcass traits. Pearson (1968) noted that, even though there is some relationship between certain linear measurements on the live animal and some carcass traits, these relationships are not high enough to be used for predicting carcass composition or other meaningful criteria. He also stated that weight per se is a more accurate predictor of composition and cutout than most linear body measurements.

It appears that the most accurate method of estimating total carcass composition involves the physical separation of fat, muscle and bone from the entire carcass; however, portions of the carcass have been used to compare differences in composition. The most widely used of these portions is the 9-10-11 rib section, followed by the wholesale round and wholesale flank. Lush (1926) and Hopper (1944) concluded that muscle, fat and bone of the wholesale rib adequately represented their respective components in the entire carcass. Hankins and Howe (1946) studied data from 84 steer and 36 heifer carcasses by comparing physical separation of fat, muscle and bone from the 9-10-11 rib section with that of the total carcass. They concluded that a high association existed between physically separable components of the 9-10-11 rib section and those of the entire carcass. Correlation coefficients between separable components of the rib section and the total carcass were 0.93, 0.85 and 0.83 for fat, muscle and bone, respectively, from the 120 carcasses. The correlation coefficient of muscle from the rib section and total carcass muscle was higher for steers (0.90) than heifers (0.72) which led Hopper (1944) to conclude that the usefulness of the 9-10-11 rib separable muscle content for estimating total

carcass muscle of heifers was questionable. The separable fat of the rib section was more highly correlated (0.93) with separable fat of the dressed carcass in steers compared to heifers (0.88) (Hankins and Howe, 1946). When ether extract and protein determination of the dressed carcass were compared to separable fat and muscle of the 9-10-11 rib section, correlations of 0.93 and 0.82, respectively, were obtained. Crown and Damon (1960) reported correlation coefficients of 0.98, 0.94 and 0.73 for separable fat, muscle and bone of the 9-10-11 rib section and corresponding components of the entire carcass. Likewise, Allen (1966) reported correlation coefficients of 0.92, 0.94 and 0.76 for muscle, fat and bone, respectively, from the 9-10-11 rib section and the entire carcass. He reported lower correlations in heavy cattle (700 to 750 lb) compared to light cattle (500 to 550 lb); therefore, Hedrick (1968) concluded that a need exists for prediction equations to be developed for well defined weight, sex and fat groups for more accurate results. Powell and Huffman (1968) compared five methods to estimate carcass composition and concluded that the Hankins and Howe method most accurately estimated carcass fat ($r = 0.94$) and carcass protein ($r = -.96$), however, it was the least practical method studied.

A number of workers have reported high correlations of separable components of the wholesale round with those of the entire carcass. Hedrick (1968) noted that since the round comprises a sizeable portion of the carcass, it should be a good indicator of total carcass composition. Allen (1966) reported correlation coefficients of 0.83, 0.91 and 0.83 between percent separable muscle, fat and bone, respectively, of the round

and percent separable components of the entire carcass. Cole, Orme and Kincaid (1960) noted that separable muscle in the round was associated with 90 percent of the variation in total separable muscle in the carcass. Hedrick (1968) concluded that research reports to date are rather consistent in the finding that trimmed round or boneless closely-trimmed retail cuts from the round are indicative of the retail yield of the entire carcass. He also noted that compositional data from the round would result in minimum economic losses and would provide adequate information for development of an equation to predict retail yield from beef carcasses. Palsson (1955), Luitingh (1962) and Butterfield (1965) have shown that the flank can be used to estimate carcass composition. Hankins and Howe (1946) reported a higher correlation between ether extractable flank fat ($r = 0.95$) than that of the 9-10-11 rib section ($r = 0.93$) and total carcass fat. They also reported a correlation coefficient of 0.89 between ether extractable flank fat and fat from the 9-10-11 rib cut. In addition, Allen (1966) observed correlation coefficients of 0.91, 0.91 and 0.32 between percent separable muscle, fat and bone, respectively, of the wholesale flank and separable components of the entire carcass. The low correlation observed for bone is expected since the flank contains only a small portion of the 13th rib (Purchas, 1969). Purchas (1969) indicated that the flank should be at least as effective as the round for estimating carcass fat but would probably not be as good an indicator of muscle or bone as the 9-10-11 rib section or wholesale round.

The cross sectional area of the longissimus muscle is often used as a measure of total carcass muscle. Cole, Ramsey and Epley (1962) reported

correlation coefficients of 0.58, 0.59, 0.39 and 0.63 between total separable muscle and the longissimus muscle area at the 5th rib, 12th rib, last lumbar vertebra and an average of these three area measurements, respectively. Hedrick et al. (1965) noted that longissimus muscle area accounted for 48 to 70 percent of the variation in weight of boneless retail cuts. Similarly, longissimus muscle area measurements accounted for 49 to 69 percent of the variation in boneless retail cuts of the entire side. In contrast to these observations, Epley et al. (1970) concluded that longissimus muscle area was of little value in estimating percent retail cuts of the four major wholesale cuts.

Fat is the most variable component of the beef carcass and since there is an almost proportional decrease in muscle as percent fat increases, many workers have studied the relationships of fat measurement to carcass composition (Hedrick, 1968). He indicated that fat thickness over the 12th rib was more highly associated with total carcass fat than any other anatomical fat measurement. Ramsey, Cole and Hobbs (1962) reported correlation coefficients of -.76, 0.83 and -.76 between fat thickness over the 12th rib and percent separable muscle, fat and bone, respectively. Nelms et al. (1970) reported that fat thickness over the 12th rib made a significant contribution to an equation for predicting retail cuts. Likewise, Powell and Huffman (1968) reported correlations of 0.89 with carcass fat and -.85 with carcass protein and they indicated that over 70 percent of the variation in carcass composition was accounted for by the fat measurement over the 12th rib.

Tenderness

Tenderness has been shown to be the most important quality factor contributing to consumer acceptability of meat (Brady, 1957; Rhodes et al., 1958; Means and King, 1959; Juillerat and Kelly, 1971); however, the range of tenderness acceptable to consumers is rather wide (Pearson, 1966). Tenderness is affected by pre- and postslaughter conditions. Before slaughter, anatomical and environmental factors combine to influence tenderness while after slaughter physiological factors and physical handling procedures are important contributors to meat tenderness. The relative importance of tenderness to the meat industry necessitates an indepth study of factors contributing to tenderness and an attempt to control or alter meat tenderness in all species of meat animals.

Nutritional Effects. Information regarding the effects of nutrition on tenderness is limited. In a review of work involving different nutritional regimens, Stringer (1970) noted that low protein diets for pigs were associated with increased marbling and tenderness. Purchas (1969) found nonsignificant differences in tenderness of steaks from Holstein heifers fed either high or low levels of nutrition although those fed high levels tended to have higher Warner-Bratzler shear values. Dube et al. (1971) reported that steaks from animals fed corn silage during the early part of the feeding period were more tender than those from hay fed animals. The differences in palatability due to the early feeding regimen were still evident at heavier weights and they noted that palatability was influenced less by the feeding regimen midway into the feeding period

than it was early in the feeding period. When animals of comparable age and of the same sex were slaughtered at 30 day intervals over a 270 day feeding period, Zinn et al. (1970) showed that the first 180 days on feed had a beneficial effect on tenderness and that shear values were lower at 150 and 180 days on feed than at all other periods. Animal age appeared to exert a greater influence after 180 days and an interaction between time on feed and animal age was apparent. Zinn et al. (1970) also reported that tenderness of all muscles was not affected equally by certain durations of feeding.

Tenderness may be dramatically affected by the nutritional state of the animal just prior to slaughter. Zessin et al. (1961) showed that pigs on a submaintenance diet had less intramuscular fat in the longissimus muscle than pigs on a fattening diet and that roasts and chops from fasted pigs were less tender than those from pigs on a fattening diet. Furthermore, Lewis, Brown and Heck (1965) concluded that limited feeding increased shear values. In a review, Hedrick (1965) referred to work involving sucrose feeding immediately prior to slaughter and noted that pigs on a high sucrose ration had pale, soft and watery muscles which were less tender than controls. In contrast, Mellor, Stringer and Mountney (1958) reported that feeding broilers sugar prior to slaughter increased the glycogen level of the muscle and improved tenderness of the pectoralis minor muscle.

Age Effects. Although most animals are marketed at a relatively early chronological age, there are some animals which, due to environmental or

genetic growth potential, are marketed at advanced ages. Dunsing (1959) reported that consumer panels consistently favored steaks from younger animals and in a review, Pearson (1966) concluded that the degree of maturity of an animal appears to have a definite influence on tenderness, although tenderness is not greatly altered within a narrow age range. Hiner and Hankins (1950), Tuma et al. (1962), Tuma et al. (1963), Goll et al. (1965), Breidenstein et al. (1968) and Webb, Kahlenberg and Naumann (1964) agreed that tenderness decreases with advancing maturity. On the other hand, Ritchey and Hostetler (1964), Romans et al. (1965) and Champagne et al. (1969) found no significant relationship between animal age and tenderness. In contrast, to both of these concepts, Alsmeyer et al. (1959) and Field, Nelms and Schoonover (1966) reported positive correlations between age and tenderness in some animals. Two possible explanations for these conflicting data may be that in some cases wide ranges in age are studied while in others only narrow ranges are tested or perhaps physiological age may be quite different from chronological age as suggested by Webb et al. (1964). Champagne et al. (1969) did not find any significant differences in Warner-Bratzler shear values of steaks from steers and bulls between birth and nine months of age. They also reported that differences in carcass characteristics attributable to castration age were, in most cases, nonsignificant. Likewise, Ritchey and Hostetler (1964) reported no clear cut evidence of age effects on tenderness in animals between 33 and 62 weeks old. Dissimilarly, Helser, Nelson and Lowe (1930) and Simone, Carroll and Chichester (1959) noted that cattle finished at 18 months of age were more tender than those finished at 30 months of age.

Reagan et al. (1971) obtained similar results when they compared bulls and steers at 385 and 484 days of age. Hiner and Hankins (1950) concluded that tenderness decreased with age in cattle between 2.5 and 66 months of age. Field et al. (1966) reported that when marbling was held constant, bulls 300 to 399 days old were significantly more tender than bulls 400 to 699 days old. However, they could find no significant tenderness differences among bulls after 400 days of age. In contrast, they found a significant positive correlation between age and tenderness in heifers and steers. Zinn et al. (1970) found a significant interaction between number of days on feed and animal age. They reported that cattle on feed 180 days were more tender than those fed for 270 days, at which time, age of the animal had a greater influence on tenderness than number of days on feed. Hunsley et al. (1971) compared shear values of cattle slaughtered at 6, 9, 12, 15 and 18 months of age and reported cattle at 6 and 18 months were more tender. Arthaud et al. (1970) reported similar results.

Sex Effects. Extensive data have been compiled on the influence of sex on tenderness. Such extensive work has been prompted by the well known facts that bulls grow faster than steers or heifers, are leaner, more efficient and have larger longissimus muscles and that steers are leaner than heifers at equal weights and are easier to manage than either bulls or heifers. However, most of the beef produced in the U.S. for the fresh meat trade is from steers and heifers because of the concensus that bull beef has inferior quality (Hedrick et al., 1969). Consumer acceptance ratings for loin steaks from young bulls have been lower than those for steers

(Field, Schoonover and Nelms, 1964), however, 90 and 88 percent of consumers who bought steaks and roasts, respectively, from bulls said they would buy them again. Shelton and Carpenter (1972) could not show any significant differences in shear values between rams, wethers and ewes slaughtered at weights ranging from 36 to 64 kilograms. Hedrick et al. (1969) showed no significant differences in Warner-Bratzler shear values of steaks from bulls less than 16 months of age and steers and heifers of comparable chronological age. However, shear values of steaks from more mature bulls were greater than those from steers or heifers at the same age. Warwick et al. (1970) also reported nonsignificant differences in Warner-Bratzler shear values when monozygotic male twins were paired and fed as a bull and a steer and slaughtered at an average weight of 408 kilograms. Champagne et al. (1969), likewise, reported no significant differences in tenderness ratings between steaks from bull and steer carcasses. In contrast, Arthaud et al. (1970) reported significant shear value differences between steaks from bulls and steers. Field et al. (1966), Hedrick et al. (1969), Hunsley et al. (1971) and Reagan et al. (1971) also reported significant differences between shear values of steaks from bulls and steers.

The data available indicate that differences in tenderness attributed to sex may actually be due to a sex x age interaction. Zinn et al. (1970) in a study involving both sex and age variables reported that muscles from heifers were more tender at 150 days on feed ($P < .05$) while steer muscles were more tender ($P < .05$) at 240 days. He suggested that the heifers may have matured at an earlier age reaching a peak tenderness about 30 days earlier than steers. That heifers mature at an earlier chronological age

was reported by Gramlich and Thalmann (1930) and Hankins (1932). Field et al. (1966) studied relationships of tenderness with sex and age in bulls, steers and heifers ranging in age from 300 to 699 days. They found no significant differences between bulls or steers and heifers between 300 to 399 days old. However, steers and heifers 400 to 499 days old had slightly higher palatability scores (more acceptable) than bulls of similar ages and shear scores indicated that bulls 500 to 599 and 600 to 699 days old were tougher ($P < .01$) than steers and heifers of comparable ages. Data presented by Hedrick et al. (1969) indicated that chronological age may have a greater adverse effect on tenderness of steaks from bulls than from steers or heifers. Although Field et al. (1966), Arthaud et al. (1969) and Reagan et al. (1971) concluded that variation in tenderness of steaks from bulls is considerably greater than steers and heifers, it appears that when animals are slaughtered at a reasonably young age there would be little consumer discrimination against any sex group in tenderness.

Breed Effects. Alsmeyer et al. (1958) working with Brahman, Shorthorn and various crosses reported that differences in tenderness due to breed of sire were significant. They showed that steaks from Shorthorn progeny were on the average more tender than steaks from progeny of Brahman and crossbred sires. However, they also reported that offspring from some Brahman bulls produced more tender meat than some of the British breeds. Kincaid (1962) showed that tenderness as measured by shear force decreased as the percent Brahman decreased. A very extensive comparison of tenderness among various breeds was conducted by Ramsey et al. (1963). Among

three types of cattle (British, Zebu and dairy breeding), loin steaks from dairy steers were more tender and Jersey steers showed the greatest tenderness. Loin steaks from British and dairy breeding were not significantly different in tenderness. Branaman et al. (1962) also reported no significant differences in tenderness between young beef and dairy type cattle. In addition, Ramsey et al. (1963) reported that steaks of steers from Brahman breeding were significantly less tender than steaks of steers from British or dairy breeding. In their studies, there were no significant tenderness differences in steaks from Hereford, Angus, Holstein or Jersey breeds. Pearson (1966) suggested that conformation or type has little to do with tenderness even though some differences have been reported between breed types.

Genetic Effects. Evidence that tenderness can be selected for is provided by Field et al. (1970). They made direct selection for either tenderness or leanness in two lines of Herefords for eleven years and found that significant differences in tenderness did exist between the two lines of Hereford bulls.

Stress Effects. Selye (1950) noted that animals exposed to a variety of stress factors reacted with an increased secretion of hormones from the adrenal gland which in turn affected the levels of muscle glycogen at the time of slaughter. Webb et al. (1959) and Webb, Kahlenberg and Naumann (1964) showed decreased tenderness in animals stressed with adrenalin injections prior to slaughter. Hedrick et al. (1959) observed that exogenous adrenalin injections reduced muscle glycogen in all animals studied. Low

levels of glycogen at slaughter limit the extent of lactic acid formation and hence results in increased postmortem muscle pH.

The high pH of postmortem muscle is responsible for "dark-cutting" meat. Hedrick et al. (1959) reported nonsignificant differences in tenderness between normal and dark-cutting beef. Loeffel (1942) reported that dark-cutting beef had lower shear values than normal beef and Lawrie (1962) indicated that the lowest tenderness scores for beef were obtained at pH 5.8 to 6.0 and the most tender meat was found at about pH 7.0. The condition of pale, soft and exudative (PSE) muscle is the result of the preslaughter stresses on susceptible pigs. In PSE muscle, lactic acid formation occurs very rapidly postexsanguination (Judge, 1969) thereby allowing muscle pH to decline while body temperature is still high. The rapid formation of lactic acid may be enhanced by the scalding procedure and high adiposity whereby the insulating effect of fat maintains near normal or slightly elevated temperatures. Carpenter (1961), Sayre et al. (1961), Lewis, Heck and Brown (1963) and Kauffman et al. (1964) reported normal muscle to be more tender than PSE muscle. In contrast, Judge et al. (1958, 1960) and Merkel (1971) showed tenderness to be greater in PSE muscle. Hedrick (1965) reviewed the available literature and concluded that when cattle and lambs were subjected to stress conditions just prior to slaughter they usually had lower muscle glycogen, higher postmortem muscle pH and improved tenderness while pigs subjected to certain stress conditions generally had lower postmortem muscle pH and were less tender than muscle from normal animals. More recently, however, Merkel (1971) presented contrasting results for pigs.

Anatomical Location. Not only does tenderness vary from animal to animal but it also varies between different muscles within an animal. Briskey and Kauffman (1971) reported that connective tissue content was greater toward the distal region of a limb and that tenderness was negatively associated with the quantity of connective tissue. Knutson et al. (1966) divided carcasses into the loin region, sirloin region and rear quarter region. Averages of muscles within these three groups showed decreasing tenderness in the order from loin region to rear quarter region, respectively. The most tender muscle measured was the longissimus and the toughest was the biceps femoris. Zinn et al. (1970) also reported a significant effect of muscle location on tenderness. In their study they attributed the low shear value of the triceps brachii and the high shear resistance of the longissimus to the variation in physiological maturity of the muscles, since it had been reported that the longissimus was more mature than the triceps brachii at comparable chronological ages.

State of Contraction. The state of muscle fiber contraction influences tenderness. Locker (1960), Herring, Cassens and Briskey (1965) and Herring et al. (1967a) have reported that muscles allowed to contract such that fiber diameter increased and sarcomere length decreased were less tender than stretched muscles with long sarcomeres and narrow fiber diameters. Herring et al. (1967a) reported fiber diameter to be linearly related to tenderness while sarcomere length was curvilinearly related to tenderness. In contrast, Covington et al. (1970), Field et al. (1970) and Hunsley et al. (1971) reported no significant relationship of fiber diameter and/or

sarcomere length with tenderness. However, Gothard et al. (1966) reported that the state of contraction after 7 days aging appeared to have a greater influence on tenderness than did state of contraction at time of maximum rigor mortis. They concluded that although contraction did not seem to be the factor most responsible for final tenderness, it appeared to have a significant influence.

Carcass Position. The recent information regarding the effects of contraction state on tenderness prompted several workers to study the relationship of carcass position before and during rigor mortis with tenderness. The normal procedure is for carcasses to be suspended by the Achilles tendon which, in general, tends to stretch muscles anterior to the femur but allows those posterior to the femur to shorten. Herring et al. (1965) hung right sides according to the normal procedure but left sides were placed horizontally, bone down, on a flat surface with the limbs fixed perpendicular to the long axis of the carcass. In the vertical position, sarcomere length and tenderness was greater in the psoas major, latissimus dorsi and rectus femoris. Muscles with greater tenderness and longer sarcomeres in the horizontally positioned carcasses were the longissimus, gluteus medius, adductor, biceps femoris and semitendinosus. Fiber diameter was highly related to shear ($r = .73$, $P < .01$). Hostetler et al. (1970) obtained similar results when carcasses were suspended from the obturator foramen. In addition to the contribution of connective tissue, carcass position appears to account for much of the remaining proportion of the variation found in tenderness.

State of Rigor. The phenomenon of rigor mortis is complete when the concentration of ATP has been depleted and glycogen is converted to lactate. Once rigor mortis has set in, muscles become relatively inextensible (Newbold and Harris, 1972) due to the formation of the actomyosin complex and concomitant shortening of muscle fibers. Marsh and Leet (1966 a, b) and Davey, Kuttel and Gilbert (1967) have shown that shortening to 20 percent of the excised (prerigor) length produced relatively small changes in tenderness, whereas further shortening from 20 to 40 percent produced a several fold increase in shear value. Herring et al. (1967a) reported that tenderness of contracted muscles did not reach acceptable levels even after 10 days of aging, but Buck, Stanley and Comssiong (1970) reported an increased tenderness among stretched rabbit muscles.

Aging. Although meat becomes tougher until rigor mortis is completed, an aging period at temperatures slightly above freezing results in subsequent increases in tenderness. Davey and Dickson (1970) reported that during the aging period the external loading required to stretch bovine sternomandibularis muscle to its fullest extent declines by 5 to 10 fold. They indicated that the loss of tensile strength was due to a weakening of the myofibrillar structures at the junction of the I filaments and the Z discs of the sarcomeres. Goll et al. (1972) suggested that resolution of rigor was due to 1) a modification of the actin-myosin interaction which results in changes in the nucleoside tri-phosphatase activities of actomyosin, changes in in vitro contractile properties of actomyosin, lengthening of the rigor shortened sarcomeres and changes in the dissociability of the actin-myosin complex; and 2) the loss of Z-disc integrity

resulting in fragmentation of myofibrils and corresponding decreases in tensile strength of the fibers.

Connective Tissue. Although the role of connective tissue in tenderness has been relegated to that of "background toughness", ultrastructural changes in connective tissue during postmortem aging have been suggested to affect tenderness. Goll et al. (1970) concluded that postmortem changes in connective tissue were probably due to changes in the number or strength of the cross bridges between connective tissue proteins. They suggested that increases in collagen solubility were possibly due to rupture or weakening of the cross linkages between collagen molecules. On the other hand, Bouton and Harris (1972) could not mechanically measure changes in connective tissue during aging and concluded that changes in connective tissue were unlikely to contribute to the increase in tenderness achieved during aging.

Protein Solubility. The solubility of meat proteins has been implicated as a factor contributing to meat tenderness. Goll et al. (1964 a) reported a decrease in connective tissue solubility with increasing age from 40 days to 10 years, 5 months among cattle. This decrease in solubility tended to parallel the decrease in tenderness attributed to age of the animal. Herring, Cassens and Briskey(1967b) and Kruggel and Field (1971) found greater collagen solubility in stretched muscle than in muscle allowed to contract. The stretched muscle was also more tender. Hegarty, Bratzler and Pearson (1963) found myofibrillar protein solubility to be positively correlated ($P < .01$) to tenderness. Davey and Gilbert

(1968) reported that approximately 52 percent of the myofibrillar proteins were extractable from unaged meat compared to 78 percent from aged meat. The aged meat was also more tender. In contrast, Dikeman and Tuma (1971) reported a negative relationship between protein solubility and tenderness and carcass maturity.

Cold and Thaw Shortening. Cold shortening occurs when prerigor muscle is exposed to temperatures approximately 0 to 15 C. The muscle fibers severely contract resulting in meat toughness. Locker and Hagyard (1963) reported cold shortening in beef to be minimal at about 15 to 20 C and it became progressively greater as prerigor temperatures deviated in either direction from 15 to 20 C.

Thaw rigor occurs when muscles are frozen in a prerigor state and are subsequently thawed over a short period of time. Upon thawing the muscles contract and become much tougher than muscles frozen after the completion of rigor mortis or after partial resolution of rigor (Marsh, Woodhams and Leet, 1968). The latter authors concluded that both cold shortening and thaw rigor are capable of producing toughness in meat.

Marbling. The U.S.D.A. quality grading system includes marbling as a factor in its grade determination. Marbling has long been believed to be associated with tenderness, but its relative contribution to tenderness is questionable. Some of the early workers (Hostetler, Foster and Hankins, 1936 and Ramsbottom, Strandine and Koonz, 1945) found no relationship between marbling and tenderness. Cover, Butler and Cartwright (1956) and Alsmeyer et al. (1959) indicated that marbling had only a

slight association with tenderness and Blumer (1963) reported that 1 to 36 percent of the variation in tenderness was attributable to marbling. Covington et al. (1970) reported that moderately marbled steaks were significantly more tender than steaks with small amounts of marbling. However, the data showing little or no relationship between marbling and tenderness predominate in the literature. Walter et al. (1965) reported no significant effect of marbling on tenderness over a wide range of maturity groups. Reagan et al. (1971) reported that at 385 days of age there was no significant difference in marbling between bulls and steers, but steers were more tender. At 484 days of age steers had more marbling than bulls but tenderness was not significantly different. Goll et al. (1965) and Moody, Jacobs and Kemp (1970) reported that steaks with fine textured marbling were more tender. The most recent work which conclusively shows a need to deemphasize marbling was reported by Parish (1972). Panel tenderness ratings were 5.2, 5.3 and 5.3 for steaks with slight, modest and moderately abundant marbling.

Assessing Tenderness

The Warner-Bratzler shear is one of the most widely used objective methods of evaluating meat tenderness (Banks, 1971). It was first developed by Warner in 1928 and modified and improved by Bratzler in 1932. Correlations between Warner-Bratzler shear force and panel sensory scores usually range between 0.60 and 0.85 (Banks, 1971). Bratzler and Smith (1963) and Banks (1971) reported that results from shear and sensory panels

were highly related when measuring tenderness of cooked meat. After reviewing many methods of objective measures of tenderness, Pearson (1963) concluded that the Warner-Bratzler shear affords one of the best relationships to sensory methods of measuring meat tenderness.

Hormones

According to the definition of Frieden and Lipner (1971), hormones are systemic-acting substances produced by specialized cells and released into the circulation to exert relatively specific effects either on all body cells or upon certain cells in specific organs. In adult animals, hormones are responsible for the integrated activity of organ systems and subsystems. They alter cellular functions in response to variation in the external environment, they induce sustained performance by cells and they change the level of activity of tissues and organs to maintain constancy of composition within the internal environment. Hormones maintain metabolic rates to meet the needs of the organism and are responsible for control of animal growth and differentiation.

Insulin is itself anabolic and is required for the action of other growth promoting hormones. Biological phenomena stimulated by insulin include transport of glucose, certain ions and amino acids, glycogen formation, glucose conversion to triglycerides, nucleic acid and protein synthesis (Krahl, 1972) and it strongly inhibits lipolysis. Growth hormone (GH) mobilizes nonesterified fatty acids from fat depots, increases blood glucose, inhibits muscle tissue utilization of glucose, increases protein

synthesis and decreases the sensitivity of tissues to insulin (Frieden and Lipner, 1971). Robinowitz and Zierler (1963) suggested that growth hormone and insulin act sequentially between periods of food intake to maintain an adequate supply of energy to tissues or to store excess energy in storage depots. Robinowitz and Zierler (1963) and Weil (1965) suggested that the major emphasis in the synergistic stimulation of protein synthesis by insulin and GH changes from insulin to GH as time after food intake increases.

Fat Metabolism (Insulin). Insulin is antilipolytic or inhibits fatty acid release from adipose tissues (Fain and Rosenberg, 1972). When insulin is absent, fatty acids are released from adipose tissues and marked ketosis is often seen in animals. However, insulin sufficient to inhibit fatty acid release and ketosis is much less than that required to affect blood glucose (Fain and Rosenberg, 1972). Most work relating the effect of insulin on fat cells has been through the capacity of insulin to stimulate the metabolism of glucose (Crofford et al., 1972). Crofford and Renold (1965 a, b) noted that insulin stimulated glucose metabolism by accelerating the carrier mediated transport of glucose into the cell. However, the antilipolytic action is not dependent on insulin's glucose transport action even though glucose metabolism has an antilipolytic action of its own. In vitro studies with isolated fat cells show that lipolysis is inhibited by insulin in both the presence and absence of glucose. In the presence of growth hormone and glucocorticoids, fatty acid release is inhibited by only one-tenth the amount of insulin required to stimulate glucose metabolism by fat cells (Fain, Kovacev and Scow, 1965; Fain, Kovacev and Scow,

1966). In addition, Murthy and Steiner (1972) reported increased lipogenesis in brown adipose tissue through an effect independent of any action on glucose transport or metabolism. He observed that in an in vitro glucose free system, insulin reversed the inhibition of lipogenesis.

The mechanism of the antilipolytic action of insulin has not been positively determined. However, Murthy and Steiner (1972) suggested that insulin may promote lipogenesis by lowering adipocyte cyclic AMP levels. They based their hypothesis on work showing a 50 percent inhibition of acetate incorporation into fatty acids when adenosine 3', 5' cyclic AMP was added and on evidence presented by Sutherland and Robinson (1969) who showed that insulin reduced cyclic AMP levels in fat cells. In contrast, Fain and Rosenberg (1972) incubated fat cells with insulin for fifteen minutes and 2.5 hours, respectively, but found no significant effect on adenyl cyclase activity of fat cell ghosts. In addition, when they added insulin to an incubation medium containing cyclic AMP, there was no reduction in lipolysis. They concluded that the antilipolytic action of insulin may be unrelated to the effects of insulin on cyclic AMP accumulation.

Fat Metabolism (Growth Hormone). In vivo, growth hormone promotes ketosis, decreases fat stores, promotes a transfer of fat from adipose tissue to the liver and increases plasma free fatty acids (Raben and Hollenberg, 1959). Weil (1965) suggested that growth hormone increased catabolism of triglycerides resulting in the production of free fatty acids. Goodman (1965) studied the effect of growth hormone administration on adipose tissues in vitro. Using hypophysectomized rats, he administered fifty micrograms of GH

and doubled the U - ^{14}C glucose uptake and incorporation into fatty acids. By one hour after injection, the GH effect was reduced and by 3.5 hours opposite effects were observed. Likewise, Bassett and Wallace (1966) reported an "insulin-like" phase with declining glucose, ketones and free fatty acids lasting for one hour after GH injection in intact sheep which was followed by a rapid increase in plasma free fatty acids to a maximum eight hours postinjection. A similar effect was observed in hypophysectomized sheep with one half the GH dose given intact animals. In a similar study with rats, Goodman (1968) reported an early "insulin-like" action of GH followed by "anti-insulin-like" actions with reduced glucose utilization and increased adipose tissue lipolysis after 3 hours. Similar results have been reported in dogs by Rathgeb et al. (1970) and in sheep by Davis, Garrigus and Hinds (1970). In contrast, Wheatley, Wallace and Bassett (1966) reported a slight increase in plasma glucose but no alteration in the concentration of plasma free fatty acids or ketone bodies with 5 mg GH injected daily for four weeks. Machlin (1972) stimulated lipolysis in pigs (in vivo) and in rat adipose tissues (in vitro) with a commercially prepared GH. However, when the GH was further purified, tibia activity more than doubled but in vitro lipolytic activity was no longer detected. He suggested that lipolytic activity is not an intrinsic part of the porcine growth hormone molecule and is not necessary for tibia growth.

Protein Metabolism (Insulin). Insulin enhances the transport of some amino acids into muscle cells and enhances the incorporation of amino acids into proteins (Manchester, 1972). In addition, Manchester and Krahl (1959)

noted that insulin enhances the incorporation of intracellular synthesized amino acids into protein and suggested that insulin's effect on protein synthesis is independent of amino acid transport into the tissues. Goldstein and Reddy (1970) studied transport of amino acids into cells and their incorporation into protein in order to determine the step in protein synthesis in which insulin is effective. Amino acid transport into muscle is sodium dependent. When they incubated muscle in a sodium free system containing insulin and adequate amino acid pools there was no incorporation into the protein. They suggested that the incorporation of labeled amino acids is a result of insulin's effect on active amino acid transport.

Krahl (1972) has suggested a mechanism of insulin action in which ions act as second messengers in the initiation of cellular protein synthesis. When Mg^{++} and Ca^{++} were omitted from an <u>in vitro</u> incubation medium insulin caused no stimulation of protein synthesis. When Mg^{++} was added, baseline protein synthesis increased in adipose tissue cells if insulin was present. Insulin increased intracellular Mg^{++} and K^{+} through the Mg^{++} - activated $(Na^{+} + K^{+})$ - ATPase enzyme system. The ATPase system plays a role in the insulin stimulated ion translocation. Based on the above information, Krahl (1972) has hypothesized that insulin is bound to the plasma membrane to initiate the Mg^{++} - activated $(Na^{+} + K^{+})$ - ATPase enzymes which results in increased intracellular K^{+} and Mg^{++}. These ions, which may be located near the inner surface of rough endoplasmic reticulum, may then act as second messengers to influence intracellular enzyme activities and protein synthesis.

Manchester (1972) has reviewed the effects of insulin on protein synthesis. He noted three factors affecting tissue protein synthesis: 1) total ribosomes present, 2) the proportion of ribosomes to polysomes and 3) regulation of the rate of movement of ribosomes along the messenger. Tissues having low rates of protein synthesis characteristically have a low polysome to ribosome ratio. When insulin is administered this ratio increases even when new RNA is not being synthesized, suggesting that insulin promotes initiation or the process of attaching ribosomes to m-RNA.

Protein Metabolism (Growth Hormone). Growth hormone increases the nitrogen content in the carcasses and pelts of hypophysectomized rats (Scow, 1959). All nitrogen fractions in the thigh muscle except alkali-soluble stroma were increased by daily GH injections (0.1 mg). When the dose was increased to 0.5 mg per day, the gains in collagen and stroma fractions were markedly increased. There was a smaller ratio of myosin to collagen in rats receiving the larger dose.

Growth hormone affects protein synthesis by increasing the transport of amino acids into tissues (Jefferson and Korner, 1967), increasing the incorporation of amino acids into protein in vitro (Kostyo, 1964), increasing RNA synthesis (Garren., Richardson and Crocco, 1967) and by initiation of peptide chain elongation (Kostyo and Rillema, 1971). Frieden and Lipner (1971) noted that recent studies on GH action point to a role in the transcription and translation steps. The latter authors also state that RNA polymerase increased within 24 hours after GH treatment. In addition, both transfer and m-RNA formation increased. However, Korner (1967)

has shown that actinomycin inhibits RNA synthesis but not protein synthesis suggesting that the effect of GH is not due to RNA synthesis. He also suggested that GH may influence some factor necessary for ribosomal function and that RNA synthesis may be a secondary effect.

Insulin and its Control. Insulin is produced by the β-cells of the pancreas through an RNA - directed mechanism involving the synthesis of a single chain polypeptide precursor (proinsulin) which is then converted to a two chain molecule by enzymes supposedly located in the Golgi apparatus (Turner and Bagnara, 1971). Crystallization of insulin was accomplished by Abel (1926). Sanger, Thompson and Kitai (1955) determined the complete amino acid sequence of insulin from several species and sheep insulin was synthesized in 1963 by Katsoyannis (1964). The bovine insulin molecule is composed of a total of 51 amino acids with 21 in the A chain and 30 in the B chain (Turner and Bagnara, 1971). The two chains are linked by disulfide bonds at positions 7 and 20 in the A chain and at 7 and 19 in the B chain. In addition, cysteine residues at positions 6 and 11 of the A chain are linked by a disulfide bridge. The three disulfide bonds are essential for molecular stability of the molecule.

Insulin has been found in the circulation of fetal humans (Milner, Ashworth and Barson, 1972), fetal sheep (Bassett and Thornburn, 1971) and fetal calves (Grigsby and Oxender, 1972) after the first trimester of pregnancy. However, the mechanism of fetal insulin release is not well understood. Davis et al, (1971) have shown an increase in fetal insulin in response to exogenous glucose and fructose but noted that glucose was

a more potent stimulant of fetal insulin release than fructose. Colwill et al. (1970) have shown that exogenous insulin injected into fetal circulation increased fetal glucose utilization and suggested that insulin is released in hyperglycemic fetuses. The ratio of maternal to fetal insulin decreased with length of gestation in rabbits (Adam et al., 1969) and cattle (Grigsby and Oxender, 1972) suggesting increased release of insulin by the developing fetal pancreas throughout gestation. Milner, Ashworth and Barson (1972) showed that plasma insulin could be increased with leucine in human fetuses greater than 200 grams while arginine was only effective in raising plasma insulin in fetuses less than 200 grams. They suggested that the development of different mechanisms for insulin release occurs during gestation.

Burr et al. (1971) have suggested a biphasic release of insulin from fetal rat pancreas in vitro. They incubated pancreatic tissue in a glucose medium and showed an early (primary) immunoreactive insulin (IRI) release followed by a smaller late (secondary) release of IRI. When pyruvate was used in place of glucose, there was no release of IRI.

Manns et al. (1967) induced an increase in plasma insulin by infusing propionate and butyrate and demonstrated that the plasma insulin increase after infusion of these volatile fatty acids (VFA) was greater than after glucose infusion in adult sheep. This finding seems reasonable since VFA are the most important source of energy in mature ruminants. Horino et al. (1968) also demonstrated an increase in insulin secretion in ruminants by several of the short chain fatty acids produced in the rumen but no such effect of VFA was observed on insulin secretion in nonruminant species.

Trenkle (1970a) studied the effects of short chain fatty acids, feeding, fasting and type of diet on plasma insulin levels. Since the proportion of propionate and butyrate increases in the rumen when readily fermentable carbohydrates are fed, he speculated that diet played a direct role on insulin secretion. After a 30 hour fast, he infused acetate, propionate, butyrate, glucose and saline into sheep. By 15 minutes postinfusion, serum insulin was greatest in sheep given propionate and butyrate with no response observed in saline infused sheep. However, the response was more prolonged in sheep infused with glucose, such that by 2 hours after infusion, insulin had fallen to fasting levels in all but the glucose infused sheep.

Grigsby _et al_. (1972) fasted pigs for 24 hours then fed 500 g of a 16 percent protein grower diet. Within one hour after feeding, serum insulin had increased 17 fold, glucose rose 50 percent but free fatty acids (FFA) and glucocorticoids were reduced 50 percent. Likewise, Trenkle (1970a) fed fasted sheep and within 4 hours the average plasma insulin rose 23 percent. However, a subsequent 72 hour fast decreased plasma insulin to 32 percent of the concentration at four hours postfeeding.

The sudden increase in insulin after feeding monogastric animals has been attributed in some cases to the release of gastrointestinal hormones. According to Lernmark, Hellman and Coore (1968), considerable evidence is available to show that secretin, pancreaozymin and cholescystokinin stimulate insulin release. They determined the effects of exogenous gastrin on insulin release from mouse pancreatic tissue _in vitro_. In the presence of low levels of gastrin, insulin release was inhibited but when large

amounts of gastrin were applied to the incubation medium, insulin was released.

Young (1963) has suggested that glucagon stimulates insulin secretion. Glucagon acts by increasing blood glucose concentration which in turn promotes insulin release. Another hypothesis for the glucagon effect on insulin is that the close proximity of the α-cells to β-cells in the pancreas may have a physiological influence on insulin secretion (Lernmark, Hellman and Coore, 1969). They have also reviewed evidence of gastrin secretion by pancreatic α-cells and suggested that the gastrin may influence the function of the β-cells so as to increase insulin secretion.

Bassett and Wallace (1967) gave adult sheep 75 mg of cortisol daily for 14 days and observed elevations in plasma glucose and insulin. However, when they increased the daily dose to 150 mg for another 14 days, plasma glucose and insulin were not significantly affected, although insulin tended to decrease during the second week of each period. They concluded that an intact sheep can maintain insulin secretion at a high rate for at least two weeks but that the sheep is unable to maintain high insulin secretion rates for prolonged periods of time. Bassett and Wallace (1967) also observed a continued hyperglycemia even when insulin concentrations were high and they suggested that glucocorticoids are antagonistic to the action of insulin on carbohydrate metabolism.

Epinephrine and norepinephrine inhibit insulin secretion, though both hormones elevate blood glucose (Turner and Bagnara, 1971). The latter authors speculated that this action may be mediated through the α-cells and β-adrenergic receptors since insulin is increased when α-receptors are

blocked by certain drugs or when the β-receptors are stimulated. However, Creyer, Herman and Sode (1971) could not increase insulin concentrations in baboons by blocking the α-adrenergic receptors.

Bassett and Wallace (1966) rapidly infused either 8 or 10 mg of ovine GH into intact sheep and reported an "insulin-like" phase lasting up to one hour. During this time, plasma glucose, FFA and ketone concentrations declined. However, FFA increased rapidly at 8 hours while glucose and ketones increased gradually. By 24 hours plasma glucose, FFA and ketones returned to normal. Bassett and Wallace (1966) also injected 0.2 mg of ovine GH per kg of body weight per day into sheep for 4 weeks. They demonstrated a marked increase in plasma glucose and insulin concentrations. In addition, the response was biphasic and was positively correlated with increased nitrogen retention. In contrast, Manns and Boda (1965) were unable to demonstrate an increase in plasma glucose or insulin when 1 mg per kg body weight of GH was injected into sheep even though FFA were elevated and plasma amino nitrogen decreased. Likewise, Head et al. (1970) reported low plasma insulin concentrations in GH treated dairy calves as well as a failure of GH to elevate plasma glucose.

Growth Hormone Control. Bayliss et al. (1968) have suggested three basic factors affecting GH secretion: 1) stress, 2) decreased energy supply and 3) increased amino nitrogen pool. In addition, Kokka (1972) noted that most current evidence suggests a central nervous system component regulating GH secretion. Müller and Pecile (1966) fasted rats for 18 hours and found that pituitary GH concentrations were reduced. This ob-

servation agrees with most work involving monogastric animals which shows decrease pituitary GH content but increased plasma GH concentration during fasting. When GH was administered to fasted rats, pituitary GH, plasma glucose and FFA increased. Insulin given to fasted rats markedly reduced plasma glucose, FFA and pituitary GH. Müller and Pecile (1966) concluded that inadequacy of available carbohydrates and the need for sources of energy other than carbohydrates, namely non-esterified fatty acids, rather than absolute hypoglycemia, seem to be the physiological state leading to GH secretion. They also suggested an auto feed-back mechanism based on the ability of exogenous GH to inhibit endogenous GH release.

Hertelendy et al. (1970) infused L-Arginine into sheep and cows which resulted in prompt and marked plasma GH increases. Plasma GH was not affected by L-Arginine in pigs. Cyclic AMP and theophylline administration in sheep nearly quadrupled plasma GH concentrations (Hertelendy, 1971). When theophylline was administered alone to rat pituitaries in vitro, pituitary cyclic AMP and GH in the medium increased (Hertelendy et al., 1971). Epinephrine exhibited an inhibitory tendency on theophylline and cyclic AMP stimulated GH secretion.

Schally et al. (1968) suggested that a hypothalamic releasing hormone which travels down the hypophyseal portal system exerted a control on GH release. This factor or hormone has been isolated and purified by Schally and Arimura (1971) and has been shown to decrease pituitary GH and increase plasma or incubation medium GH both in vivo and in vitro, respectively. The hypothalamic GH releasing hormone not only induces GH release from the pituitary but also initiates de novo GH synthesis by somatotrophs. The

centers for hypothalamic GH control have been found to be localized in the ventromedial and arcuate nuclei and in the median eminence in rats. Stimulation of these areas by electrical shock increased plasma GH in 80 to 90 percent of the rats studied (Frohman et al., 1971). If lesions are electrically induced in these nuclei, GH release is inhibited.

Relationship of Growth to Hormones. Baird, Nalbandov and Norton (1952) measured pituitary GH in two lines (rapid and slow gaining) of pigs. They noted that rapid gaining pigs consistently had more GH per unit of anterior pituitary tissue than the slow gaining line. Nalbandov (1963) suggested that vigorous growth can occur only as long as the ratio of circulating GH per unit of body tissue is high enough to stimulate bone and muscle growth.

Siers and Hazel (1970) bled pigs at 15, 45 and 90 kg live weight. During this time, serum GH decreased from 5.4 to 2.8 ng per milliliter. They stated that GH level declined with age and was negatively correlated with growth rate, longissimus muscle area, carcass length and percent ham and loin. They also suggested that a negative relationship may exist between hormone utilization rate and plasma GH concentration. These hypotheses have been contested by Trenkle and Irwin (1970). The latter authors did not find any significant differences in plasma GH concentration between cattle 18, 198 or 393 days of age. This suggests that growth stasis due to dilution does not seem to be warranted.

Siers and Swiger (1971) studied the interaction of age and weight on plasma GH in pigs. They reported negative correlations between average daily gain and weight of lean cuts per day of age. They noted that pigs

differing in age but not size had similar plasma GH concentrations but that pigs of increasing age and size had a lower plasma GH concentration. They concluded that size and not age was the factor responsible for the decreased plasma GH concentration.

Turman and Andrews (1955) injected pigs with GH and although GH did not increase rate of gain, carcass fat content was reduced and nitrogen retention was increased leading them to conclude that true growth was stimulated. Lind et al. (1968) injected porcine GH (PGH) and measured several growth parameters. PGH increased muscle growth in the semitendinosus muscle as evidenced by increased fiber diameter. PGH appeared to have a depressing effect on long bone length and circumference; however, rate of gain was not significantly affected. Likewise, Wheatley, Wallace and Bassett (1966) injected 5 mg ovine GH per day for 4 weeks into adult sheep and although no measurable change in body weight occurred, nitrogen retention and wool growth was increased.

Trenkle (1970b) measured plasma insulin and GH in finishing cattle. Cattle fed stilbestrol had higher insulin GH levels and higher average daily gains than controls. Plasma insulin increased during the feeding period but presumably as a result of increased concentrates in the ration. Although this study (Trenkle,1970b) was limited to the finishing stage of growth, plasma insulin tended to be positively related to gain while GH was negatively related to gain.

Curl et al. (1968) observed higher pituitary GH concentration in bull calves (29 to 37 kg) than feedlot steers (308 to 378 kg). Cattle with high GH per unit of body weight had carcasses with higher specific gravities

(less fat). They also had greater daily gains and higher percentages of body protein. Body weight was highly correlated with pituitary, adrenal and pancreatic gland weights. Macmillan and Hafs (1968) also found a close relationship between body weight and anterior pituitary weight. Both body weight and anterior pituitary (AP) weight increased linearly from birth to one year of age. The only deviation from linearity was a decrease in AP weight at 6 months. However, this was not associated with any marked changes in body growth even though it represented the onset of puberty. They suggested an increased sensitivity of the pituitary to increasing androgen titers.

Dev and Lasley (1969) did not find any differences in serum GH between dwarfs, dwarf gene carriers or normal cattle. They suggested a failure of the target cells and organs to respond to GH rather than insufficient GH release to be the cause of dwarfism. No significant correlation of GH with growth rate was observed among the cattle in these studies.

Trenkle (1970b) observed a close relationship between plasma insulin in finishing cattle to consumption of grain. As the ratio of grain to hay increased, plasma insulin increased. This resulted in a trend for insulin to be positively related to daily gain.

Acetate has been suggested to be a chemostatic regulator of ruminant feed intake. Therefore if insulin decreases plasma acetate, then its effect on daily gain may be a result of increased feed consumption. However, when Muller and Colenbrander (1970) injected insulin into sheep they were able to decrease blood acetate but feed intake was unaffected.

Macmillan and Hafs (1968) have suggested that heifers have heavier anterior pituitaries than bulls and attributed this to a possible androgen sensitivity of the anterior pituitary. Trenkle and Irwin (1970) compared steers and heifers and reported no significant differences in plasma GH or insulin at 18 or 198 days of age but at 393 days males had higher plasma insulin concentrations. This was not attributed to sex but to the higher grain ration fed to steers compared to the roughage ration received by heifers. Likewise, Grigsby and Oxender (1972) did not observe any sex differences in Holstein fetal insulin.

Relationship of Carcass Quality to Hormones. Purchas et al. (1971a) reported lower Warner-Bratzler shear values for heifers fed melengestrol acetate (MGA). Heifers fed MGA also had lower plasma GH before slaughter which was significantly and negatively related to growth but not to any measures of carcass composition. Glucocorticoids were negatively related to growth and tenderness. Although Hafs, Purchas and Pearson (1971) have suggested that insulin may have an important effect on carcass quality, there have been no reports of such a relationship to date.

Relationship of Body Composition to Hormones. Data relating hormones to certain growth parameters in meat animals (i.e. body weight, average daily gain, etc.) are found in the literature but very few studies have involved the relationship of hormones to carcass composition. In addition, little work has been reported relating endogenous hormones to either growth or composition primarily due to the inadequacy of assays in determining low physiological concentrations. One of the earlier reports involving

meat animals was that by Turman and Andrews (1955). They injected GH at five different levels (2.25, 3, 4.5, 5 and 10 mg per 15 kg body weight per day and saline into controls) into barrows weighing 45.4 to 52.2 kg. Differences in carcass characteristics among the hormone treated pigs were small but differences between treated and nontreated pigs were great. Average backfat thickness of controls was 4.5 cm compared to 3.6 cm for GH injected pigs. Chemical composition of controls was 10.8, 39.4 and 49.3 percent protein, moisture and fat, respectively, while the corresponding components in GH treated pigs were 13.5, 49.1 and 36.8 percent, respectively. In addition, carcass length was significantly greater in GH treated animals.

Lind et al. (1968) injected 3 and 6 mg of porcine GH (PGH) per 15 kg body weight per day or guinea pig antiporcine GH (APGH) into Duroc barrows. Pigs given PGH had larger longissimus muscle areas and less backfat than controls and anti-PGH treated pigs. There was no difference in separable fat, muscle or bone of the left ham between treatments but pigs given 3 mg of PGH per 15 kg body weight per day tended to be most muscular. The GH treated pigs had higher percentages of lean cuts than the other groups. Likewise, Machlin (1972) reported increased growth rate and muscle mass and decreased fat thickness and percent fat of the ham among GH treated pigs.

Purchas et al. (1971b) fed heifers MGA beginning at 2.5 months of age or at first estrus. Heifers fed MGA had reduced jugular GH levels. Heifers fed MGA from 2.5 months of age had less bone growth relative to total carcass weight and as a result had a higher muscle to bone ratio as well as

lower percentages of fat. Therefore, high endogenous plasma GH was generally associated with greater bone growth, lower percentages of muscle and higher percentages of fat in the round.

Trenkle and Irwin (1970) studied plasma GH and insulin relationships to growth and carcass characteristics in suckling, weanling and yearling cattle. Plasma GH concentrations were 18 and 13 ng per ml in sucklings, 16.5 and 14.3 ng per ml in weanlings and 13.5 and 10.8 ng per ml in yearlings for males and females, respectively. Plasma insulin concentrations were 20.3 and 24.3 μ U per ml, 21.1 μ U per ml and 45.2 and 18.3 μ U per ml for male and female sucklings, weanlings and yearlings, respectively. The only significant correlation between GH and longissimus muscle area (0.514) was found in yearlings. GH was negatively related to fat thickness in sucklings. Insulin was not significantly related to longissimus muscle area or fat thickness at any age. Trenkle and Irwin (1970) gave three possible explanations for the low correlations between plasma GH and insulin with carcass characteristics: 1) hormones may not be limiting factors in these animals, 2) high variations between animals, and 3) plasma level and secretion rate may not be closely related. However, Trenkle (1971a) has reported a correlation of 0.97 between GH secretion and plasma GH concentration. Of the limited data available relating plasma insulin or GH to composition in meat animals, most suggest very low relationships.

Melengestrol Acetate. Melengestrol acetate (MGA) has been shown to consistently increase feedlot gain in heifers. Bloss et al. (1966) suggested that MGA might exert its effect on growth by allowing continued

endogenous estrogen secretion since MGA had no effect on spayed heifers. Purchas et al. (1971a) observed a 10 percent increase in daily gain with MGA while the mean jugular GH was lower among treated heifers compared to controls. In addition, MGA tended to improve tenderness and percent muscle of the round. MGA increased plasma insulin concentration in feedlot heifers which tended to be positively related to daily gain (Trenkle, 1970b). Kalkhoff, Jacobson and Lemper (1970) have suggested that progesterone evokes an enhanced plasma insulin response to insulinogenic stimuli which may explicate the increased gain effect of MGA.

Diethylstilbestrol. Diethylstilbestrol (DES) increases growth rate in steers provided adequate carbohydrate sources are available (Clegg and Cole (1954). Hafs et al. (1971) noted that in almost all cases both cattle and sheep have responded to DES with increased daily gains and increased pituitary weights. Trenkle (1970b) observed increased daily gain, pituitary weight and elevated plasma GH in steers and increased plasma insulin and GH in heifers receiving DES. Hafs et al. (1971) suggested that increased growth response to DES is probably a function of insulin secretion.

Bidner et al. (1972) observed that DES plus methyltestosterone (MT) increased gain efficiency and pituitary weights in barrows and gilts and daily gain in barrows. The hormones also decreased carcass fat and increased muscling. The hormone treatment did not affect taste panel evaluation of roasted loins. These results were observed among pigs studied on a weight constant basis. The effect of DES + MT upon muscle and fat were not evident among pigs when studied on a constant age basis.

MATERIALS AND METHODS

This study was not designed to be a controlled experiment with regard to breed, sex, age, size, ration or housing. In fact, the cattle used in this study were included in order to maximize variability between groups as well as their availability at Michigan State University.

Experimental Animals

Sixteen Hereford bulls, 17 Angus steers, 40 Holstein heifers, 19 Holstein steers and 13 Holstein bulls were used (table 1) in this study. All cattle were fed twice daily, at 7 am and 3 pm and were on experiment either in 1967 to 1968 or 1970 to 1971. All animals were weighed monthly and again just prior to slaughter.

TABLE 1. EXPERIMENTAL ANIMALS.

Breed	Sex	Number of animals	Group number
Hereford	Bulls	16	1
Angus	Steers	17	2
Holstein	Heifers	40	3
Holstein	Steers	19	4
Holstein	Bulls	13	5

Hereford Bulls. These bulls were selected from an experiment conducted at MSU in which selection of two separate lines based on tenderness

or leanness was made over a period of 12 years. They were housed communally in an open-fronted shed (MSU Beef Cattle Research Center) and were fed 2.7 kg of corn silage, 3.9 kg of corn and 0.45 kg of a 64% protein supplement (85% dry matter basis) per day. These bulls were slaughtered at approximately 14 to 16 months of age.

Angus Steers. These steers were compact, small framed animals which were purchased primarily for silage "cleanup" and were not fed the same ration throughout the feeding period. They were fed shelled corn and corn silage supplemented with Pro-Sil (liquid suspension of anhydrous ammonia, minerals and molasses, Ruminant Nitrogen Products Co., Adrian, Michigan). Proportions of corn and silage varied throughout the experiment. They were housed in an outside lot. Since ages were not known, weight or subjective estimation of market finish was used as an indication of being ready for slaughter.

Holstein Heifers. These heifers were part of an experiment conducted by Purchas (1969). Beginning at 2.5 months of age, 20 heifers were fed 0.9 kg of grain per day (low) and the other 20 were fed 4.5 kg of grain per day (high). Ten heifers from each group were fed 0.45 mg of melengestrol acetate (MGA, The Upjohn Co., Kalamazoo, Michigan) per animal per day (table 2). All heifers were fed corn silage and alfalfa hay ad libitum. They were kept in loose housing dry-lot facilities (MSU dairy department) and penned communally according to nutritional treatments. The heifers were slaughtered at breeding size (120 cm withers height).

TABLE 2. GROUPING OF HOLSTEIN HEIFERS ACCORDING TO TREATMENT.

Group number	Number of animals	Level of nutrition	MGA administration	Slaughter criteria
8	10	4.5 kg grain/day	0.45 mg/day from 2.5 mo.	Breeding size
9	10	0.9 kg grain/day	0.45 mg/day from 2.5 mo.	Breeding size
10	10	4.5 kg grain/day	None	Breeding size
11	10	0.9 kg grain/day	None	Breeding size

Holstein Steers. These steers were included in a nutritional experiment involving the feeding of two different maturity levels of corn silage. They were penned communally in two lots on the basis of nutritional treatment (table 3). Group 12 was fed corn silage harvested at 35% dry matter (DM) and group 13 was fed corn silage harvested at 46% DM. Silage at both levels of DM was treated with 22.5 g of Pro-Sil/kilogram of 35% DM silage (Henderson et al., 1971). These steers were slaughtered earlier than originally planned because of the depletion of the supply of corn silage.

Holstein Bulls. These bulls were provided by Michigan Animal Breeders Co-Op and Select Sires, Inc. They were housed inside either individually or in groups of 3 or 4 animals. They were fed a growing ration consisting of 4.4 kg of corn, 2.2 kg of oats, 0.5 kg of SBM, 0.1 kg of trace mineralized salt, 0.14 kg of molasses and 3.6 kg of mixed hay per day. These bulls were used for semen collection and were not slaughtered.

TABLE 3. GROUPING OF HEREFORD BULLS AND HOLSTEIN STEERS ACCORDING TO TREATMENT.

Group number	Breed	Sex	Number of animals	Treatment
6	Hereford	Bull	9	Selected for tenderness
7	Hereford	Bull	7	Selected for leanness
12	Holstein	Steer	10	Corn silage harvested at 35% DM + Pro-Sil
13	Holstein	Steer	9	Corn silage harvested at 46% DM + Pro-Sil

Serum Collection

The Holstein bulls were bled by tail vein puncture using vacutainers to collect 40 ml of blood. The remaining cattle were bled by jugular vein puncture while secured in a restraining chute. Both Angus steers and Hereford bulls became excited during blood collection. All blood was collected in polyethylene centrifuge tubes, allowed to stand at room temperature for 2 to 3 hr., loosened from the walls of the centrifuge tubes and cooled at 4 C for 24 hours. Serum was separated by centrifugation at 10,000 rpm in a Sorval RC-2B centrifuge (Ivan Sorval Inc., Norwolk, Connecticut), decanted into 7 dram plastic snap-cap vials and frozen at -30 C. The plasma collected from Holstein heifers was stored as described above for serum. Prior to hormone determinations, the serum or plasma was thawed overnight at 4 C and then at room temperature for one hour. Homogeneity of each sample was assured by inverting the vials 3 times.

Subsequent refreezing and thawing was carried out as described above.

Slaughter Procedures

Hereford bulls were slaughtered at the MSU Meat Laboratory in May, 1971. They were fasted for 24 hr. and transported about 2 miles from the Beef Cattle Research Center on the morning of slaughter. The cattle were stunned with a captive bolt pistol and exsanguinated within 3 min. of stunning. Blood for serum hormone measurement was collected immediately after the jugular vein was severed during exsanguination.

Angus and Holstein steers were transported 80 miles to a commercial abattoir (Farmer Peet's Packing Co., Bay City, Michigan) one day prior to slaughter. Two cattle were stunned at one time by captive bolt pistol followed by conventional exsanguination. The Holstein steers were slaughtered in March and Angus steers in June and July of 1971.

Holstein heifers were transported approximately 5 miles to a commercial abattoir (VanAlstine Packing Co., Okemos, Michigan), weighed, stunned with a captive bolt pistol, and bled within 3 hr. after being removed from their experimental pens at Michigan State University.

Thirty-six to 48 hr. postmortem, the right wholesale rib of Angus and Holstein steer and Hereford bull carcasses were removed, identified and shipped to the MSU Meat Laboratory where they were stored at 4 C for 24 hours. The ribs were then frozen in 46 x 76 cm cryovac bags (Cryovac Co., Cedar Rapids, Iowa) and stored at -30 C for 1 to 3 months. The right hind quarters of the Holstein heifer carcasses were identified and delivered to the MSU Meat Laboratory within 4 days of slaughter.

Measurement of Body Composition

Round. Physical separation analysis of the right round of Holstein heifers was determined as described by Purchas (1969). The right hind quarter was held at approximately 4 C and all dissection procedures were carried out at this temperature within 6 days post-slaughter. After removal of the perirenal fat from the hindquarter, the round was separated by cutting between the fourth and fifth sacral vertebrae through a point 2 cm anterior to the exposed portion of the symphysis pubis. The flank was removed by making a cut along a line and parallel to the plane of the exposed lumbar vertebrae, starting at a point located laterally from the longissimus muscle equal to its lateral axis on the exposed muscle surface. This cut was extended to meet a second cut made tangential to the ventral surface of the rectus femoris muscle. The rounds were weighed and dissected into fat, muscle and bone plus tendon with no attempt being made to dissect the distal 15 cm of the round which consisted mainly of tendons and bone.

9-10-11 Rib Section. Physical separation of the 9-10-11 rib section was carried out as an estimate of carcass fat, muscle and bone. The cutting procedure used was a modification of that described by Hankins and Howe (1946). Wholesale ribs were thawed at 4 C for 3 to 5 days depending on the size of the rib. If they were not completely thawed on the morning that separation was to take place, the ribs were thawed at room temperature for about 2 hours. The 9-10-11 rib section was removed by cutting parallel and adjacent to the posterior edges of the eighth and eleventh ribs. The

longissimus muscle and thoracic vertebrae were cut perpendicular to the long axis of the vertebral column of the wholesale rib. The distal ends of ribs 9, 10 and 11 were removed by cutting perpendicular to the ribs on a line measured 25 cm from the dorsal processes of the vertebrae.

Fat along the ventral edges of the vertebrae and medial side of the ribs as well as excessive juice and bone dust were removed from the 9-10-11 rib sections before they were weighed. Each 9-10-11 rib section was weighed to the nearest gram and placed into plastic bags until physical separation analyses were made. If a rib section was not completely separated after being started on one day, it was wrapped in a plastic bag and stored at 4 C overnight and completed the next day. All separations were carried out at 4 to 8 C.

The longissimus muscle was removed and trimmed free of fat but the epimysium was left intact. It was then weighed and immediately refrozen for later tenderness evaluation. The other large muscles of the 9-10-11 rib section were removed, freed of all separable fat and weighed as quickly as possible to decrease moisture loss from dripping and evaporation. Extreme care was taken to separate fat from the remaining muscles of each rib section. Components were weighed every 30 min. to minimize evaporation. All bones including cartilage were weighed and recorded as bone after being completely cleaned. The separable components were weighed to the nearest gram.

Longissimus Muscle Area. A compensating polar planimeter was used to measure tracings made on acetate paper of the exposed surface (12th rib)

of the longissimus muscle of the right hindquarter of all cattle.

Fat Thickness. A single fat thickness measurement was made of the subcutaneous fat at the 12th rib after ribbing. The measurement was made perpendicular to the outer fat surface at a point 3/4 the lateral length of the longissimus muscle from the vertical process of the 12th thoracic vertebra (American Meat Science Association, 1967) on the exposed 12th rib surface of the hindquarter.

Quality Measurements

Tenderness. Tenderness was measured by three different methods on the Angus and Holstein steers and Hereford bulls. Tenderness measurements were made on uncooked longissimus muscles using the Armour Tenderometer (Armour and Co., Chicago, Illinois) which is a nondestructive probe-type apparatus. The needles of the tenderometer probe were inserted into the longissimus muscle along its longitudinal axis between the 12th and 13th ribs of the wholesale ribs or intact forequarters. The probe assembly contained 10 penetration needles mounted on a manifold which in turn was attached to an electronic strain gage (Banks, 1971) from which the readings were directly obtained. Two separate readings were made on each rib by inserting the probe into the longissimus muscle until the penetration guide touched the muscle surface (5 cm penetration). Care was taken to prevent touching bone, large connective tissue strands or heavy intermuscular fat deposits. An average of the two readings was calculated and used in the subsequent statistical analysis.

Warner-Bratzler shear values and taste panel tenderness scores were determined on cooked steaks from the 9-10-11 rib sections of Angus and Holstein steers, 12th rib sections of Hereford bulls and steaks from the anterior end of the short loin of the Holstein heifers. Only the longissimus muscle of each of these steaks was evaluated for tenderness. Steaks from the Holstein heifers were cut approximately 2.5 cm thick, wrapped in aluminum foil and roasted to an internal temperature of 63 C in an electric oven preheated to 150 C. Shear measurements were determined 24 to 36 hours later on six - 2.2 cm cores with a Warner-Bratzler shear device.

Steaks 3.8 cm thick were sawed from frozen 9-10-11 rib sections of Angus and Holstein steers and thawed overnight at 4 C. Steaks from Hereford bulls were not frozen before cooking. The steaks from Angus and Holstein steers and Hereford bulls were cooked in deep fat (lard, 138 C) to an internal temperature of 63 to 71 C. Internal temperatures were monitored with a polyprobe potentiometer. The cooked steaks used for Warner-Bratzler shear determinations were stored overnight at 4 C. Ten - 1.2 cm cores were removed and used for the shear determinations.

Taste panels were conducted with untrained panelists (16 members) to evaluate tenderness, juiciness and overall acceptability. A standard 9 point hedonic scale was used. After cooking, the steaks were trimmed to remove the browned surfaces and they were then cut into approximately 1.2 cm cubes for tasting. A total of 13 different panels were conducted with four steaks being tested at each panel evaluation.

Hormone Determinations

Radioimmunoassay for GH. Purchas (1969) developed the double antibody radioimmunoassay for GH used in this study. Antibodies to bovine GH (NIH-GH-B12) were produced in guinea pigs with an initial subcutaneous injection of bovine GH and Freund's complete adjuvant followed by subsequent injections of GH and Freund's incomplete adjuvant (appendix I.C.2). Purchas (1969) determined that a 1:3200 dilution which bound roughly 50% of the iodinated GH was most suitable. Antibodies to guinea pig gamma globulin (Pentex, Kankakee, Illinois, Fraction II) were produced in the sheep as described above for guinea pigs (appendix I.C.1).

The assay (presented in detail in appendix I.E) involved the reaction of the unknown serum and GH standards with 200 µl of guinea pig anti-bovine GH (GPABGH) for 24 hr followed by the addition of 100 µl (30,000 cpm per 100 µl) of ^{125}I-GH and incubation for 24 hours. Two hundred µl of sheep anti-guinea pig-gamma globulin (SAGPGG) were then added and incubated at 4 C for 72 hours. After the incubation period, 3 ml of 0.01 M phosphate buffered saline (PBS), pH 7.0, were added and each tube centrifuged at 2500 x g for 30 min in a Sorval RC-3 swinging bucket centrifuge (Ivan Sorval, Inc., Norwolk, Connecticut). The supernatant was decanted, tubes inverted and allowed to drain for 30 min before being counted for 10 min or to 10,000 counts in a Nuclear-Chicago Model 4230 autogamma crystal scintillation counter.

Radioimmunoassay for Insulin. A modification of the radioimmunoassay for prolactin reported by Koprowski and Tucker (1971) was utilized to

quantify insulin. The assay consisted of a double antibody system using guinea pig antibovine insulin serum (GPABI) and sheep anti-guinea pig gamma globulin (SAGPGG) to form an insoluble complex with mass great enough to be precipitated when centrifuged at 2500 x g for 30 minutes.

Standards were prepared from purified bovine insulin (Eli Lilly and Co., Indianapolis, Indiana, lot 795372, 24.2 units per mg) with 100 µl of each standard containing 0.04, 0.06, 0.08, 0.1, 0.2, 0.3, 0.4, 0.5, 0.6, 0.8, 1.0, 1.2, 1.4, 1.6, 1.8, 2.0, 3.0, 4.0 or 5.0 ng of insulin (appendix I.B.6).

Guinea pig anti-bovine insulin (GPABI) was purchased from Miles Laboratories, Inc., Kankakee, Illinois (lot No. 21). The lyophilized serum was reconstituted with 1 ml of deionized water, diluted 1:400 in EDTA-PBS pH 7.0, (EDTA-PBS, appendix I.B.2) and frozen in 10 ml aliquots at -30 C. To determine the correct GPABI concentration the following titer check was carried out:

1) A microsyringe (Hamilton Co., Whittier, California) was used to transfer GPABI (1:400, appendix I.B.8) to Erlenmeyer flasks where various dilutions with normal guinea pig serum (NGPS, 1:400) as diluent were prepared.

2) 200 µl of the various dilutions of GPABI were added to 12 x 75 mm disposable culture tubes (Scientific Products Co., Romulus, Michigan).

3) 100 µl of ^{125}I-Insulin (approximately 18,000 cpm per 100 µl, Amersham Searle Co., Arlington Heights, Illinois, specific activity 50 µ ci per µg) were added to all tubes. They were then shaken and incubated at 4 C for 18 hours.

4) 200 µl SAGPGG (appendix I.B.9) were added, the tubes shaken and incubated for 24 hr under the same conditions described above.

5) Three ml of 0.01 M PBS were added to each tube and then centrifuged at 2500 x g for 30 minutes.

6) The supernatant was decanted and the tubes left inverted for 30 min on absorbent paper. The tubes were then wiped dry and counted in a Nuclear-Chicago Model 4230 autogamma scintillation counter.

7) The percent of labeled insulin bound was calculated for each dilution of GPABI. These results are presented in table 4 and figure 1.

A dilution of 1:105,000 GPABI sera was used in subsequent assays for the determination of unknown amounts of insulin. This dilution provides approximately 35% binding of the labeled insulin which is adequate to provide a reasonably fast counting time but low enough to keep nonspecific binding to a minimum. In addition, a range of 30 to 40% binding provided the greatest working range on the standard curve. If greater than 40% binding was used, sensitivity at the low concentrations of the curve was lost. In contrast, if less than 30% binding was used, sensitivity at higher concentrations was lost. At approximately 35% binding sensitivity of the assay ranged from .08 to 1.0 ng per tube (figure 2).

Validation of Insulin Assay. Hunter (1967) recommended that all unknown plasma should be determined at different dilutions as a check for dose response parallelism. A microsyringe was used to add 50 to 500 µl bovine and ovine sera to assay tubes to check dose response. Figure 2 shows parallel dose response curves of bovine and ovine seral dilutions to

TABLE 4. VARIOUS DILUTIONS OF GPABI AND THE CORRESPONDING PERCENT ^{125}I-INSULIN BOUND.

Dilution	Percent of ^{125}I-insulin bound
1:3600	82.3
1:6800	79.6
1:8800	81.0
1:20,800	74.8
1:40,000	60.7
1:50,000	50.3
1:60,000	48.5
1:70,000	44.0
1:80,000	40.2
1:90,000	37.1
1:100,000	34.5
1:110,000	30.7
1:120,000	29.2
1:130,000	27.2
1:140,000	24.6
1:150,000	24.9

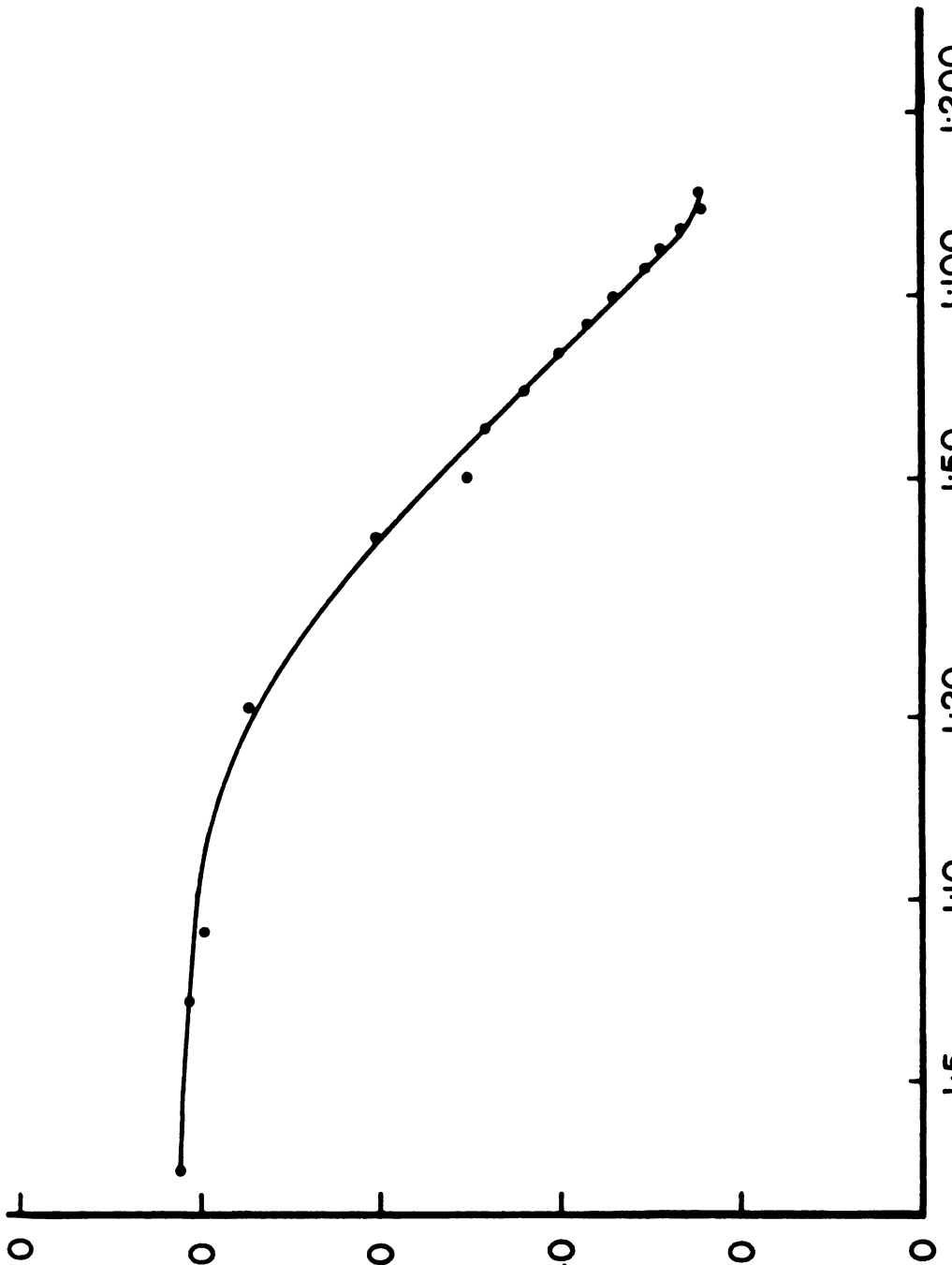

Figure 1. Dilution-response curve for guinea pig anti-bovine insulin serum.

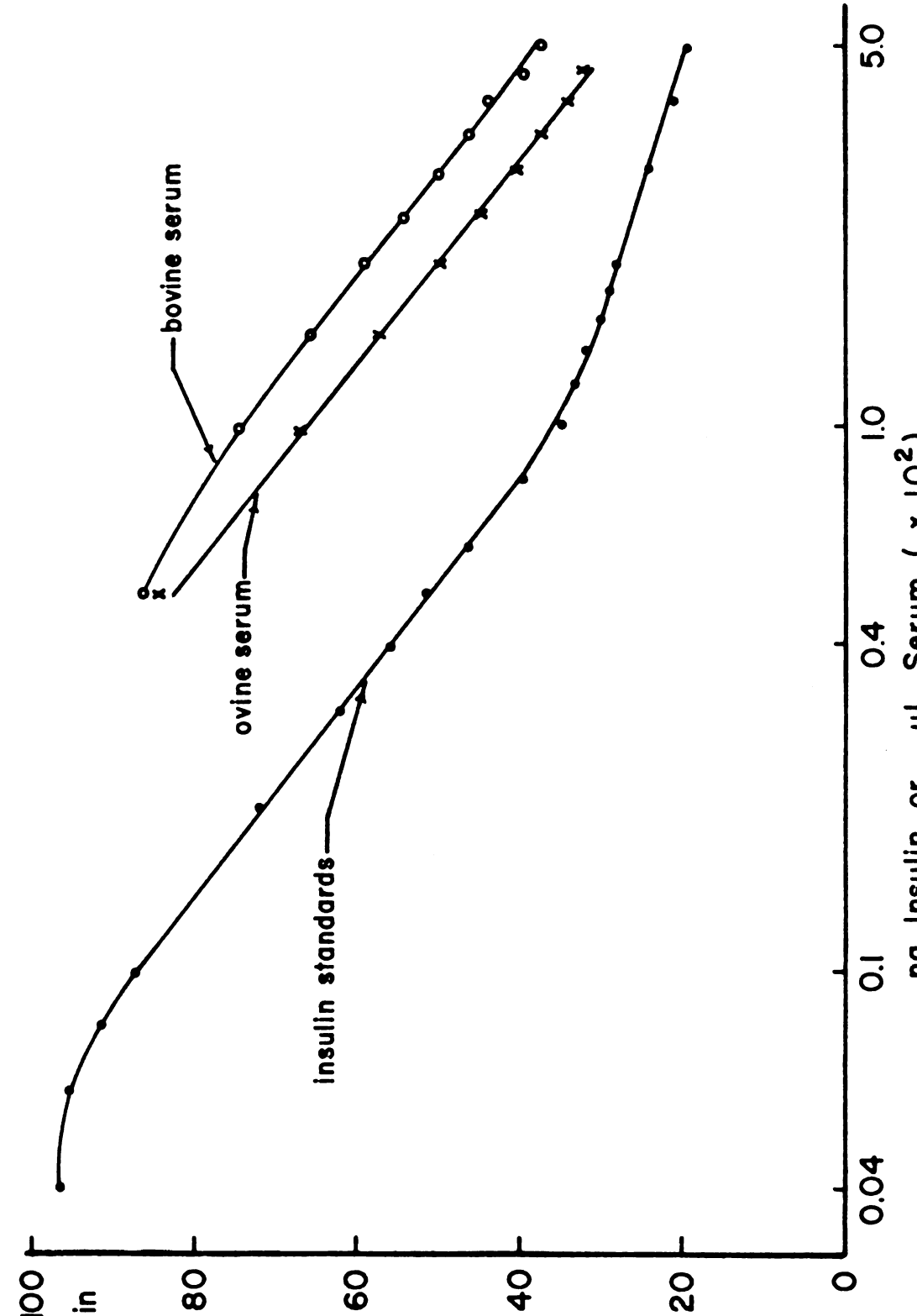

Figure 2.--Dose-response curves for insulin standards and for ovine and bovine sera.

the bovine insulin standard curve. The similar parallel curves of bovine and ovine serum suggest validity in using the bovine system to measure relative concentrations of ovine insulin.

Koprowski and Tucker (1971) suggested that close agreement between two quantities of diluted sera in estimating prolactin concentration indicates that a specific hormone is being measured. Therefore, all serum samples were usually assayed at 150 and 250 μl sera providing insulin concentration was such that values were on the working portion of the standard curve. In some cases, the serum had to be further diluted to be within the workable range of the standard curve. Normal bovine sera included with each assay at 150 and 250 μl had an insulin concentration of 36.8 ± 1 μ U per ml (1.52 ng per ml). This also provides an indication of the repeatability of the assay since the concentration given above is an average of 36 determinations over a 5 month period.

Another validation step of the assay was the determination of insulin recovery. One hundred μl of standards (0.1, 0.2, 0.3, 0.4, 0.5, 0.6, 0.8 and 1.0 ng per 100 μl) were added to 150 μl of normal bovine sera. Since the insulin concentration in the normal bovine sera was known to be 0.23 ng per 150 ul, the sum of the insulin standards plus bovine normal sera could be compared to the insulin concentration actually measured when the standards and normal sera were combined. Table 5 and figure 3 show the results of insulin recovery determinations.

Five hundred ng of bovine GH, FSH, LH and prolactin were incubated in the presence of GPABI to determine the specificity of the antibody for bovine insulin. In all cases, the binding of these hormones did not exceed

TABLE 5. DETERMINATION OF PERCENT INSULIN RECOVERED.

ng of insulin std added per tube	+	ng of insulin in 150 µl normal bovine sera	=	Total ng insulin added	ng insulin actually measured	Difference
0.1		0.23		0.33	0.35	+ .02
0.2		0.23		0.43	0.45	+ .02
0.3		0.23		0.53	0.56	+ .03
0.4		0.23		0.63	0.65	+ .02
0.5		0.23		0.73	0.72	- .01
0.6		0.23		0.83	0.82	- .01
0.8		0.23		1.03	0.98	- .05
1.0		0.23		1.23	1.15	- .08

the binding in those tubes which had only PBS-1% BSA. The sensitivity of the bovine insulin antibody for bovine proinsulin or fragments of bovine insulin was not determined. However, Kitabchi (1970) has reported that bovine and porcine proinsulins exhibit a similar degree of immunoreactivity and indicated that proinsulin reactivity with the insulin antibody was 25 to 33% that of insulin. Bovine and porcine connecting peptides had no significant immunoreactivity.

Procedure for Insulin Assay. 1) Either 250 or 350 µl of 0.05 M phosphate buffered saline - 1% bovine serum albumin, pH 7.4, (hereafter called Buffer B 1, appendix I.B.5) were added to all tubes prepared for serum samples. Four hundred µl of B 1 were added to the tubes for the

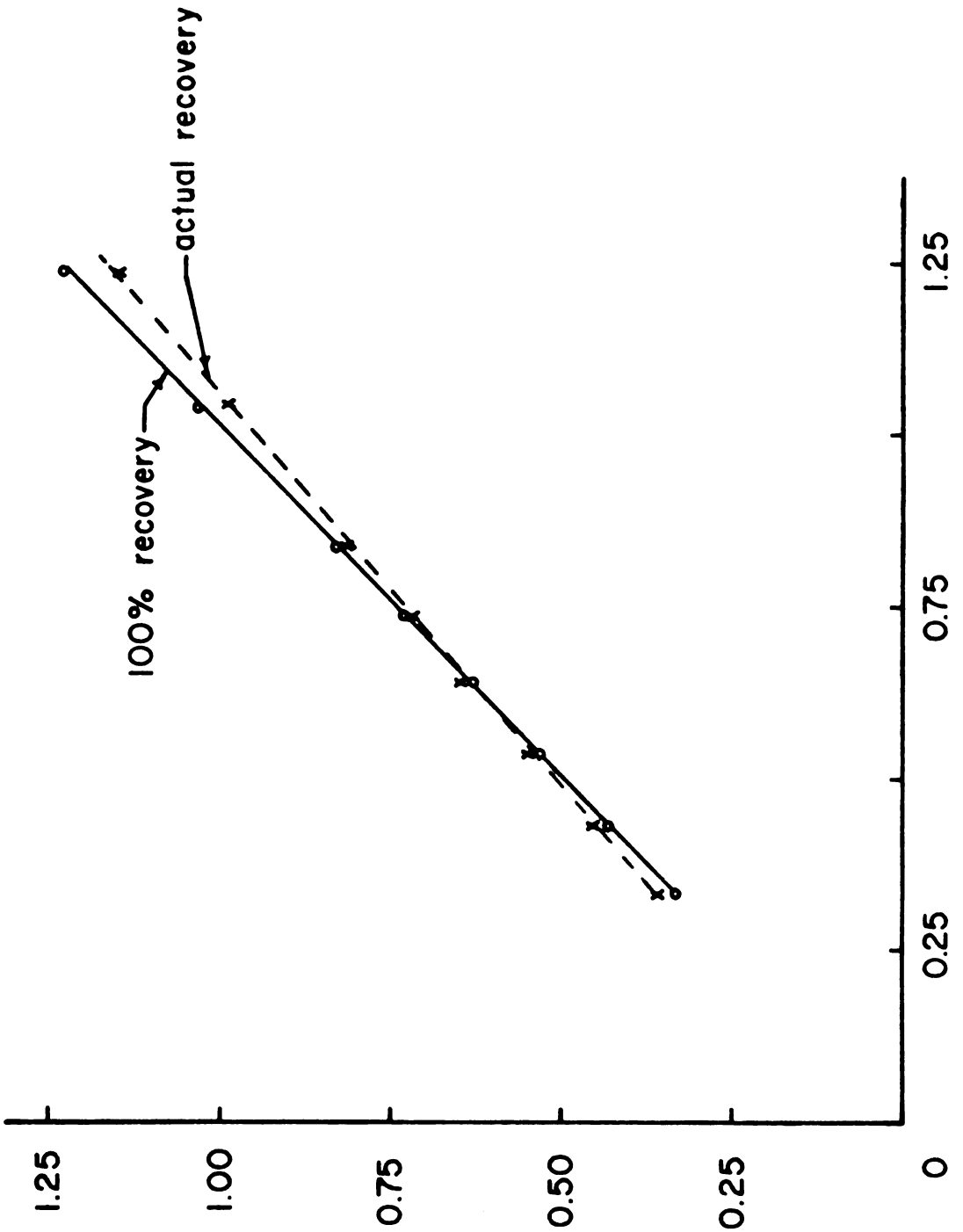

Figure 3. Recovery of exogenous bovine insulin added to 150 µl of bovine serum.

bovine insulin standards. This was added before volume was brought to 500 µl with serum samples (150 or 250 µl) and standards (100 µl), respectively, to prevent any binding of the hormone to walls of the glass culture tubes.

2) On day zero, 200 µl of GPABI diluted 1:105,000 (appendix I.B.8) in NGPS were added, the solution was shaken with a whirli-mixer, and then incubated for 24 hr at 4 C.

3) One hundred µl of ^{125}I-Insulin (approximately 18,000 cpm) were added on day one, shaken and incubated at 4 C for 24 hours.

4) On day two, 200 µl of SAGPGG (dilution which would optimally precipitate GPABI) were added, vortexed and allowed to incubate at 4 C for 96 hours.

5) Following incubation, 3 ml of PBS were added to each tube (except total count) and all tubes were then centrifuged at 2500 x g for 30 min in a refrigerated centrifuge with a swinging bucket rotor (Sorval Model RC-3, Ivan Sorval Inc., Norwolk, Connecticut).

6) The supernatant was decanted and the tubes left inverted on absorbent paper for 30 minutes. The tubes were wiped dry and counted for 10 min or 10,000 counts, whichever came first, in a Nuclear-Chicago Model 4230 autogamma scintillation counter. Tube number and counting time was simultaneously punched onto a paper tape (Teletype Corp., Skokie, Illinois) which was subsequently used in the calculation of unknown insulin concentrations.

Calculation of Results. A standard curve was calculated on the basis of percent of labeled insulin bound. Those tubes with 500 µl of B 1 in lieu of standard insulin were arbitrarily set at 100% binding. As the concentration of unlabeled insulin increased the percent of labeled insulin decreased in a dose response relationship (figure 2).

The average time for each standard concentration in four sets of standards was calculated and punched onto cards with their respective insulin concentrations. The calculated standard curve consistently accounted for greater than 99.5% of the variation in the curve. Regression coefficients calculated by the C.D.C. 3600 were entered into an Olivetti computer (Programma 101, Olivetti Underwood, New York, New York) which corrected for dilution and automatically calculated hormone concentrations of unknown sera as counting time and tube number were entered via the punched tape editor (Beckman Model 6912 Tape Editor, Beckman Instruments, Inc., Fullerton, California).

All antibody preparations, buffers and other reagents in the study are given in detail in appendix I.

Statistical Analysis

Data were analyzed on the C.D.C. 3600 computer at the Michigan State University Computer Laboratory. A least squares analysis was used to determine differences between treatment means. When significant differences were observed by least squares analysis, the Duncan's Multiple Range test was utilized to determine the group means differing from each other

(Snedecor and Cochran, 1969). Simple correlation coefficients were determined as described by Snedecor and Cochran (1969).

RESULTS AND DISCUSSION

The means and standard error of the means for all variables and correlation coefficients between the variables are presented in tables 8 to 24. The codes for each variable and their specific units used in the presentation of the results are defined in table 6. Each group of cattle and the treatments within groups are numbered and defined in table 7. The raw data for all variables are presented in appendix II.

Growth Rates

The Holstein steers were significantly ($P \leq .05$) heavier than Hereford bulls, Angus steers and Holstein heifers one month prior to slaughter and again at one day prior to slaughter (table 8). Hereford bulls had the highest ($P \leq .05$) average daily gain over the entire feeding period (ADGT) followed by Holstein heifers and steers which were not significantly ($P > .05$) different. Angus steers had the lowest ($P \leq .05$) ADGT. However, when average daily gain (ADGB) was computed for the period from the first to last bleeding (average of approximately 2.5 months) all groups were different ($P \leq .05$) from each other. Holstein steers had the highest ADGB followed by Holstein heifers, Hereford bulls and finally Angus steers. The data suggest that Holstein heifers and steers were in the acceleratory portion of the growth curve during the bleeding periods since their ADGB was greater than their ADGT. On the other hand, Hereford bulls and Angus steers had apparently reached the upper inflection

TABLE 6. NUMBER, CODE AND DEFINITION OF VARIABLES.

Number	Code	Definition
1	BR	Breed
2	SEX	Sex
3	RTN	Ration
4	T-L	Line selection in Hereford bulls (6 = tender line; 7 = lean line)
5	AN-NO	Animal number
6	C-WT	Carcass weight
7	FT-TH	Fat thickness over the twelfth rib (inches)
8	LMA	Longissimus muscle area (square inches)
9	RD-WT	Weight of the wholesale round (pounds)
10	RD-LN	Weight of separable lean from the wholesale round (pounds)
11	RD-FT	Weight of separable fat from the wholesale round (pounds)
12	RD-BN	Weight of separable bone from the wholesale round (pounds)
13	RD-L/BN	Round, lean to bone ratio
14	RD-L/FT	Round, lean to fat ratio
15	RD-% LN	Round, percent lean
16	RD-% FT	Round, percent fat
17	RD-% BN	Round, percent bone
18	ADGT	Average daily gain during total feeding period (pounds/day)
19	ADGB	Average daily gain during the period from first to last bleeding (pounds/day)
20	W-B	Warner-Bratzler shear values (pounds/square inch)
21	AT	Armour Tenderometer (pounds)
22	JUI	Taste panel juiciness (9 point hedonic scale)
23	OAA	Taste panel overall acceptability (9 point hedonic scale)
24	TEND	Taste panel tenderness (9 point hedonic scale)
25	RB-WT	Weight of the 9-10-11th rib section (pounds)
26	RB-LN	Weight of separable lean from the 9-10-11th rib section (pounds)
27	RB-FT	Weight of separable fat from the 9-10-11th rib section (pounds)
28	RB-BN	Weight of separable bone from the 9-10-11th rib section (pounds)
29	RB-L/BN	9-10-11 rib section, lean to bone ratio
30	RB-L/FT	9-10-11 rib section, lean to fat ratio
31	RB-% LN	Rib percent, lean
32	RB-% FT	Rib percent, fat
33	RB-% BN	Rib percent, bone

TABLE 6. NUMBER, CODE AND DEFINITION OF VARIABLES (continued)

Number	Code	Definition
34	WT-0	Live weight at slaughter or final bleeding (pounds)
35	WT-1	Live weight one bleeding period before slaughter (pounds)
36	WT-2	Live weight two bleeding periods before slaughter (pounds)
37	WT-3	Live weight three bleeding periods before slaughter (pounds)
38	WT-4	Live weight four bleeding periods before slaughter (pounds)
39	AGE-0	Age at slaughter or final bleeding (days)
40	AGE-1	Age one bleeding period before slaughter (days)
41	AGE-2	Age two bleeding periods before slaughter (days)
42	AGE-3	Age three bleeding periods before slaughter (days)
43	AGE-4	Age four bleeding periods before slaughter (days)
44	IN-A	Average serum insulin concentration of all bleeding periods (μU/ml)
45	IN-0	Serum insulin concentration at slaughter or final bleeding (μU/ml)
46	IN-1	Serum insulin concentration one bleeding before slaughter (μU/ml)
47	IN-2	Serum insulin concentration two bleedings before slaughter (μU/ml)
48	IN-3	Serum insulin concentration three bleedings before slaughter (μU/ml)
49	IN-4	Serum insulin concentration four bleedings before slaughter (μU/ml)
50	GH-A	Average serum GH concentration for all bleedings (ng/ml)
51	GH-0	Serum GH concentration at slaughter or final bleeding (ng/ml)
52	GH-1	Serum GH concentration one bleeding before slaughter (ng/ml)
53	GH-2	Serum GH concentration two bleedings before slaughter (ng/ml)
54	GH-3	Serum GH concentration three bleedings before slaughter (ng/ml)
55	GH-4	Serum GH concentration four bleedings before slaughter (ng/ml)

TABLE 7. GROUP NUMBER AND DESCRIPTION.

Group number	Description
1	Hereford bulls
2	Angus steers
3	Holstein heifers
4	Holstein steers
5	Holstein bulls
6	Hereford bulls selected for tenderness
7	Hereford bulls selected for leanness
8	Holstein heifers fed high nutrition plus MGA
9	Holstein heifers fed low nutrition plus MGA
10	Holstein heifers fed high nutrition without MGA
11	Holstein heifers fed low nutrition without MGA
12	Holstein steers fed corn silage harvested at 35% DM
13	Holstein steers fed corn silage harvested at 46% DM

TABLE 8. MEANS AND STANDARD ERRORS OF WEIGHTS, AGES AND GROWTH RATES OF VARIOUS BREED AND SEX GROUPS.

Variable code and number[a]	Group				
	Hereford bulls (1)	Angus steers (2)	Holstein heifers (3)	Holstein steers (4)	Holstein bulls (6)
WT-0 (34)	832.12 ± 19.34[b]	813.12 ± 15.67[b]	830.68 ± 12.02[b]	1111.16 ± 13.42[c]	1162.69 ± 42.14[c]
WT-1 (35)	804.25 ± 20.64[b]	784.18 ± 12.73[b]	772.06 ± 13.65[b]	998.42 ± 11.61[c]	1121.23 ± 38.72[d]
WT-2 (36)			699.64 ± 13.01[b]		1075.00 ± 47.80[c]
WT-3 (37)			628.42 ± 12.07		
WT-4 (38)			569.55 ± 12.21		
AGE-0 (39)	413.19 ± 4.24[b]		375.02 ± 7.54[c]		584.08 ± 19.10[d]
AGE-1 (40)	397.19 ± 4.24[b]		345.38 ± 7.15[c]		556.08 ± 19.10[d]
AGE-2 (41)			317.30 ± 7.06[b]		528.08 ± 19.10[c]
AGE-3 (42)			288.25 ± 7.08		
AGE-4 (43)			259.10 ± 7.04		
ADGT (18)	2.53 ± 0.05[b]	1.30 ± 0.06[d]	2.17 ± 0.06[c]	2.19 ± 0.06[c]	
ADGB (19)	1.74 ± 0.17[b]	0.85 ± 0.22[e]	2.27 ± 0.07[c]	3.13 ± 0.20[d]	1.57 ± 0.28[b]

[a]Units for each variable are defined in table 6.
[b,c,d,e]On any line, means with different superscripts differ significantly ($P < .05$).

of the growth curve (the period of growth rate deceleration) since their ADGB was less than ADGT (table 8). Live weight was significantly ($P \leq .05$) related to ADGT ($r = 0.28$) and ADGB ($r = 0.30$) among Hereford bulls, Angus and Holstein steers and Holstein heifers.

There were no significant differences in WT-0, ADGT or ADGB between Hereford bulls selected for leanness or tenderness (table 9). However, bulls selected for tenderness tended to have lower live weights and slightly higher daily gains than bulls selected for leanness.

Holstein heifers on a high level of nutrition (table 9) tended to have heavier live weights at slaughter. On a high level of nutrition, heifers had a higher ($P \leq .05$) ADGT than that observed for the ADGB. In contrast, heifers on a low level of nutrition gained faster ($P \leq .05$) during the last three months on feed (ADGB) than for the entire feeding period (ADGT). Thus, heifers on a low level of nutrition had lower ADG but maintained this growth rate for a longer period of time than heifers on a high level of nutrition; consequently, final weights were not significantly different between them. Similar results have been reported for pigs (Robinson, 1969) and feedlot cattle (Henrickson et al., 1965; Waldman, 1971). MGA treated heifers had higher ($P \leq .05$) ADGT but WT-0 and ADGB did not differ (table 9) compared to heifers receiving no MGA.

Both ADGT and ADGB (table 9) were higher ($P \leq .05$) among Holstein steers that received 35% DM corn silage compared to steers fed 46% DM silage. Henderson et al. (1971) noted a decrease in lactic acid from 10.8% in 35% DM silage to 7.3% lactic acid in 46% DM silage and suggested that the poorer fermentation in 46% DM silage possibly accounted for much of the reduced performance of steers fed 46% DM silage.

TABLE 9. MEANS AND STANDARD ERRORS OF GROWTH TRAITS OF TREATMENT SUBGROUPS WITHIN HEREFORD BULLS, HOLSTEIN HEIFERS AND HOLSTEIN STEERS.

Variable code and number[a]		Group[b]			
		Hereford bulls		Holstein steers	
		Tender	Lean	35% DM	46% DM
WT-0	(34)	809.44 ± 21.88	861.29 ± 32.64	1127.50 ± 17.23	1093.00 ± 20.15
WT-1	(35)	779.44 ± 23.62	836.14 ± 34.30	998.00 ± 18.08	998.89 ± 15.22
ADGT	(18)	2.56 ± 0.06	2.50 ± 0.08	2.30 ± 0.06[c]	2.07 ± 0.09[d]
ADGB	(19)	1.87 ± 0.21	1.57 ± 0.28	3.60 ± 0.22[c]	2.61 ± 0.27[d]
		Holstein heifers[e]		Holstein heifers[f]	
		High nutrition	Low nutrition	MGA	No MGA
WT-0	(34)	847.70 ± 18.59	813.65 ± 14.75	847.00 ± 20.06	814.35 ± 12.76
ADGT	(18)	2.84 ± 0.10[c]	2.06 ± 0.65[d]	2.26 ± 0.08[c]	2.08 ± 0.07[d]
ADGB	(19)	2.34 ± 0.13	2.20 ± 0.07	2.37 ± 0.10	2.16 ± 0.10

[a]Units for each variable are defined in table 6.

[b]Group treatments are defined in tables 2, 3 and 7.

[c,d]On any line within any breed and sex group, means with different superscripts differ significantly ($P < .05$).

[e]Means and standard errors of growth traits of Holstein heifers fed either high or low levels of nutrition regardless of MGA treatment.

[f]Means and standard errors of growth traits of Holstein heifers fed MGA or no MGA regardless of nutritional level.

Carcass Characteristics

Holstein steers had the heaviest ($P \leq .05$) carcasses followed in order by Angus steers, Hereford bulls and Holstein heifers (table 10). Holstein heifers had lighter carcasses ($P \leq .05$) than the other breed and sex groups. Longissimus muscle area did not differ among these four groups. However, 12th rib fat thickness was greatest in Angus steers followed by Hereford bulls and Holstein steers ($P \leq .05$). FT-TH was not measured in Holstein heifers. A significant correlation coefficient ($r = 0.31$, $P \leq .01$) was observed between LMA and carcass weight (not shown in table 11) and carcass weight was negatively ($P \leq .05$) related ($r = -.25$) to FT-TH (table 11). The negative relationship of C-WT with FT-TH was primarily a result of the low FT-TH of Holstein steers which had the heaviest carcasses combined with the high FT-TH of the Angus steers which had light weight carcasses. When sex and breed groups were compared, C-WT was correlated with FT-TH in Hereford bulls ($r = 0.58$, $P \leq .05$), Angus steers ($r = 0.28$, $P > .05$) and Holstein steers ($r = 0.34$, $P > .05$) (tables 12, 13 and 15).

Weights of the 9-10-11 rib sections did not differ significantly between Hereford bulls, Angus steers or Holstein steers; however, significant differences in separable components were observed (table 10). Angus steers had less RB-LN ($P \leq .05$) and more RB-FT ($P \leq .05$) than Hereford bulls or Holstein steers while Holstein steers had more RB-LN than Angus steers ($P \leq .05$) and less RB-FT and more RB-BN than either Hereford bulls or Angus steers ($P \leq .05$). Hedrick (1968) reported lower percentages of subcutaneous fat among dairy breeds compared to beef breeds and Branaman et al.

TABLE 10. MEANS AND STANDARD ERRORS OF CARCASS TRAITS OF VARIOUS BREED AND SEX GROUPS.

Variable code and number [a]		Group			
		Hereford bulls (1)	Angus steers (2)	Holstein heifers (3)	Holstein steers (4)
C-WT	(6)	489.50 ± 13.30[b]	519.76 ± 8.93[b]	442.85 ± 8.38[c]	592.05 ± 7.18[d]
FT-TH	(7)	0.23 ± 0.02[b]	0.62 ± 0.03[c]		0.11 ± 0.01[d]
LMA	(8)	10.62 ± 0.25	9.88 ± 0.23	9.91 ± 0.23	9.61 ± 0.28
RD-WT	(9)			55.75 ± 0.81	
RD-LN	(10)			37.43 ± 0.57	
RD-FT	(11)			7.62 ± 0.33	
RD-BN	(12)			10.75 ± 0.12	
RD-L/BN	(13)			3.49 ± 0.04	
RD-L/FT	(14)			5.19 ± 0.19	
RD-% LN	(15)			67.14 ± 0.37	
RD-% FT	(16)			13.26 ± 0.38	
RD-% BN	(17)			19.36 ± 0.21	
WB Shear	(20)	35.09 ± 1.33[b]	32.08 ± 1.33[b]	25.94 ± 0.78[c]	35.55 ± 1.63[b]
AT	(21)	17.78 ± 0.50[c]	15.23 ± 0.37[b]		16.75 ± 0.61[c]
JUI	(22)		6.53 ± 0.13[b]		6.11 ± 0.17[c]
OAA	(23)		6.73 ± 0.12[b]		6.25 ± 0.15[c]
TEND	(24)	6.17 ± 0.23			
RB-WT	(25)	8.30 ± 0.26	8.62 ± 0.16		8.34 ± 0.13
RB-LN	(26)	4.54 ± 0.13[c]	3.80 ± 0.08[b]		4.77 ± 0.09[c]
RB-FT	(27)	2.49 ± 0.11[b]	3.54 ± 0.15[c]		1.86 ± 0.07[d]
RB-BN	(28)	1.11 ± 0.04[b]	1.11 ± 0.02[b]		1.52 ± 0.03[c]
RB-L/BN	(29)	4.13 ± 0.12[b]	3.43 ± 0.10[c]		3.14 ± 0.06[d]
RB-L/FT	(30)	1.85 ± 0.06[b]	1.11 ± 0.06[c]		2.62 ± 0.11[d]
RB-% LN	(31)	54.89 ± 0.66[b]	44.27 ± 0.88[c]		57.58 ± 0.62[d]
RB-% FT	(32)	29.82 ± 0.61[b]	40.80 ± 1.18[c]		22.44 ± 0.70[d]
RB-% BN	(33)	13.43 ± 0.31[b]	12.85 ± 0.36[b]		18.40 ± 0.30[c]

[a]Units for each variable are defined in table 6.
[b,c,d]On any line, means with different superscripts differ significantly ($P < .05$).

(1962) reported higher percentages of bone in Holstein steers compared to Hereford steers. The above studies are in agreement with data presented in table 10. Although Holstein steers had the highest RB-% LN, Hereford bulls had higher R-L/BN ratios ($P \leq .05$) because of the higher ($P \leq .05$) RB-BN of Holstein steers. The RB-L/FT ratios were highest among Holstein steers ($P \leq .05$) and lowest for Angus steers ($P \leq .05$).

Carcass weight was significantly ($P \leq .01$) correlated with RB-WT ($r = 0.50$), RB-LN ($r = 0.55$), RB-BN ($r = 0.73$), RB-L/BN ($r = -.43$), RB-L/FT ($r = 0.38$) and RB-% BN ($r = 0.54$) as shown in table 11. FT-TH was related to RB-LN ($r = -.68$, $P \leq .01$), RB-BN ($r = -.52$, $P \leq .01$) and RB-% LN ($r = -.92$, $P \leq .01$). Since RB-LN and RB-BN were positively related to ADGT ($P \leq .05$) and ADGB ($P \leq .01$), and since both ADGT and ADGB were highly related to FT-TH ($P \leq .01$), the data suggest that 12th rib fat measurements should be a good selection trait if higher daily gains and percentages of lean are desired. LMA was related to RB-WT and RB-LN ($P \leq .05$) but not to other measures of growth or composition (table 11).

ADGT was highly related to RB-L/FT ($r = 0.55$), RB-% LN ($r = 0.70$) and RB-% FT ($r = -.62$). Likewise, ADGB was highly related ($P \leq .01$) to RB-L/FT ($r = 0.68$), RB-% LN ($r = 0.58$), RB-% FT ($r = -.66$) and RB-% BN ($r = 0.69$). These data suggest that selection for increased growth rate would also be effective in increasing the percent lean and lean to fat ratio while decreasing the percent fat of the 9-10-11 rib section or that of the entire carcass. Increasing percent carcass lean through selection for high daily gains would be extremely useful in animal production since daily gains are easier and less costly to obtain than carcass composition

and they can be measured without slaughtering the animal.

Tenderness (table 10), as measured by the Warner-Bratzler shear device was significantly ($P \leq .05$) higher (lower shear values) among Holstein heifers than Hereford bulls or Angus and Holstein steers. The latter three groups did not differ in tenderness. The difference in tenderness may be due to the method of cookery since steaks from Holstein heifers were roasted in foil while steaks from the other three groups were cooked in deep fat. In most cases, slow methods of cookery (roasting) results in greater tenderness providing other cooking variables are held constant. Angus steers had more acceptable ($P \leq .05$) juiciness (JUI) and overall acceptability (OAA) scores as determined by taste panel evaluation than Holstein steers. Parrish et al. (1970) have reported low correlations for juiciness and OAA with marbling. However, in this study steaks from Angus steers had significantly higher marbling scores and were judged to be more acceptable by taste panelists than those from Holstein steers. Juiciness and OAA (table 11) were related ($P \leq .05$) to FT-TH ($r = 0.30$ and $r = 0.36$, respectively). Taste panel tenderness was not related to any growth or compositional paramater of Hereford bulls.

Hereford bulls selected for tenderness had smaller ($P \leq .05$) LMA and less RB-BN than those selected for leanness (table 16). RB-LN was not significantly different between tenderness groups but bulls selected for tenderness had higher RB-L/BN ratios ($P \leq .05$) primarily as a result of their lower RB-BN than bulls selected for leanness. Although selection for LMA was effective, neither fat nor lean on a weight or percentage basis was affected by this selection. It appears that the selection

TABLE 11. SIMPLE CORRELATION COEFFICIENTS BETWEEN SOME CARCASS TRAITS OF HEREFORD BULLS, ANGUS STEERS AND HOLSTEIN STEERS.[a,b]

Variable code and number		Variable code								
		WT-0	C-WT	FT-TH	LMA	RB-WT	RB-LN	RB-FT	RB-BN	RB-L/BN
WT-0	(34)	1.00								
C-WT	(6)	--	1.00							
FT-TH	(7)	-.57	-.25	1.00						
LMA	(8)	--	--	0.03	1.00					
RB-WT	(25)	0.21	0.50	0.29	0.54	1.00				
RB-LN	(26)	0.63	0.55	-.68	0.43	0.40	1.00			
RB-FT	(27)	-.50	-.14	0.88	0.24	0.55	-.50	1.00		
RB-BN	(28)	0.85	0.73	-.52	-.08	0.19	0.61	-.53	1.00	
RB-L/BN	(29)	-.47	-.43	-.03	0.41	0.02	0.12	0.14	-.69	1.00
RB-L/FT	(30)	0.68	0.38	-.85	-.09	-.26	0.70	-.92	0.68	-.15
RB-% LN	(31)	0.53	0.22	-.92	0.02	-.31	0.74	-.93	0.48	0.12
RB-% FT	(32)	-.64	-.32	0.91	0.10	0.32	-.69	0.97	-.66	0.14
RB-% BN	(33)	0.78	0.54	-.64	-.34	-.21	0.42	-.76	0.90	-.70
ADGT	(18)	--	--	-.71	--	0.06	0.62	-.56	0.25	0.29
ADGB	(19)	--	--	-.62	--	-.13	0.44	-.61	0.61	-.30
WB Shear	(20)	--	--	-.28	--	-.15	0.17	-.30	0.23	-.09
AT	(21)	0.11	0.02	-.36	0.08	-.08	0.18	-.19	-.02	0.20
JUI	(22)	-.30	-.28	0.30	0.11	0.05	-.25	0.27	-.36	0.28
OAA	(23)	-.37	-.34	0.36	0.07	0.04	-.33	0.33	-.46	0.32

[a] n = 52

[b] $P \leq .05 = 0.273$; $P \leq .01 = 0.354$

TABLE 11. SIMPLE CORRELATION COEFFICIENTS BETWEEN SOME CARCASS TRAITS OF HEREFORD BULLS, ANGUS STEERS AND HOLSTEIN STEERS.[a,b] (continued)

Variable code and number		Variable code									
		RB-L/FT	RB-% LN	RB-% FT	RB-% BN	ADGT	ADGB	WB	AT	JUI	OAA
WT-0	(34)										
C-WT	(6)										
FT-TH	(7)										
LMA	(8)										
RB-WT	(25)										
RB-LN	(26)										
RB-FT	(27)										
RB-BN	(28)										
RB-L/BN	(29)										
RB-L/FT	(30)	1.00									
RB-% LN	(31)	0.93	1.00								
RB-% FT	(32)	-.97	-.96	1.00							
RB-% BN	(33)	0.78	0.61	-.79	1.00						
ADGT	(18)	0.55	0.70	-.62	0.30	1.00					
ADGB	(19)	0.68	0.58	-.66	0.69	--	1.00				
WB Shear	(20)	0.27	0.28	-.30	0.29	--	--	1.00			
AT	(21)	0.13	0.25	-.19	0.03	0.43	0.28	0.17	1.00		
JUI	(22)	-.22	-.25	0.27	-.33	-.30	-.36	-.39	-.12	1.00	
OAA	(23)	-.31	-.32	0.35	-.41	-.34	-.41	-.50	-.18	0.91	1.00

[a] n = 52

[b] $P \leq .05 = 0.273$; $P \leq .01 = 0.354$

TABLE 12. SIMPLE CORRELATION COEFFICIENTS BETWEEN SOME CARCASS TRAITS OF HEREFORD BULLS.[a,b]

Variable code and number		Variable code								
		WT-0	C-WT	FT-TH	LMA	RB-WT	RB-LN	RB-FT	RB-BN	RB-L/BN
WT-0	(34)	1.00								
C-WT	(6)	0.98	1.00							
FT-TH	(7)	0.58	0.58	1.00						
LMA	(8)	0.69	0.69	0.36	1.00					
RB-WT	(25)	0.78	0.76	0.49	0.64	1.00				
RB-LN	(26)	0.82	0.81	0.32	0.61	0.92	1.00			
RB-FT	(27)	0.62	0.60	0.60	0.48	0.92	0.71	1.00		
RB-BN	(28)	0.66	0.63	0.41	0.68	0.79	0.64	0.75	1.00	
RB-L/BN	(29)	-.12	-.09	-.25	-.33	-.22	0.05	-.34	-.73	1.00
RB-L/FT	(30)	-.15	-.13	-.55	-.13	-.50	-.16	-.80	-.51	0.54
RB-% LN	(31)	-.12	-.10	-.52	-.24	-.45	-.08	-.72	-.61	0.74
RB-% FT	(32)	0.18	0.17	0.56	0.07	0.50	0.17	0.80	0.45	-.45
RB-% BN	(33)	0.03	0.00	0.01	0.26	-.02	-.18	0.02	0.59	-.92
ADGT	(18)	0.62	0.64	0.44	0.21	0.45	0.56	0.36	0.15	0.28
ADGB	(19)	-.43	-.51	-.48	-.17	-.31	-.26	-.32	-.25	0.09
WB Shear	(20)	-.28	-.26	0.03	0.03	-.12	-.37	0.13	0.04	-.40
AT	(21)	0.15	0.15	-.05	0.25	0.21	0.18	0.29	0.05	0.08
TEND	(24)	0.24	0.21	-.04	0.23	0.42	0.41	0.27	0.54	-.28

[a] n = 16

[b] $P \leq .05 = 0.497$; $P \leq .01 = 0.623$

TABLE 12. SIMPLE CORRELATION COEFFICIENTS BETWEEN SOME CARCASS TRAITS OF HEREFORD BULLS.[a,b] (continued)

Variable code and number		Variable code								
		RB-L/FT	RB-% LN	RB-% FT	RB-% BN	ADGT	ADGB	WB shear	AT	TEND
WT-0	(34)									
C-WT	(6)									
FT-TH	(7)									
LMA	(8)									
RB-WT	(25)									
RB-LN	(26)									
RB-FT	(27)									
RB-BN	(28)									
RB-L/BN	(29)									
RB-L/FT	(30)	1.00								
RB-% LN	(31)	0.94	1.00							
RB-% FT	(32)	-.99	-.89	1.00						
RB-% BN	(33)	-.19	-.43	0.09	1.00					
ADGT	(18)	-.07	0.12	0.14	-.36	1.00				
ADGB	(19)	0.22	0.21	-.25	0.01	-.22	1.00			
WB Shear	(20)	-.49	-.56	0.46	0.27	-.35	0.05	1.00		
AT	(21)	-.27	-.11	0.31	-.18	0.38	0.35	0.21	1.00	
TEND	(24)	0.00	-.13	-.04	0.32	-.10	-.15	-.37	-.28	1.00

[a] n = 16

[b] $P \leq .05 = 0.497$; $P \leq .01 = 0.623$

TABLE 13. SIMPLE CORRELATION COEFFICIENTS BETWEEN SOME CARCASS TRAITS OF ANGUS STEERS.[a,b]

Variable code and number		Variable code									
		WT-0	C-WT	FT-TH	LMA	RB-WT	RB-LN	RB-FT	RB-BN	RB-L/BN	RB-L/FT
WT-0	(34)	1.00									
C-WT	(6)	0.82	1.00								
FT-TH	(7)	0.41	0.28	1.00							
LMA	(8)	0.61	0.53	-.03	1.00						
RB-WT	(25)	0.76	0.91	0.36	0.69	1.00					
RB-LN	(26)	0.26	0.49	-.32	0.69	0.51	1.00				
RB-FT	(27)	0.71	0.80	0.55	0.42	0.89	0.07	1.00			
RB-BN	(28)	-.21	-.45	-.03	-.14	-.45	-.06	-.60	1.00		
RB-L/BN	(29)	0.32	0.64	-.23	0.59	0.65	0.74	0.44	-.71	1.00	
RB-L/FT	(30)	-.59	-.64	-.67	-.10	-.67	0.25	-.92	0.56	-.19	1.00
RB-% LN	(31)	-.50	-.44	-.70	0.04	-.49	0.49	-.82	0.40	0.09	0.95
RB-% FT	(32)	0.60	0.65	0.62	0.16	0.71	-.21	0.95	-.62	0.26	-.99
RB-% BN	(33)	-.46	-.66	-.09	-.54	-.78	-.36	-.78	0.83	-.81	0.59
ADGT	(18)	0.72	0.39	0.42	0.33	0.37	0.07	0.32	0.23	-.07	-.26
ADGB	(19)	0.66	0.24	0.46	0.33	0.27	-.15	0.34	0.14	-.19	-.34
WB Shear	(20)	-.05	0.12	-.31	0.14	0.01	0.59	-.31	0.26	0.24	0.44
AT	(21)	0.09	-.01	-.20	-.01	-.07	-.25	0.09	-.34	0.07	-.15
JUI	(22)	0.38	0.21	0.02	0.38	0.19	0.12	0.16	-.14	0.18	-.03
OAA	(23)	0.43	0.23	-.06	0.22	0.14	0.08	0.11	-.06	0.11	-.05

[a] n = 17

[b] $P \leq .05 = 0.482$; $P \leq .01 = 0.606$

TABLE 13. SIMPLE CORRELATION COEFFICIENTS BETWEEN SOME CARCASS TRAITS OF ANGUS STEERS.[a,b] (continued)

Variable code and number		Variable code								
		RB-% LN	RB-% FT	RB-% BN	ADGT	ADGB	WB shear	AT	JUI	OAA
WT-0	(34)									
C-WT	(6)									
FT-TH	(7)									
LMA	(8)									
RB-WT	(25)									
RB-LN	(26)									
RB-FT	(27)									
RB-BN	(28)									
RB-L/BN	(29)									
RB-L/FT	(30)									
RB-% LN	(31)	1.00								
RB-% FT	(32)	-.94	1.00							
RB-% BN	(33)	0.42	-.68	1.00						
ADGT	(18)	-.29	0.24	-.05	1.00					
ADGB	(19)	-.41	0.33	-.03	0.65	1.00				
WB Shear	(20)	0.57	-.46	0.12	0.05	-.40	1.00			
AT	(21)	-.17	0.17	-.19	-.08	0.35	-.44	1.00		
JUI	(22)	-.04	0.08	-.20	0.24	0.17	-.14	-.07	1.00	
OAA	(23)	-.04	0.06	-.05	0.37	0.27	-.18	-.04	0.81	1.00

[a] n = 17

[b] $P \leq .05 = 0.482$; $P \leq .01 = 0.606$

TABLE 14. SIMPLE CORRELATION COEFFICIENTS BETWEEN SOME CARCASS TRAITS OF HOLSTEIN HEIFERS.[a,b]

Variable code and number		Variable code						
		WT-0	C-WT	LMA	RD-WT	RD-LN	RD-FT	RD-BN
WT-0	(34)	1.00						
C-WT	(6)	0.92	1.00					
LMA	(8)	0.64	0.71	1.00				
RD-WT	(9)	0.86	0.92	0.73	1.00			
RD-LN	(10)	0.83	0.80	0.67	0.93	1.00		
RD-FT	(11)	0.52	0.62	0.34	0.52	0.27	1.00	
RD-BN	(12)	0.40	0.52	0.49	0.67	0.59	0.19	1.00
RD-L/BN	(13)	0.65	0.50	0.36	0.53	0.70	0.13	-.15
RD-L/FT	(14)	-.25	-.41	-.15	-.25	0.06	-.90	-.01
RD-% LN	(15)	0.06	-.16	-.03	-.04	0.34	-.61	-.14
RD-% FT	(16)	0.42	0.57	0.28	0.43	0.12	0.84	0.08
RD-% BN	(17)	-.73	-.68	-.46	-.64	-.65	-.49	0.13
ADGT	(18)	0.35	0.31	0.40	0.25	0.24	0.05	0.16
ADGB	(19)	0.39	0.28	0.41	0.24	0.24	0.04	0.21
WB Shear	(20)	-.09	-.09	0.06	0.00	0.04	-.03	0.12

[a] n = 40

[b] $P \leq .05 = 0.312$; $P \leq .01 = 0.403$

TABLE 14. SIMPLE CORRELATION COEFFICIENTS BETWEEN SOME CARCASS TRAITS OF HOLSTEIN HEIFERS.[a,b] (continued)

Variable code and number		Variable code							
		RD-L/BN	RD-L/FT	RD-% LN	RD-% FT	RD-% BN	ADGT	ADGB	WB shear
WT-0	(34)								
C-WT	(6)								
LMA	(8)								
RD-WT	(9)								
RD-LN	(10)								
RD-FT	(11)								
RD-BN	(12)								
RD-L/BN	(13)	1.00							
RD-L/FT	(14)	0.11	1.00						
RD-% LN	(15)	0.55	0.81	1.00					
RD-% FT	(16)	0.06	-.91	-.77	1.00				
RD-% BN	(17)	-.88	0.31	-.11	-.49	1.00			
ADGT	(18)	0.16	0.01	0.02	0.08	-.13	1.00		
ADGB	(19)	0.12	0.06	0.06	-.01	-.07	0.86	1.00	
WB Shear	(20)	-.03	0.11	0.15	-.19	0.13	0.06	0.01	1.00

[a] n = 40

[b] $P \leq .05 = 0.312$; $P \leq .01 = 0.403$

TABLE 15. SIMPLE CORRELATION COEFFICIENTS BETWEEN SOME CARCASS TRAITS OF HOLSTEIN STEERS.[a,b]

Variable code and number		Variable code									
		WT-0	C-WT	FT-TH	LMA	RB-WT	RB-LN	RB-FT	RB-BN	RB-L/BN	RB-L/FT
WT-0	(34)	1.00									
C-WT	(6)	0.84	1.00								
FT-TH	(7)	0.38	0.34	1.00							
LMA	(8)	0.15	0.44	0.04	1.00						
RB-WT	(25)	0.49	0.69	0.49	0.64	1.00					
RB-LN	(26)	0.31	0.45	0.17	0.75	0.74	1.00				
RB-FT	(27)	0.35	0.60	0.22	0.30	0.48	-.05	1.00			
RB-BN	(28)	0.52	0.51	0.44	0.21	0.58	0.54	0.02	1.00		
RB-L/BN	(29)	-.25	-.11	-.34	0.52	0.09	0.42	-.09	-.54	1.00	
RB-L/FT	(30)	-.09	-.24	-.08	0.13	0.00	0.57	-.84	0.27	0.29	1.00
RB-% LN	(31)	-.19	-.28	-.21	0.22	-.02	0.58	-.78	0.02	0.57	0.95
RB-% FT	(32)	0.11	0.29	0.07	0.00	0.08	-.47	0.90	-.31	-.15	-.98
RB-% BN	(33)	0.16	-.07	0.25	-.45	-.13	-.10	-.45	0.67	-.81	0.32
ADGT	(18)	0.65	0.30	0.38	-.35	0.02	-.11	-.12	0.19	-.30	0.05
ADGB	(19)	0.50	0.15	0.23	-.38	-.05	-.06	-.17	0.22	-.28	0.11
WB Shear	(20)	0.23	0.00	-.04	-.18	-.20	-.06	-.34	0.27	-.30	0.25
AT	(21)	0.05	0.15	-.08	-.17	-.18	-.31	0.13	-.23	-.05	-.26
JUI	(22)	-.25	-.29	-.11	-.08	-.19	-.04	-.26	-.20	0.18	0.24
OAA	(23)	-.33	-.32	-.10	-.08	-.20	-.06	-.21	-.34	0.29	0.17

[a] n = 19

[b] $P \leq .05 = 0.456$; $P \leq .01 = 0.575$

TABLE 15. SIMPLE CORRELATION COEFFICIENTS BETWEEN SOME CARCASS TRAITS OF HOLSTEIN STEERS.[a,b] (continued)

Variable code and number		Variable code								
		RB-% LN	RB-% FT	RB-% BN	ADGT	ADGB	WB shear	AT	JUI	OAA
WT-0	(34)									
C-WT	(6)									
FT-TH	(7)									
LMA	(8)									
RB-WT	(25)									
RB-LN	(26)									
RB-FT	(27)									
RB-BN	(28)									
RB-L/BN	(29)									
RB-L/FT	(30)									
RB-% LN	(31)	1.00								
RB-% FT	(32)	-.90	1.00							
RB-% BN	(33)	0.02	-.46	1.00						
ADGT	(18)	-.03	-.10	0.35	1.00					
ADGB	(19)	0.02	-.17	0.35	0.72	1.00				
WB Shear	(20)	0.12	-.31	0.49	0.53	0.61	1.00			
AT	(21)	-.23	0.26	-.11	0.06	0.07	0.24	1.00		
JUI	(22)	0.25	-.21	-.05	-.21	-.42	-.43	0.01	1.00	
OAA	(23)	0.25	-.15	-.20	-.23	-.47	-.58	-.07	0.94	1.00

[a] n = 19

[b] $P \leq .05 = 0.456$; $P \leq .01 = 0.575$

pressure for LMA affected only RB-BN. Additionally, LMA and RB-BN were significantly related ($r = 0.68$, $P \leq .05$). No other measures of carcass composition were affected by the selection pressures for tenderness or leanness. Likewise, selection for tenderness was not effective in increasing tenderness as measured by Warner-Bratzler shear and taste panel.

Variation in dry matter of corn silage did not affect any measure of rib composition of Holstein steers (table 16). However, steers fed 46% DM silage had lower ($P \leq .05$) W-B shear values and more acceptable taste panel JUI and OAA scores.

Holstein heifers fed a high level of nutrition had heavier carcasses and round weights and more separable fat ($P \leq .05$) than heifers fed a low level of nutrition (table 16). In addition, the low level of nutrition decreased RD-% FT and increased RD-L/FT ratios and RD-% LN ($P \leq .05$) when compared to the high levels of nutrition. Nutritional level did not influence tenderness. MGA depressed RD-BN and consequently increased ($P \leq .05$) the RD-L/BN. Other carcass characteristics (table 16) were not affected by MGA treatment except for tenderness which was greater among heifers fed MGA ($P \leq .05$).

Hormones

All animals were bled at approximately 30 day intervals; however, all breed and sex groups were not bled an equal number of times (table 18). The average serum hormone values (IN-A and GH-A) include all monthly bleedings as well as those at slaughter.

TABLE 16. MEANS AND STANDARD ERRORS OF CARCASS TRAITS OF TREATMENT SUBGROUPS WITHIN HEREFORD BULLS, HOLSTEIN HEIFERS AND HOLSTEIN STEERS.

Variable code and number[a]		Group[b]			
		Hereford bulls		Holstein steers	
		Tender	Lean	35% DM	46% DM
C-WT	(6)	474.89 ± 14.06	508.29 ± 23.81	601.10 ± 10.49	582.00 ± 9.12
LMA	(8)	10.20 ± 0.26[c]	11.17 ± 0.38[d]	9.35 ± 0.25	9.89 ± 0.52
FT-TH	(7)	0.21 ± 0.02	0.26 ± 0.02	0.12 ± 0.01	0.11 ± 0.01
RB-WT	(25)	8.21 ± 0.39	8.41 ± 0.33	8.46 ± 0.19	8.20 ± 0.19
RB-LN	(26)	4.58 ± 0.18	4.50 ± 0.19	4.76 ± 0.10	4.78 ± 0.17
RB-FT	(27)	2.43 ± 0.15	2.56 ± 0.17	1.92 ± 0.11	1.80 ± 0.09
RB-BN	(28)	1.04 ± 0.06[c]	1.21 ± 0.03[d]	1.56 ± 0.06	1.49 ± 0.03
RB-L/BN	(29)	4.44 ± 0.12[c]	3.73 ± 0.13[d]	3.08 ± 0.07	3.21 ± 0.10
RB-L/FT	(30)	1.91 ± 0.06	1.79 ± 0.11	2.55 ± 0.15	2.71 ± 0.16
RB-% LN	(31)	55.89 ± 0.71	53.58 ± 1.05	56.99 ± 0.79	58.23 ± 0.98
RB-% FT	(32)	29.39 ± 0.57	30.38 ± 1.22	22.86 ± 1.03	21.97 ± 0.98
RB-% BN	(33)	12.65 ± 0.23[c]	14.44 ± 0.41[d]	18.61 ± 0.46	18.17 ± 0.38
WB Shear	(20)	33.86 ± 1.78	36.67 ± 1.99	38.91 ± 2.40[c]	31.82 ± 1.53[d]
AT	(21)	17.89 ± 0.57	17.64 ± 0.94	17.28 ± 0.47[c]	16.17 ± 1.17
JUI	(22)			5.69 ± 0.14[c]	6.57 ± 0.24[d]
OAA	(23)			5.88 ± 0.13[c]	6.68 ± 0.19[d]
TEND	(24)	6.11 ± 0.29	6.24 ± 0.39		

TABLE 16. MEANS AND STANDARD ERRORS OF CARCASS TRAITS OF TREATMENT SUBGROUPS WITHIN HEREFORD BULLS, HOLSTEIN HEIFERS AND HOLSTEINS STEERS. (continued)

Variable code and number[a]		Group[b]			
		Holstein heifers[e]		Holstein heifers[f]	
		High nutrition	Low nutrition	MGA	No MGA
C-WT	(6)	468.20 ± 11.14[c]	417.50 ± 9.84[d]	448.95 ± 14.41	436.75 ± 8.77
LMA	(8)	10.57 ± 0.35	9.26 ± 0.23	9.88 ± 0.38	9.95 ± 0.27
RD-WT	(9)	57.58 ± 1.08[c]	53.92 ± 1.08[d]	55.38 ± 1.37	56.11 ± 0.89
RD-LN	(10)	37.91 ± 0.77	36.96 ± 0.85	37.50 ± 0.94	37.37 ± 0.67
RD-FT	(11)	8.51 ± 0.39[c]	6.73 ± 0.45[d]	7.41 ± 0.43	7.83 ± 0.58
RD-BN	(12)	10.95 ± 0.13	10.55 ± 0.20	10.47 ± 0.16[c]	11.03 ± 0.17[d]
RD-L/BN	(13)	3.47 ± 0.06	3.52 ± 0.06	3.58 ± 0.06[c]	3.41 ± 0.05[d]
RD-L/FT	(14)	4.59 ± 0.18[c]	5.79 ± 0.27[d]	5.27 ± 0.22	5.11 ± 0.31
RD-% LN	(15)	65.82 ± 0.40[c]	68.46 ± 0.47[d]	67.69 ± 0.50	64.82 ± 1.70
RD-% FT	(16)	14.69 ± 0.51[c]	11.83 ± 0.37[d]	13.24 ± 0.52	13.27 ± 0.57
RD-% BN	(17)	19.10 ± 0.32	19.63 ± 0.26	19.03 ± 0.31	19.69 ± 0.28
WB Shear	(20)	25.60 ± 1.16	26.28 ± 1.05	24.65 ± 0.82[c]	27.20 ± 1.28[d]

[a]Units for each variable are defined in table 6.

[b]Group treatments are defined in tables 2, 3 and 7.

[c,d]On any line within any breed and sex group, means with different superscripts differ significantly ($P < .05$).

[e]Means and standard errors of growth traits of Holstein heifers fed either high or low levels of nutrition regardless of MGA treatment.

[f]Means and standard errors of growth traits of Holstein heifers fed MGA or no MGA regardless of nutritional level.

TABLE 17. MEANS AND STANDARD ERRORS OF GROWTH, CARCASS AND ENDOCRINE TRAITS OF HOLSTEIN HEIFERS.

Variable code and number [a]		Treatment [b] High nutrition with MGA (8)	Low nutrition with MGA (9)	High nutrition without MGA (10)	Low nutrition without MGA (11)
WT-0	(34)	890.20 ± 25.17^{c}	803.80 ± 25.53^{d}	805.20 ± 20.60^{d}	823.50 ± 15.65^{cd}
WT-1	(35)	829.40 ± 26.24^{c}	747.14 ± 38.57^{d}	753.89 ± 19.33^{d}	748.50 ± 20.48^{d}
WT-2	(36)	749.50 ± 24.83	676.71 ± 38.68	694.44 ± 21.23	670.50 ± 16.70
WT-3	(37)	669.10 ± 23.51	604.10 ± 28.53	640.50 ± 22.72	600.00 ± 16.95
WT-4	(38)	598.60 ± 24.37	549.40 ± 30.72	585.90 ± 22.73	544.30 ± 17.00
ADGT	(18)	2.54 ± 0.09^{c}	1.98 ± 0.06^{d}	2.02 ± 0.13^{d}	2.13 ± 0.07^{d}
ADGB	(19)	2.73 ± 0.10^{c}	2.02 ± 0.08^{d}	1.94 ± 0.16^{d}	2.37 ± 0.09^{cd}
C-WT	(6)	491.90 ± 15.32^{c}	406.00 ± 15.24^{d}	444.50 ± 12.81^{d}	429.00 ± 12.13^{d}
LMA	(8)	10.97 ± 0.50	8.78 ± 0.31	10.16 ± 0.47	9.73 ± 0.28
RD-WT	(9)	58.92 ± 1.60^{d}	51.86 ± 1.63^{c}	56.26 ± 1.41^{d}	55.98 ± 1.16^{cd}
RD-LN	(10)	39.16 ± 1.11	35.84 ± 1.37	36.66 ± 0.97	38.08 ± 0.93
RD-FT	(11)	8.73 ± 0.55^{d}	6.09 ± 0.29^{c}	8.29 ± 0.57^{d}	7.36 ± 0.82^{cd}
RD-BN	(12)	10.87 ± 0.17^{c}	10.08 ± 0.21^{d}	10.02 ± 0.20^{d}	11.03 ± 0.27^{c}
RD-L/BN	(13)	3.60 ± 0.08	3.55 ± 0.10	3.33 ± 0.08	3.48 ± 0.07
RD-L/FT	(14)	4.62 ± 0.26^{c}	5.93 ± 0.19^{d}	4.57 ± 0.26^{c}	5.66 ± 0.51^{d}
RD-% LN	(15)	66.48 ± 0.65^{c}	68.90 ± 0.54^{d}	65.16 ± 0.39^{cd}	68.02 ± 0.77^{cd}
RD-% FT	(16)	14.75 ± 0.72^{c}	11.74 ± 0.37^{d}	14.63 ± 0.75^{c}	11.92 ± 0.65^{d}
RD-% BN	(17)	18.53 ± 0.38	19.52 ± 0.45	19.67 ± 0.48	19.71 ± 0.30
WB Shear	(20)	25.08 ± 1.17^{cd}	24.24 ± 1.17^{c}	26.11 ± 2.04^{cd}	28.30 ± 1.58^{d}
IN-A	(44)	67.43 ± 8.89^{c}	64.93 ± 11.33^{c}	31.78 ± 4.00^{d}	38.31 ± 6.05^{d}
IN-0	(45)	65.30 ± 11.25	52.58 ± 13.71	28.55 ± 4.55	34.77 ± 6.50
IN-1	(46)	77.28 ± 8.10^{c}	76.14 ± 20.64^{c}	32.17 ± 8.46^{d}	50.34 ± 9.03^{cd}
IN-2	(47)	83.90 ± 26.81^{c}	76.42 ± 19.25^{c}	30.15 ± 5.93^{d}	31.67 ± 3.00^{d}
IN-3	(48)	59.98 ± 18.96	66.72 ± 20.07	36.68 ± 5.52	28.23 ± 3.66
IN-4	(49)	58.80 ± 13.58	58.82 ± 13.34	32.07 ± 4.22	45.71 ± 18.44
GH-A	(50)	3.03 ± 0.54	2.74 ± 0.29	3.40 ± 0.59	3.48 ± 0.55
GH-0	(51)	2.05 ± 0.31	3.73 ± 0.64	3.57 ± 0.59	2.39 ± 0.51
GH-1	(52)	2.61 ± 0.85	3.17 ± 0.68	2.37 ± 0.54	2.00 ± 0.33
GH-2	(53)	1.92 ± 0.26	1.89 ± 0.34	2.37 ± 0.55	1.96 ± 0.29
GH-3	(54)	3.78 ± 1.57	2.22 ± 0.62	6.20 ± 2.59	7.12 ± 2.33
GH-4	(55)	4.68 ± 1.92	2.51 ± 0.54	2.57 ± 0.46	3.93 ± 1.05

[a]Units for each variable are defined in table 6. [b]Levels of nutrition are defined in table 2.
[c,d]On any line, means with different superscripts differ significantly ($P < .05$).

TABLE 18. MEANS AND STANDARD ERRORS OF SERUM GROWTH HORMONE AND INSULIN OF VARIOUS BREED AND SEX GROUPS.

Variable code and number [a]	Group				
	Hereford bulls (1)	Angus steers (2)	Holstein heifers (3)	Holstein steers (4)	Holstein bulls (5)
IN-A (44)	53.12 ± 4.72[b]	44.78 ± 6.00[b]	50.61 ± 4.62[b]	76.81 ± 9.28[c]	36.74 ± 3.16[b]
IN-0 (45)	46.97 ± 6.13	47.75 ± 10.28	45.82 ± 5.24	48.26 ± 4.69	32.34 ± 2.68
IN-1 (46)	59.22 ± 6.76[bc]	43.99 ± 7.55[b]	58.98 ± 6.81[b]	87.91 ± 15.01[c]	37.94 ± 5.76[b]
IN-2 (47)		34.35 ± 4.27[b]	54.65 ± 8.65[b]	94.27 ± 13.98[c]	40.44 ± 4.40[b]
IN-3 (48)		52.94 ± 7.87	47.59 ± 7.16		
IN-4 (49)			48.59 ± 6.70		
GH-A (50)[e]	19.93 ± 2.27[b]	32.72 ± 3.84[d]	3.16 ± 0.25[c]	35.47 ± 3.87[d]	16.45 ± 2.26[b]
GH-0 (51)[e]	17.96 ± 3.04[b]	41.61 ± 7.72[d]	2.94 ± 0.28[c]	35.46 ± 5.39[d]	14.43 ± 3.51[b]
GH-1 (52)[e]	21.89 ± 2.57[b]	31.99 ± 5.90[d]	2.52 ± 0.31[c]	33.41 ± 6.13[d]	14.09 ± 1.76[b]
GH-2 (53)[e]		27.09 ± 4.48[d]	2.04 ± 0.19[b]	37.53 ± 5.05[c]	20.85 ± 3.26[d]
GH-3 (54)[e]		29.78 ± 5.96[b]	4.83 ± 0.98[c]		
GH-4 (55)[e]			3.45 ± 0.58		

[a]Units for each variable are defined in table 6.
[b,c,d]On any line, means with different superscripts differ significantly ($P < .05$).
[e]Multiply GH of Holstein heifers x 2.5 to correct for differences in NIH-GH standards.

Neither serum insulin nor GH of Hereford bulls, Angus steers, Holstein heifers or Holsteins bulls changed significantly during the bleeding periods. Serum insulin of Holstein steers decreased from 94 μ U/ml at 2 months prior to slaughter to 48 μ U/ml on the day of slaughter; however, GH concentration did not change during this same period of time. Trenkle (1970) reported an increase in insulin concentration with length of time in the feedlot. He attributed this observation to increased grain consumption.

Since hormone concentrations showed little change throughout the bleeding period, presentation of the results and subsequent discussion will be restricted to average hormone concentrations (GH-A and IN-A). Holstein steers had significantly ($P \leq .05$) higher IN-A than Hereford bulls, Angus steers and Holstein heifers and bulls. The latter four groups did not differ significantly but Holstein bulls tended to have the lowest insulin concentrations (table 18). Trenkle (1970b) noted that the concentration of plasma insulin in finishing cattle appeared to be closely related to consumption of grain and concentrate in the ration. He also reported that lambs fed corn plus alfalfa hay had nearly twice as much plasma insulin as lambs fed only alfalfa hay. When readily fermentable carbohydrates are fed to ruminants a greater proportion of total volatile fatty acids produced in the rumen is made up of propionate and butyrate (Trenkle, 1970a). These fatty acids have been shown to significantly increase insulin concentrations when infused into mature sheep (Trenkle, 1970a). In the present study, Holstein steers were fed a corn silage diet that had been treated with Pro-Sil (anhydrous ammonia, molasses and minerals). The high insulin values of Holstein steers may be due to the corn silage diet since corn silage is readily fermentable and has been found to produce high levels of

butyrate (Bergen personal communication). In addition, possible nonfermented or residual molasses could provide a source of readily digestible carbohydrate which might account for the increased serum insulin of Holstein steers.

There were no significant differences in GH-A between Hereford and Holstein bulls or between Angus steers and Holstein steers; however, bulls of both breeds had less GH-A ($P \leq .05$) than Angus and Holstein steers. Holstein heifers had significantly ($P \leq .05$) lower GH-A than the other breed and sex groups. Trenkle (1971 b) reported that dietary energy level had no effect on plasma GH concentration, although in one experiment he observed that sheep on grain diets tended to have lower plasma GH levels than those fed high roughage diets. In addition, he reported that neither feeding nor fasting for 72 hr had any influence on plasma GH, even though blood glucose was depressed and plasma FFA were elevated among fasted animals. Although no record was made of the consumption of roughage of each breed and sex group in the present study, it is possible that the Angus and Holstein steers received higher roughage diets than the other breed and sex groups since they were fed primarily corn silage.

The plasma GH values for Holstein heifers were determined by Purchas (1969). When plasma GH was assayed on these same samples for this study (1971), approximately 2.5 fold greater values were observed. The only explanation appears to be the possibility that a change in GH standards may have been responsible for the observed 2.5 fold differences since all other variables were the same as those described by Purchas (1969). In addition, pooled normal plasma concentrations in four GH assays were nearly

identical, essentially eliminating the possibility that the 2.5 fold difference occurred merely by chance alone. In order to compare GH values between breed and sex groups, a 2.5 fold correction should be applied to the Holstein heifers. Even after applying a 2.5 fold correction, Holstein heifers still had the lowest plasma GH levels compared to Hereford bulls, Angus and Holstein steers or Holstein bulls. Siers and Swiger (1971) reported that sex did not influence serum GH levels in pigs. They noted that pigs at a constant age and size did not differ in serum GH levels but that differences in animal size were responsible for differences observed in circulating GH concentrations. In contrast, Stern, Baile and Mayer (1971) have suggested that ruminants responded differently at different ages (suckling, weanling and mature) to various GH stimulants. For example, injections of deoxy-glucose into ruminants increased GH most among suckling calves and least in mature cattle while arginine infusion increased GH least among suckling ruminants.

The GH values reported in this study were somewhat higher than those reported by Trenkle (1967; 1971 b) for sheep but are within the ranges reported for cattle (Trenkle, 1967; Dev and Lasley, 1969; Yousef et al., 1969; Stern et al., 1971). Eaton, Klosterman and Johnson (1968a) have reported that bleeding chute stresses increased serum GH from 6 ng/ml to 116 ng/ml. This high level (116 ng/ml) declined rapidly to 13.5 ng/ml after 7 minutes. Additionally, they noted that GH levels determined in 24 daily samples fluctuated markedly from day to day with a standard error greater than the mean. Koprowski, Tucker and Convey (1972) reported that serum GH of dairy cows did not exhibit circadian periodicity and was much

more stable than prolactin values from the same cows. Eaton et al. (1968a) noted that the day to day variation as well as mean GH values decreased as age increased in Holstein cattle. Eaton et al. (1968 b) observed some breed, sex and age differences for serum GH in cattle but concluded that, in view of the effects of stress, any differences should be cautiously interpreted. He suggested that different GH levels in stressed animals may reflect either true differences in normal circulating levels or may reflect differences due to stress-response, length of stress, pituitary reserves or combinations of these and other factors.

Correlation coefficients between serum insulin and GH in this study were low (table 19) and nonsignificant. Trenkle (1971 c) reported that large doses of insulin were required to create hypoglycemic conditions in sheep. He further stated that hypoglycemia per se was not a stimulus for growth hormone secretion but rather sudden decreases in blood glucose provided the stimulus for GH secretion. The low but consistently negative correlations found between serum insulin and GH levels in this study may be a result of the insensitivity of ruminants to insulin induced hypoglycemia.

There were no significant differences in serum insulin or GH between the tender and lean lines of Hereford bulls (table 20). However, the tender line tended to have higher insulin and lower GH concentrations than the lean line of bulls. Among the Holstein steers, nutritional treatment did not affect serum IN-A but GH-A was significantly ($P \leq .05$) higher among steers receiving 46% DM corn silage compared to steers fed 35% DM corn silage.

TABLE 19. SIMPLE CORRELATION COEFFICIENTS BETWEEN VARIOUS HORMONE VALUES[a].

Variable code and number[b]	Variable code and number[b]					
	IN-A	IN-0	IN-1	GH-A	GH-0	GH-1
IN-A	1.0					
IN-0	0.54	1.0				
IN-1	0.83	0.34	1.0			
GH-A	-.03	-.11	-.07	1.0		
GH-0	-.05	-.12	-.07	0.79	1.0	
GH-1	-.03	-.10	-.09	0.90	0.63	1.0

[a] $n = 105$; $P \leq .05 = 0.192$; $P \leq .01 = 0.251$.
[b] Units for each variable are defined in table 6.

The level of grain in the diet did not influence plasma IN-A or GH-A of Holstein heifers (table 20) which is in contrast to the findings of Trenkle (1970a, 1970b, 1971b) for ruminants. However, feeding MGA to Holstein heifers significantly ($P \leq .05$) increased plasma insulin. Haist (1965) suggested that adrenal and gonadal steroids increased insulin producing tissue and concomitantly increased insulin secretion. Bassett and Wallace (1967) reported that daily injections of cortisol produced hyperglycemia in sheep and resulted in a marked increase in plasma insulin. These authors observed that among cortisol treated animals, glucose and insulin changes were exaggerated following feeding; however, insulin was not related to increases in glucose. A direct effect of cortisol on insulin secretion has not been reported, thus, Bassett and Wallace (1967) suggested that cortisol and possibly all glucocorticoids are antagonistic to the action of insulin on glucose metabolism in the sheep. It is possible that other steroids may act similarily to cortisol in decreasing glucose metabolism. If this in fact is true, the elevated insulin levels observed for both progestin and estrogen treated cattle may be the result of the attempt of the pancreas to overcome the steroid antagonism to its action on glucose metabolism. Trenkle (1970b) has reported plasma insulin values of 46.8 μU/ml, 67.6 μU/ml and 107.0 μU/ml in control, MGA and stilbestrol treated finishing heifers, respectively. In this study, plasma GH was depressed by MGA but the differences were not significant. If blood glucose remains elevated during MGA treatment and if the hypothesis that GH is released only when blood glucose levels fall rapidly is accepted, then the observation that MGA depresses GH would be expected. However, blood

TABLE 20. MEANS AND STANDARD ERRORS OF SERUM INSULIN AND GROWTH HORMONE VALUES OF TREATMENT SUBGROUPS WITHIN HEREFORD BULLS, HOLSTEIN HEIFERS AND HOLSTEIN STEERS.

Variable code and number[a]		Group[b]			
		Hereford bulls		Holstein steers	
		Tender	Lean	35% DM	46% DM
IN-A	(44)	57.88 ± 6.23	47.00 ± 7.05	77.40 ± 13.65	76.16 ± 13.27
IN-0	(45)	51.54 ± 8.08	41.09 ± 9.60	47.61 ± 5.64	48.99 ± 8.04
IN-1	(46)	64.16 ± 8.78	52.89 ± 10.83	103.72 ± 24.16[c]	70.34 ± 16.34[d]
IN-2	(47)			80.88 ± 17.09	109.16 ± 22.61
GH-A	(50)	17.90 ± 1.57	22.54 ± 4.82	29.53 ± 4.27[c]	42.07 ± 6.18[d]
GH-0	(51)	16.43 ± 3.05	19.92 ± 5.97	30.15 ± 5.91	41.36 ± 9.28
GH-1	(52)	19.37 ± 3.08	25.14 ± 4.29	19.18 ± 5.39[c]	49.22 ± 9.09
GH-2	(53)			39.26 ± 8.29	35.60 ± 5.87
		Holstein heifers[e]		Holstein heifers[f]	
		High nutrition	Low nutrition	MGA	No MGA
IN-A	(44)	49.60 ± 6.26	51.62 ± 6.96	66.18 ± 7.02[c]	33.42 ± 3.79[d]
IN-0	(45)	48.97 ± 7.48	42.68 ± 7.17	59.64 ± 8.60[c]	32.01 ± 4.09[d]
IN-1	(46)	54.72 ± 7.70	63.24 ± 11.35	76.71 ± 10.79[c]	41.26 ± 6.37[d]
IN-2	(47)	54.04 ± 13.56	55.22 ± 11.28	79.74 ± 15.53[c]	30.87 ± 3.34[d]
IN-3	(48)	47.72 ± 9.55	47.48 ± 10.87	63.53 ± 13.50[c]	32.46 ± 3.37[d]
IN-4	(49)	45.44 ± 7.57	51.92 ± 11.38	58.81 ± 9.28[c]	38.89 ± 9.34[d]
GH-A	(50)	3.22 ± 0.39	3.11 ± 0.31	2.88 ± 0.30	3.44 ± 0.39
GH-0	(51)	2.81 ± 0.37	3.06 ± 0.42	2.89 ± 0.40	2.98 ± 0.40
GH-1	(52)	2.49 ± 0.49	2.55 ± 0.38	2.87 ± 0.54	2.18 ± 0.31
GH-2	(53)	2.14 ± 0.30	1.93 ± 0.22	1.91 ± 0.21	2.16 ± 0.31
GH-3	(54)	4.99 ± 1.50	4.67 ± 1.30	3.00 ± 0.84	6.66 ± 1.69
GH-4	(55)	3.62 ± 0.99	3.26 ± 0.61	2.30 ± 0.42	3.25 ± 0.58

[a]Units for each variable are defined in table 6.

[b]Group treatments are defined in tables 2, 3 and 7.

[c,d]On any line within any breed and sex group, means with different superscripts differ significantly ($P < .05$).

[e]Means and standard errors of growth traits of Holstein heifers fed either high or low levels of nutrition regardless of MGA treatment.

[f]Means and standard errors of growth traits of Holstein heifers fed MGA or no MGA regardless of nutritional level.

glucose was not measured in these heifers and any relationship between blood glucose levels and hormone values are cautiously inferred.

Relationship of Hormones to Growth and Carcass Characteristics

Carcass compositional data were determined on the 9-10-11 rib section of Hereford bulls, Angus steers and Holstein steers and on the wholesale round of Holstein heifers. Although correlations of hormones with carcass traits varied somewhat at different bleeding times, the discussion will be primarily restricted to the relationship of the average hormone values with growth and carcass characteristics. The Holstein bulls were not slaughtered and they will not be included in this section of the results and discussion. In addition, discussion of simple correlation coefficients will be limited to breed and sex groups since relationships of hormones with growth and carcass criteria were not determined for subgroups within breed and sex groups.

Average insulin values tended to be positively correlated with ADGT and ADGB (table 21) among Hereford bulls and Angus and Holstein steers and with WT-0 in Angus and Holstein steers. The only significant relationships of IN-A were observed with RB-L/BN ($r = 0.51$, $P \leq .05$) and RB-% BN ($r = -.55$, $P \leq .05$) of Angus steers. Although nonsignificant, IN-A of Hereford bulls and Angus and Holstein steers was negatively correlated with 12th rib fat thickness and positively correlated with Warner-Bratzler shear values. There did not appear to be any consistent trend, either negative or positive, among the correlations of IN-A with any other carcass characteristic for the Hereford bulls, Angus steers or Holstein steers.

TABLE 21. SIMPLE CORRELATION COEFFICIENTS OF SOME SERUM INSULIN VALUES WITH VARIOUS GROWTH AND CARCASS TRAITS.

Variable code and number[a]		Variable code and number[a]								
		IN-A (44)			IN-0 (45)			IN-1 (46)		
		Group[b]			Group			Group		
		1[c]	2[d]	4[e]	1	2	4	1	2	4
WT-0	(34)	0.00	0.40	0.40	-.05	0.49	0.09	0.04	0.48	0.48
ADGT	(18)	0.10	0.32	0.30	0.34	0.44	0.10	-.16	0.30	0.38
ADGB	(19)	0.12	0.32	0.27	0.19	0.38	0.31	0.00	0.26	0.42
C-WT	(6)	0.07	0.45	0.10	0.01	0.44	-.03	0.09	0.51	0.22
FT-TH	(7)	-.14	-.18	-.05	0.19	-.21	0.16	-.37	-.23	0.00
LMA	(8)	0.05	0.34	-.13	-.06	0.24	0.09	0.13	0.35	-.14
WB shear	(20)	0.06	0.24	0.07	0.32	0.07	0.14	-.21	-.06	0.17
OAA	(23)	--	0.33	-.27	--	0.33	-.08	--	0.33	-.43
TEND	(24)	-.32	--	--	-.53	--	--	0.04	--	--
RB-WT	(25)	-.03	0.41	0.00	-.05	0.27	0.05	-.01	0.41	0.08
RB-LN	(26)	0.09	0.24	-.10	-.07	0.22	0.05	0.20	0.19	-.04
RB-FT	(27)	-.11	0.39	0.19	0.07	0.23	0.10	-.22	0.42	0.18
RB-BN	(28)	-.27	-.46	0.13	-.20	-.35	0.22	-.19	-.51	0.26
RB-L/BN	(29)	0.38	0.51	-.24	0.14	0.42	-.18	0.40	0.50	-.32
RB-L/FT	(30)	0.22	-.26	-.21	-.19	-.14	-.07	0.48	-.29	-.16
RB-% LN	(31)	0.31	-.16	-.25	-.06	-.05	-.12	0.49	-.21	-.25
RB-% FT	(32)	-.17	0.31	0.19	0.25	0.18	0.04	-.47	0.35	0.15
RB-% BN	(33)	-.37	-.55	0.12	-.26	-.40	0.15	-.28	-.57	0.21

[a]Units for each variable are defined in table 6.
[b]Group numbers are identified in table 7.
[c]n = 16; $P \leq .05 = 0.497$; $P \leq .01 = 0.623$.
[d]n = 17; $P \leq .05 = 0.482$; $P \leq .01 = 0.606$.
[e]n = 19; $P \leq .05 = 0.456$; $P \leq .01 = 0.575$.

Pooled correlations for these three groups will be discussed later.

Growth hormone tended to be negatively correlated with WT-0 and ADGT of Hereford bulls, Angus steers and Holstein steers (table 22) and with ADGB of Angus and Holstein steers. The only significant relationship involving GH-A was observed with ADGT ($r = -.48$, $P \leq .05$) and ADGB ($r = -.48$, $P \leq .05$) among the Holstein steers. GH-A was correlated with FT-TH ($r = -.54$, $P \leq .05$) in Hereford bulls and with LMA ($r = -.51$, $P \leq .05$) and ($r = 0.60$, $P \leq .01$) in Angus and Holstein steers, respectively. RB-WT, RB-LN, RB-FT and RB-L/BN tended to be negatively related to GH-A of Hereford bulls and Angus steers while the same relationships were positive among Holstein steers. GH-A was significantly related to RB-% FT ($r = -.50$, $P \leq .05$) and RB-% BN ($r = 0.66$, $P \leq .01$) among the Hereford bulls only. Although the relationships were not significant, there was a trend for RB-L/FT and RB-% LN to be positively correlated with GH-A in all three groups of cattle. However, similar to the observations with insulin, there did not appear to be any consistent trends among the relationships of GH-A with other carcass traits (table 22).

Among the Holstein heifers, IN-A (table 23) was related to WT-0 ($r = 0.51$, $P \leq .01$) and tended to be positively related to ADGT and ADGB. IN-A was positively correlated with C-WT ($r = 0.39$, $P \leq .01$) and RD-WT ($r = 0.35$, $P \leq .01$). IN-A was not related to any measure of fat but was highly related ($P \leq .01$) to all lean and bone compositional characteristics except for RD-BN. All expressions of bone were negatively related with IN-A while lean measurements were positively correlated with IN-A ($P \leq .01$).

The relationships of GH-A with growth and carcass characteristics were generally opposite in sign to those of IN-A. However, exceptions to

TABLE 22. SIMPLE CORRELATION COEFFICIENTS OF SOME SERUM GROWTH HORMONE VALUES WITH VARIOUS GROWTH AND CARCASS TRAITS.

Variable code and number[a]		Variable code and number[a]								
		GH-A (50)			GH-0 (51)			GH-1 (52)		
		Group[b]			Group			Group		
		1[c]	2[d]	4[e]	1	2	4	1	2	4
WT-0	(34)	-.35	-.47	-.09	-.40	-.68	0.27	-.14	-.24	-.23
ADGT	(18)	-.44	-.11	-.48	-.46	-.53	-.14	-.24	-.04	-.54
ADGB	(19)	0.28	-.40	-.48	0.26	-.40	-.16	0.19	-.22	-.56
C-WT	(6)	-.33	-.40	0.25	-.38	-.63	0.45	-.13	-.25	0.04
FT-TH	(7)	-.54	0.01	-.09	-.54	-.23	0.20	-.31	0.11	-.04
LMA	(8)	0.11	-.51	0.60	0.03	-.50	0.76	0.16	-.11	0.44
WB shear	(20)	0.06	-.09	-.29	0.04	-.37	-.11	0.05	-.11	-.47
OAA	(23)	--	-.43	0.23	--	-.14	0.12	--	-.51	0.39
TEND	(24)	0.28	--	--	0.33	--	--	0.11	--	--
RB-WT	(25)	-.35	-.43	0.28	-.36	-.62	0.51	-.20	-.18	0.10
RB-LN	(26)	-.30	-.36	0.44	-.27	-.47	0.64	-.21	-.20	0.27
RB-FT	(27)	-.48	-.31	0.02	-.49	-.45	0.11	-.27	-.11	-.02
RB-BN	(28)	0.10	0.15	0.13	-.06	0.05	0.19	0.25	0.09	-.04
RB-L/BN	(29)	-.40	-.35	0.30	-.14	-.37	0.44	-.54	-.21	0.30
RB-L/FT	(30)	0.40	0.15	0.18	0.46	0.29	0.27	0.17	0.04	0.12
RB-% LN	(31)	0.16	0.07	0.22	0.28	0.16	0.31	-.04	-.01	0.19
RB-% FT	(32)	-.50	-.19	-.13	-.53	-.29	-.14	-.25	-.05	-.09
RB-% BN	(33)	0.66	0.35	-.18	0.40	0.34	-.30	0.69	0.14	-.22

[a]Units for each variable are defined in table 6.
[b]Group numbers are identified in table 7.
[c]$n = 16$; $P \leq .05 = 0.497$; $P \leq .01 = 0.623$
[d]$n = 17$; $P \leq .05 = 0.482$; $P \leq .01 = 0.606$
[e]$n = 19$; $P \leq .05 = 0.456$; $P \leq .01 = 0.575$

TABLE 23. SIMPLE CORRELATION COEFFICIENTS OF SOME SERUM HORMONE VALUES WITH VARIOUS GROWTH AND CARCASS TRAITS OF HOLSTEIN HEIFERS.[a]

Variable code and number[a]		Hormone[b]					
		Insulin			GH		
		IN-A (44)	IN-0 (45)	IN-1 (46)	GH-A (50)	GH-0 (51)	GH-1 (52)
WT-0	(34)	0.51	0.32	0.53	-.25	-.35	-.06
ADGT	(18)	0.29	0.11	0.27	0.04	-.27	-.07
ADGB	(19)	0.20	0.08	0.29	0.15	-.34	-.05
C-WT	(6)	0.39	0.27	0.35	-.17	-.26	-.03
LMA	(8)	0.15	0.10	0.18	-.05	-.29	-.15
WB Shear	(20)	-.01	-.04	0.17	0.07	-.07	-.04
RD-WT	(9)	0.35	0.23	0.27	-.16	-.38	-.07
RD-LN	(10)	0.49	0.28	0.38	-.21	-.45	-.08
RD-FT	(11)	0.04	0.08	0.04	0.03	-.02	0.00
RD-BN	(12)	-.15	-.15	-.11	0.20	-.31	-.07
RD-L/BN	(13)	0.74	0.45	0.55	-.40	-.29	-.03
RD-L/FT	(14)	0.09	0.01	0.11	-.06	-.08	0.00
RD-% LN	(15)	0.45	0.18	0.34	-.17	-.24	-.03
RD-% FT	(16)	0.02	0.10	0.01	-.13	0.03	0.03
RD-% BN	(17)	-.62	-.46	-.48	0.42	0.22	0.00

[a]Units for each variable are defined in table 6.
[b]$n = 40$; $P \leq .05 = 0.312$; $P \leq .01 = 0.403$.

the preceding statement involves the relationships of IN-A and GH-A with ADGT and ADGB both of which were nonsignificantly but psoitively correlated. The only significant relationship of GH-A was observed with RD-L/BN ($r = -.40$, $P \leq .05$) and RD-% BN ($r = 0.42$, $P \leq .05$). In all but three variables (W-B shear, RD-BN and RD-% FT), the correlations of IN-A with growth and carcass characteristics were higher than those of GH-A (table 23).

Since the number of animals in each breed and sex group was limited and since there did not appear to be any consistent relationships of hormones with any other variable among the breed and sex groups, the groups were pooled and simple correlations computed for the groups having common variables. The pooled correlation coefficients and groups represented in the pool for any one variable are designated by the superscripts b, c, d, and e in table 24.

When all five groups of cattle (superscript b) were pooled (Hereford bulls, Angus and Holstein steers and Holstein heifers and bulls), IN-A was highly correlated with WT-0 ($r = 0.26$, $P \leq .01$). GH-A was also significantly related to WT-0 ($r = 0.25$, $P \leq .05$). Trenkle and Irwin (1970) reported low but negative correlations for both weaning and yearling weights with plasma GH and insulin at 198 days of age. However, at 393 days of age insulin was correlated with weaning weight ($r = 0.285$, $P \leq .05$) and yearling weight ($r = 0.414$, $P \leq .01$) while the relationships of GH with weaning and yearling weights were lower ($r = 0.197$, $P > .05$; $r = 0.277$, $P \leq .05$, respectively). However, the significant positive relationships of insulin at 393 days of age with weaning and yearling weights were attributed to increased insulin values which in turn were due to high levels of

grain in the diets of steers. In this study, no one variable can be implicated as a causitive factor for the positive relationship between IN-A and WT-0 since nutritional treatment was not controlled.

No explanation is apparent for the differences in correlations of GH-A with WT-0 between the pooled groups and that of the individual breed and sex groups. In all of the individual groups, GH-A was negatively correlated with WT-0 but a positive correlation was obtained when the groups were pooled. Siers and Hazel (1970) and Bidner et al. (1973) have reported negative relationships between serum GH and live weight in pigs while Dev and Lasley (1969) reported negative correlations between GH and weight of cattle. It appears that any particular trend for serum GH to be either negatively or positively correlated with WT-0 is dependent upon the homogeniety of the particular group involved. In this study and in the reported literature, negative relationships between GH and body weight are prevelant among the more homogenous groups in both cattle and pigs. As heterogeniety increased, the relationship became negative due to variation in both body weight and GH-A among the groups.

ADGT was computed only for Hereford bulls, Angus steers, Holstein heifers and Holstein steers, while ADGB included the Holstein bulls in addition to the other four breed and sex groups. Insulin was positively correlated with ADGT ($r = 0.26$, $P \leq .05$) and ADGB ($r = 0.37$, $P \leq .01$) among the pooled groups of cattle. In addition, these relationships were positive, although nonsignificant, for the individual breed and sex groups. Trenkle (1970b) reported elevated insulin levels and increased daily gain in feedlot heifers fed MGA or stilbestrol when compared to control heifers.

In both steers and heifers, insulin was positively related (nonsignificantly) to feedlot gain (Trenkle, 1970b). Hafs et al. (1971) suggested that body growth may be influenced by estrogens through the action of estrogen on the pancreas since both lambs and cattle had been reported to respond to DES with increased gain and plasma insulin. They concluded that increased growth in response to DES was probably a function of insulin secretion. In contrast, Trenkle and Irwin (1970) reported nonsignificant negative correlations between plasma insulin and feedlot gain in cattle.

ADGT (table 24) was significantly related to GH-A ($r = -.32$, $P \leq .05$) but the relationship of GH-A with ADGB was nonsignificant ($r = -.15$, $P \leq .05$). Although Trenkle and Irwin (1970) reported a positive relationship between feedlot gain and plasma GH concentration in cattle, negative relationships have been reported by Trenkle (1970b), in cattle and by Siers and Hazel (1970), Siers and Swiger (1971) and Bidner et al. (1973) in pigs. Purchas, Macmillan and Hafs (1970) reported a positive relationship for plasma GH concentration with "specific growth rate" but when total GH content of the plasma was computed and correlated with specific growth rate the relationship was negative and highly significant. In addition, they reported a low relationship between plasma and pituitary GH concentrations. However, it should not be concluded from these observations that the negative relationship of serum GH with measures of growth implies that endogenous GH is antagonistic to body growth. Indeed exogenous GH has been shown many times to increase nitrogen retention and long bone growth as well as improve feed efficiency and daily gain of pigs, cattle and sheep. If GH was being utilized or removed from the

circulation more rapidly in rapidly growing animals, then the negative relationship would merely be a function of GH turnover. Siers and Swiger (1971) noted that circulating GH decreased as size of pigs increased and suggested that since slower growing pigs generally are smaller at any particular age the negative relationship between serum GH and growth rate is not unexpected. Trenkle and Irwin (1970) found that the plasma GH concentrations of yearling cattle were within the normal range of values found in young cattle. They suggested that a more plausible explanation for the low correlation between plasma GH and growth rate might be that with maturity, target tissues become less responsive to low physiological levels of these hormones in biological fluids. The possibility also exists that GH *per se* may not be directly responsible for increasing growth but may influence secondary hormones which may be potent stimulators of body growth. In a review, Tanner (1972) suggested that somatomedin, a peptide of molecular weight about 4000, may be the "growth hormone stimulated" secondary hormone responsible for body growth. In addition, he noted that after exogenous GH treatment, somatomedin remained elevated for 24 hours. If somatomedin is the true "growth hormone", then it is possible that adequate amounts of somatomedin to promote growth are secreted by the liver when low levels of GH are present in the circulation and that GH is not the limiting substance controlling body growth rate. Hafs *et al*. (1971) did not find any evidence that plasma levels of GH or androgen limited growth rates of 65 bulls studied.

In the present study, fat thickness was measured in carcasses of the Hereford bulls, Angus steers and Holstein steers. Insulin was negatively related to FT-TH ($r = -.37$, $P \leq .01$) but FT-TH was not significantly related

to GH-A. The negative relationship of IN-A with FT-TH is surprising in view of the fact that insulin is a lipogenic hormone and is in contrast to work reported by Trenkle and Irwin (1970). In their studies, insulin measured at 18 and 198 days of age was positively and more highly related to fat thickness than insulin measured at 393 days of age. In the same study, plasma GH was negatively related to fat thickness, although the relationships were significant only when insulin was measured at 18 days of age. Turman and Andrews (1955) and Lind et al. (1968) have reported that exogenous GH decreased backfat in pigs when compared to untreated controls. Siers and Hazel (1970) reported a negative correlation between backfat of pigs and plasma GH at weaning but when GH was measured at 90 kg the relationship was positive. They suggested that since the time of most rapid fat deposition was in the latter growth stages, the correlation of backfat with GH at 90 kg was more meaningful than that at weaning. Bidner et al. (1973) reported that data from one experiment strongly suggested a negative relationship between GH and fat thickness in pigs; however, they did not observe this same relationship in a second experiment.

Neither IN-A nor GH-A were related with LMA in this study. At 393 days of age, Trenkle and Irwin (1970) reported a positive and significant relationship between GH and LMA, while insulin only tended to be related to LMA. Siers and Hazel (1970) and Bidner et al. (1973) found inconsistent and low relationships between LMA and GH at various body weights of pigs.

The only significant relationship of hormones with carcass quality (tenderness) was between Warner-Bratzler shear and GH-A ($r = -.69$, $P \leq .01$).

TABLE 24. SIMPLE CORRELATION COEFFICIENTS OF SOME SERUM HORMONE VALUES WITH VARIOUS GROWTH AND CARCASS TRAITS.

Variable code and number[a]		n	Variable code and number[a] IN-A (44)	GH-A (50)
WT-0	(34)	105[b]	0.26	0.25
ADGT	(18)	92[c]	0.26	-.32
ADGB	(19)	105[b]	0.37	-.15
C-WT	(6)	92[c]	0.38	0.55
FT-TH	(7)	52[d]	-.37	0.01
LMA	(8)	92[c]	0.04	0.01
WB Shear	(20)	92[c]	-.01	-.69
OAA	(23)	36[e]	-.26	0.05
RB-WT	(25)	52[d]	0.03	-.09
RB-LN	(26)	52[d]	0.28	-.02
RB-FT	(27)	52[d]	-.24	-.13
RB-BN	(28)	52[d]	0.31	0.29
RB-L/BN	(29)	52[d]	-.08	-.40
RB-L/FT	(30)	52[d]	0.30	0.16
RB-% LN	(31)	52[d]	0.27	0.01
RB-% FT	(32)	52[d]	-.30	-.11
RB-% BN	(33)	52[d]	0.31	0.29

[a]Units for each variable are defined in table 6.
[b]Groups included = 1, 2, 3, 4 and 5 (See table 7 for group identification); $P \leq .05 = 0.192$; $P \leq .01 = 0.251$.
[c]Groups included = 1, 2, 3 and 4; $P \leq .05 = 0.205$; $P \leq .01 = 0.267$.
[d]Groups included = 1, 2 and 4; $P \leq .05 = 0.273$; $P \leq .01 = 0.354$.
[e]Groups included = 2 and 4; $P \leq .05 = 0.320$; $P \leq .01 = 0.412$.

Insulin tended to be negatively related to overall acceptability, but was not related to W-B shear. To my knowledge, relationships of endogenous insulin with carcass quality characteristics have not been previously reported.

Weight of the 9-10-11 rib was not related to either IN-A or GH-A (table 24). IN-A was positively related with RB-LN ($r = 0.28$, $P \leq .05$) and RB-BN ($r = 0.31$, $P \leq .05$) but tended to be negatively related with RB-FT ($r = -.24$, $P > .05$). GH-A was also related to RB-BN ($r = 0.29$, $P \leq .05$) but was not significantly related to RB-LN or RB-FT. To my knowledge, no other work has been reported involving the study of the relationship of insulin to composition; however, a number of workers have correlated carcass composition with serum GH. For example, Siers and Hazel (1970) reported negative relationships for percent ham and loin with plasma GH at weaning, 45 kg and at 90 kg live weight but these relationships were not significant. Additionally, Siers and Swiger (1971) reported low, but negative correlations for pounds of lean cuts per day of age with serum GH at 71, 104 and 147 days of age in pigs. They suggested that if GH utilization rate could be measured it should be positively correlated with the percent lean cuts and percent ham and loin. Since their correlations were negative, they suggested that animals which utilized GH at the fastest rate had the lowest circulating levels. These inferences were made based on the fact that GH has a positive influence on protein deposition and a negative influence on fat deposition. Machlin (1972) reported significantly higher percentages of protein and lower percentages of fat in hams from GH treated pigs when compared to untreated controls. In contrast,

Bidner et al. (1973) reported negative correlations between serum GH and fat trim but positive relationships of serum GH with percentages of ham and loin. In a second experiment, these authors reported that the relationships for the same traits were low and inconsistent.

RB-L/BN was not significantly related to IN-A but was negatively related to GH-A ($r = -.40$, $P \leq .01$) in this study. The negative relationship appeared to be due to the high amount of bone in Holstein steers (which also had high serum GH) rather than to any differences of RB-LN. When the RB-L/FT was correlated with hormones, the relationships were significant ($P \leq .05$) and positive with insulin ($r = 0.30$) but nonsignificantly positive with GH-A. These relationships are difficult to explain in view of the lipogenic role of insulin and the lipolytic effects of GH. Thus the data in this study do not conform to the expected physiological effects of GH and insulin in lipid and protein metabolism.

Among the carcass components expressed as percentages, only RB-% BN was significantly ($P \leq .05$) correlated with serum GH ($r = 0.29$). Exogenous GH has a positive influence on long bone growth in rats. Insulin was significantly ($P \leq .05$) correlated with RB-% BN ($r = 0.31$) and RB-% FT ($r = -.30$) while RB-% LN ($r = 0.27$) approached significance. The relationships of hormones to weight of separable components are comparable and generally of the same sign as relationships for percentages of separable components.

SUMMARY

Sixteen Hereford bulls, 17 Angus steers, 40 Holstein heifers, 19 Holstein steers and 13 Holstein bulls were used to study the relationship of bovine serum growth hormone (GH) and insulin to various growth and carcass criteria. The Hereford bulls were divided into two groups based on selection for either leanness or tenderness; Holstein heifers were fed on either a high or low level of nutrition with or without MGA treatment; Holstein steers were fed either 35% or 46% DM corn silage.

Live weight (WT-0) was greatest ($P \leq .05$) among Holstein steers and bulls compared to Hereford bulls, Angus steers or Holstein heifers. Hereford bulls had the highest ($P \leq .05$) ADGT (total feeding period) while Holstein steers had the highest ($P \leq .05$) ADGB (bleeding period). Holstein steers and heifers had higher ADGB than ADGT. Angus steers had significantly ($P \leq .05$) lower ADGB than the other breed and sex groups. Selection for tenderness or leanness did not affect ADG. ADGT and ADGB were significantly ($P \leq .05$) increased among Holstein steers that were fed 35% DM corn silage compared to those fed 46% DM corn silage. High levels of nutrition or addition of MGA to the diet increased ($P \leq .05$) daily gains among Holstein heifers.

Physical separation of the 9-10-11 rib section showed that RB-LN and RB-BN were greatest for Holstein steers and RB-FT was greatest ($P \leq .05$) for Angus steers. LMA did not differ significantly but FT-TH was greatest ($P \leq .05$) among Angus steers. In general, selection for leanness or tenderness in Hereford bulls or variation in maturity levels of corn silage

fed to Holstein steers did not influence carcass composition. However, Holstein heifers fed high levels of nutrition had heavier ($P \leq .05$) round weights which was primarily due to increased fat deposition. MGA depressed ($P \leq .05$) RD-BN and Warner-Bratzler shear values among Holstein heifers. Steaks from Holstein heifers were more tender ($P \leq .05$) than steaks from the other breed and sex groups but this was probably due to the cookery method.

Neither serum insulin nor GH (radioimmunoassay) was significantly affected by time on feed among the breed and sex groups except for Holstein steers which had only 50% of the insulin at slaughter that was observed two months prior to slaughter. Holstein steers had significantly ($P \leq .05$) more serum insulin (IN-A) than any other breed and sex group. Bulls had less GH (GH-A) than steers and heifers had less GH-A than either steers or bulls. Hormone concentrations did not differ significantly between lean and tender line Hereford bulls but the tender line tended to have higher IN-A and lower GH-A than the lean line. Holstein steers fed 35% DM silage had significantly ($P \leq .05$) less GH-A but IN-A was not affected by silage maturity. The level of grain fed Holstein heifers had no significant influence on any hormone value determined but a two-fold increase in serum insulin ($P \leq .05$) was observed among MGA treated heifers.

Although the relationships were not all significant, IN-A was positively related to growth criteria among individual breed and sex groups. Among Holstein heifers, IN-A was positively ($P \leq .01$) correlated with all measures of lean and negatively related to measures of bone; however, IN-A was not significantly related to fat. GH-A was positively related to

RD-% BN ($P \leq .01$) but not to other carcass variables in Holstein heifers. When the data of Hereford bulls, Angus steers and Holstein steers were pooled, IN-A was positively related to lean and bone criteria but negatively related to fat. In contrast, GH-A was negatively related to ADG and positively related to measures of bone.

Breed, sex, size, nutrition and housing were different among the five breed and sex groups of cattle used in this study making it extremely difficult to imply that any particular factor was responsible for the differences observed in growth, quality characteristics or composition of these cattle. However, these data suggest that diets with a high proportion of grain increased fat deposition but did not affect serum hormone concentrations. In addition, corn silage with Pro-Sil as well as MGA treatment significantly increased serum insulin but had little effect on circulating GH levels. Insulin was generally more highly correlated with a greater number of growth and carcass characteristics than was GH. It would appear that if circulating hormones were to be used in a selection program for cattle, insulin would be the hormone of choice among those studied, since it is positively and significantly related to daily gains and measures of lean.

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APPENDIX

APPENDIX I. Composition of Reagents Used In Radioimmunoassays (RIA)

A. Reagents for radioiodination

1. 0.5 M sodium phosphate buffer, pH 7.5
   Monobasic (0.5 M)
   Add 69.05 g $NaH_2PO_4 \cdot H_2O$ to distilled water.
   Dissolve, dilute to 1 liter.
   Dibasic (0.5 M)
   Add 70.98 g Na_2HPO_4 to distilled water.
   Heat to dissolve, then dilute to 1 liter.
   Mix monobasic and dibasic to give pH 7.5.
   Dispense in 1 ml portions, store at -20 C.
   Store the monobasic and dibasic buffers at 4 C.

2. 0.05 M sodium phosphate buffer, pH 7.5
   Solution A
   $NaH_2PO_4 \cdot H_2O$---------- 2.78 g
   Merthiolate---------- 0.01 g
   Dilute to 100 ml with distilled water.
   Solution B
   $NaHPO_4/7\ H_2O$---------- 26.825 g
   Merthiolate---------- 0.05 g
   Dilute to 500 ml with distilled water.
   Use 16 ml Solution A, 84 ml Solution B, dilute to 400 ml with distilled water.
   Adjust pH to 7.5 with NaOH, if necessary.
   Store at 4 C.

3. Chloramine - T
   Upon receiving chloramine-T dispense into small tightly sealed vials, cover with foil, and store at -20 C.
   Dilute 30 mg chloramine-T to 10 ml with 0.05 M $NaPO_4$, pH 7.5 buffer.
   Use within 30 minutes of preparation. Discard chloramine-T remaining in vial.

4. Sodium metabisulfite, 2.5 $\mu g/\mu l$
   Dilute 25 mg $Na_2S_2O_5$ to 10 ml with 0.05 M $NaPO_4$, pH 7.5 buffer.
   Use within 30 minutes of preparation.

5. Transfer solution
   Sucrose---------- 1.6 g
   KI---------- 0.1 g
   Dilute to 10 ml with distilled water.
   Dispense in 1 ml portions, store at -20 C.

6. Rinse solution
   Sucrose---------- 0.8 g
   KI---------- 0.1 g
   Bromphenol blue---------- 0.001 g

Dilute to 10 ml with distilled water.
Dispense in 1 ml portions, store at -20 C.

B. Reagents for radioimmunoassay (GH and Insulin)

1. 0.01 M phosphate buffered saline, pH 7.0 (PBS)
   NaCl---------- 143 g
   Monobasic phosphate---------- 120 ml
   (See Appendix I. A. 1)
   Dibasic phosphate---------- 240 ml
   (See Appendix I. A. 2)
   Merthiolate---------- 1.75 g
   Dissolve in distilled water and transfer to a large container.
   Dilute to 17.5 liters with distilled water.
   Adjust pH to 7.0 with NaOH, if necessary, store at 4 C.

2. 0.05 M Disodium Ethylenediamine Tetraacetate (EDTA) - PBS, pH 7.0
   Disodium EDTA---------- 18.612 g
   Add approximately 950 ml PBS.
   Adjust pH to 7.0 with 5 N NaOH while stirring.
   Dilute to 1 liter, store at 4 C.

3. Phosphate Buffered Saline - 1% Bovine Serum Albumin (PBS-1% BSA)
   BSA (Fraction V, Sterile, 35% solution serological, NBC, Cleveland, Ohio)---------- 50 ml
   Add 1750 ml PBS.
   Mix over magnetic mixer.
   Store in 100 ml portions at 4 C or -20 C.

4. Buffer A_1
   $NaH_2PO_4 \cdot 2\ H_2O$---------- 6.2 g
   Merthiolate---------- 0.25 g
   BSA---------- 14.6 ml
   (See Appendix I. B. 3)
   Add 950 ml distilled water.
   Adjust pH to 7.5 with 5 N NaOH.
   Dilute to 1 liter, store at 4 C.

5. Buffer B_1
   NaCl---------- 9.0 g
   Dissolve with 1 liter Buffer A_1.
   Store at 4 C.

6. Hormone Standards (GH and Insulin)
   PBS-1% BSA is used for GH and Buffer B_1 is used for insulin; hereafter they will be referred to as buffers.
   Rinse a small screw cap vial with buffer, dry.
   Weigh 200-500 µg hormone on a Cahn Electrobalance and transfer to the screw cap vial.

Add 0.85% saline to 1 mg/ml. (Make saline slightly basic, pH 8.5, for GH and slightly acidic, pH 5.0, for insulin).
Make stock hormones to 500 ng/ml with buffer.
Add buffer to 100 ml volumetric flasks.
Using Hamilton microliter syringes, add appropriate volumes of the stock solutions to volumetric flasks to obtain the following concentrations:
GH - 0.2, 0.6, 1.0, 1.6, 2.0, 3.0, 4.0, 6.0, 8.0 and 10.0 ng/ml.
Insulin - 0.4, 0.6, 0.8, 1.0, 2.0, 3.0, 4.0, 5.0, 6.0, 8.0, 10.0, 12.0, 14.0, 16.0, 18.0, 20.0, 30.0, 40.0 and 50.0 ng/ml.
Add buffer to final volume in each volumetric flask.
Dispense each standard in quantities suitable for one assay (3 ml/tube for GH; 2 ml/tube for insulin).
Freeze at -20 °C and store.
Thaw at room temperature or rapidly in a 38 °C water bath.

7. 1:400 Normal Guinea Pig Serum (NGPS)
Obtain blood from guinea pigs that have not been used to develop antibodies.
Allow blood to clot, recover serum and store the serum in convenient quantities at -20 C.
Add 2.5 ml of guinea pig serum to a 1 liter volumetric flask, dilute to 1 liter with 0.05 M PBS-EDTA, pH 7.0 (See Appendix I. B. 2).
Divide into 100 ml portions and store at-20 C.

8. Guinea Pig Anti-bovine GH (GPABGH) and Guinea Pig Anti-bovine Insulin (GPABI); hereafter referred to as antibody I.
Dilute the antisera to 1:400 with 0.05 M PBS-EDTA, pH 7.0.
Dispense in small quantities, store at -20 C.
On day of use, dilute the 1:400 antisera to the required concentration using 1:400 NGPS as diluent.

9. Anti-gamma Globulin
Use sheep anti-guinea pig gamma globulin (SAGPGG) obtained from sheep injected with guinea pig gamma globulin.
Dilute antisera to required concentration with 0.05 M PBS-EDTA, pH 7.0.
Store at 4°C or at -20 C.

C. Production of Antibodies

1. Sheep Anti Guinea Pig Gamma Globulin (SAGPGG)
Dissolve 50 mg guinea pig gamma globulin (Pentex, Kankakee, Illinois, Fraction II) in 5 ml .85% sterile saline.
Emulsify in 5 ml Freund's complete adjuvant by continuous flux through an 18 gauge needle. (Considered emulsified if a droplet retains a bead form when dropped on a water surface).

The antigen was then injected subcutaneously in 6-8 sites on the animals side.
Repeat injections every two weeks substituting Freund's incomplete for the second and subsequent injections.
Antisera was collected approximately 6 weeks after the initial injection by juglar vein puncture. (Approximately 600 ml blood from a 70 kg sheep).

2. Guinea Pig Anti-bovine Growth Hormone (GPABGH)
Two mg bovine GH (NIH-GH-B12) was dissolved in 0.5 ml saline and emulsified with Freund's complete adjuvant as described above.
Subsequent injections of 0.5 mg emulsified in Freund's incomplete adjuvant were made at two week intervals (maximum of 7 injections)
Blood was collected by heart puncture under either anaesthesia using a 10 ml syringe and a 1.5 inch, 18 gauge needle. Serum was recovered by centrifugation (ca 15,000 g for 30 min) after coagulation.
Antisera frozen at -20 C.

D. Iodination Procedures

A microsyringe was used to transfer 25 µl of 0.5 M phosphate buffer (pH 7.5) to a 1 ml glass vial.
5 µg NIH-GH-B12 (1 µg/ul of 0.05 M phosphate buffer, 0.85% NaCl, pH 8.5) was added.
One mCi of a solution of $Na^{125}I$ in NaOH (50 mCi/ml, Iso-Serve Division of Cambridge Nuclear Corp., Cambridge, Massachusetts) was added by microsyringe and contents gently shaken.
After adding 75 µg chloramine-T (Eastman Organic Chemicals, Rochester, New York) the vial was gently shaken for 2 mins.
The reaction was stopped at exactly two min. by adding 125 µg sodium metabisulfite. This reduces excess chloramine-T and converts residual iodine to iodate.
After thorough mixing, 25 µl of 2.5% BSA in 0.01 M phosphate buffered saline, pH 7.0, was added.
A 1 x 12 cm glass column packed with Bio Gel P-60, 50-100 mesh (Bio Rad Labs, Richmond, California) was equilibrated previously by passing 0.05 M sodium phosphate buffer, pH 7.5, through the column and then 2 ml PBS-2.5% BSA were added and eluted with buffer to reduce non-specific binding of the protein hormone to the column.
100 µl of Transfer Solution (Appendix I. A. 5) were added to the iodinated GH and the contents of the vial were layered beneath the buffer on the surface of the column.
70 µl of Rinse Solution (Appendix I. A. 6) were added to the vial, recovered, and layered beneath the buffer on the column.

The iodinated GH was eluted from the column under gravity with 0.05 M sodium phosphate buffer and 15 1 ml aliquots were collected in 12 x 75 mm disposable culture tubes containing 1 ml of PBS-2% BSA.

The elution profile was determined by quantifying the radioactivity of 10 µl portions from each of the 15 tubes.

In the elution curve the first peak represented the iodinated hormone and the second represented free ^{125}I. The peak ^{125}I-GH tube was used in the assay for GH.

GH iodinated for more than 10 days was passed through a 1.2 x 20 cm Sephadex G 100 column, (Pharmacia Fine Chemicals Inc., New Market, New Jersey) to reduce the content of radiation damaged hormone. Elution procedures were the same as above.

The first peak appeared to represent damaged GH as indicated by the fact that when an equal number of cpm from peaks one and two were incubated with anti-GH, more than twice as much activity from peak two was bound. Peak 3 appeared to represent free ^{125}I.

E. Radioimmunoassay for Growth Hormone

On day zero PBS-1% BSA (Appendix I. B. 3) and the standards or serum to be assayed were added to 12 x 75 mm disposable culture tubes to a total volume of 500 µl.

Four complete sets of NIH-GH-B 12 at concentrations of 0.1, 0.3, 0.5, 0.8, 1.0, 1.5, 2.0, 3.0, 4.0 and 5.0 ng/tube were included with each assay.

The assay time was started when 200 µl of GPABGH(1:3200) were added to all tubes except the total count tubes (those with only 100 µl ^{125}I-GH, 30,000 cpm/100 µl) and shaken gently.

After a 24 hr incubation at 4°C, 100 µl of ^{125}I-GH in PBS-1% BSA were added (about 30,000 cpm/100 µl) and incubated for 24 hr at 4 C.

On day 2, 200 µl of SAGPGG at an appropriate dilution were added, tubes were shaken and incubated at 4 C for 72 hours.

After incubation, 3 ml PBS was added to each tube and centrifuged for 30 mins. at 2500 g. The tubes were decanted and left in an inverted position for 30 mins. before being wiped dry and counted.

Methods of calculating results was identical to those for the insulin RIA.

APPENDIX II. Raw Data

A.1. Codes used to identify each animal within each variable.

a Identification of variable number (appendix II. A. 2. and table 6).

b 1 = Hereford; 2 = Angus; 3 = Holstein

c 1 = bull; 2 = heifer; 3 = steer

d 1 = ration fed Hereford bulls
2 = ration fed Angus steers
3 = corn silage harvested at 35% DM plus Pro-Sil
4 = corn silage harvested at 46% DM plus Pro-Sil
5 = ration fed Holstein bulls
6 = high nutrition plus MGA
7 = low nutrition plus MGA
8 = high nutrition without MGA
9 = low nutrition without MGA

e 1 = selected for tenderness
2 = selected for leanness

APPENDIX II. Raw Data

A.2. Identification of Variable Number[a].

Variable number	Variable code	Decimal places	Variable number	Variable code	Decimal places
1	BR[b]	0	29	RB-L/BN	2
2	SEX[c]	0	30	RB-L/FT	2
3	RTN[d]	0	31	RB-% LN	2
4	T-L[e]	0	32	RB-% FT	2
5	AN-NO	0	33	RB-% BN	2
6	C-WT	0	34	WT-0	0
7	FT-TH	2	35	WT-1	0
8	LMA	2	36	WT-2	0
9	RD-WT	2	37	WT-3	0
10	RD-LN	2	38	WT-4	0
11	RD-FT	2	39	AGE-0	0
12	RD-BN	2	40	AGE-1	0
13	RD-L/BN	2	41	AGE-2	0
14	RD-L/FT	2	42	AGE-3	0
15	RD-% LN	2	43	AGE-4	0
16	RD-% FT	2	44	IN-A	1
17	RD-% BN	2	45	IN-0	1
18	ADGT	2	46	IN-1	1
19	ADGB	2	47	IN-2	1
20	WB Shear	2	48	IN-3	1
21	AT	2	49	IN-4	1
22	JUI	2	50	GH-A	1
23	OAA	2	51	GH-0	1
24	TEND	2	52	GH-1	1
25	RB-WT	2	53	GH-2	1
26	RB-LN	2	54	GH-3	1
27	RB-FT	2	55	GH-4	1
28	RB-BN	2			

[a,b,c,d,e]Codes identifying each group are given in Appendix II. A.1.

APPENDIX II. RAW DATA

VARIABLE NUMBER[a]

1	2	3	4	5	6	7	8	18	19	20	21	22	23	24	
1	1	1	1	0005	460	23	1045	277	325	0723	2150			560	1
1	1	1	1	0008	506	20	1095	271	138	0492	1650			780	1
1	1	1	1	0009	498	16	1050	257	206	0765	1950			610	1
1	1	1	1	0014	497	29	1155	241	156	0790	1600			650	1
1	1	1	1	0015	537	28	0990	284	112	0506	1700			680	1
1	1	1	1	0021	403	17	0895	228	206	0680	1750			510	1
1	1	1	1	0022	495	15	0990	251	138	0706	1850			640	1
1	1	1	1	0023	441	20	1005	245	200	0650	1750			510	1
1	1	1	1	0026	437	19	0955	249	206	0664	1700			560	1
1	1	1	2	0051	589	30	1285	273	156	0678	2300			590	1
1	1	1	2	0053	405	18	1025	231	262	0832	1800			580	1
1	1	1	2	0056	495	29	1100	253	106	0663	1650			560	1
1	1	1	2	0057	505	31	1055	259	031	0900	1750			580	1
1	1	1	2	0059	584	28	1155	273	156	0646	1600			520	1
1	1	1	2	0061	494	30	1005	237	188	0649	1600			760	1
1	1	1	2	0062	486	15	1195	221	200	0664	1650			780	1
2	3	2		0112	510	75	0875	186	071	0760	1250	638	694		1
2	3	2		0124	512	75	0786	106	035	0553	1500	662	656		1
2	3	2		0141	482	50	0900	095	041	0547	1600	719	738		1
2	3	2		0154	505	60	0894	108	000	0554	1537	569	631		1
2	3	2		0160	506	70	0905	112	027	0527	1650	606	600		1
2	3	2		0166	510	50	0955	119	082	0729	1625	550	625		1
2	3	2		0197	551	60	1100	128	082	0502	1487	688	700		1
2	3	2		0202	543	60	1105	128	057	0728	1175	694	725		1
2	3	2		0219	572	70	1125	135	041	0800	1400	694	638		1
2	3	2		0226	533	77	1060	169	200	0469	1537	719	744		1
2	3	2		0232	419	40	0960	115	020	0666	1525	663	650		1
2	3	2		0255	536	60	0905	111	027	0638	1475	606	644		1
2	3	2		0274	565	50	1060	166	160	0626	1750	719	744		1
2	3	2		0276	516	85	1020	163	372	0466	1675	638	663		1
2	3	2		0278	563	55	1040	137	100	0704	1725	662	700		1
2	3	2		0301	491	65	0985	107	037	0690	1512	675	688		1
2	3	2		0746	522	67	1036	131	092	0738	1475	600	606		1

APPENDIX II. RAW DATA (CONTINUED)

VARIABLE NUMBER[a]

1234	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	
3155															000		1
3159															046		1
3170															211		1
3164															145		1
3165															098		1
3166															157		1
3169															243		1
3183															175		1
3174															084		1
3175															396		1
3173															105		1
3163															207		1
3179															180		1
0221	425			1074	5740	3980	0560	1190	3347	1169	3409	7620	7320	4271		1650	1
0226	445			0966	5685	3865	0665	1150	3365	8167	9811	7020	2321	3621		1640	1
0229	460			0970	6000	3790	0945	1200	2164	0163	1715	7520	0002	2223		1600	1
0238	470			1040	6080	4220	0695	1165	3626	0769	4111	4319	1619	7227		1320	1
0242	405			0834	4280	3240	1386	1000	3242	3465	0613	8620	0817	8203		1840	1
0251	465			1057	6850	4195	0520	1120	3758	0771	7108	8919	1424	2238		1470	1
0263	440			1027	5490	3730	0737	0980	3815	0667	9413	4217	8521	1213		1480	1
0264	345			0849	5040	3520	0512	0987	3576	8860	8410	1961	9582	1622		2400	1
0266	395			0883	5470	3690	0650	1160	3415	6867	4611	8821	2125	2291		1610	1
0278	440			1028	5640	3855	0695	1080	3575	5568	2912	3119	1319	6205		1640	1
0204	440			1110	5890	4030	0700	1140	3545	7668	4211	8819	3519	4164		2210	1
0214	500			0990	5930	3785	1063	1072	3533	5638	3179	2180	8318	3196		1400	1
0217	435			0909	5360	3460	0700	1140	3044	9464	5513	0621	2711	4610		1990	1
0223	495			1031	6240	4080	0970	1200	3404	2165	3815	5419	2322	4167		1280	1
0241	465			0932	5780	3740	0930	1090	3434	0264	7016	0918	8618	2192		1520	1
0254	440			1041	5595	3605	0920	1055	3423	9264	4316	4418	8613	6136		1050	1
0255	390			0892	4930	3210	0690	0980	3284	6565	1114	0019	8824	5262		1080	1
0262	410			0926	5110	3320	0640	1080	3075	1964	9712	5221	1423	0240		1380	1
0279	480			1394	6150	3980	1075	1083	3673	7064	7217	4817	6125	6253		1770	1
0225	400			0940	5270	3450	0600	1180	3225	7365	4611	3822	3922	8261		1680	1

APPENDIX II. RAW DATA (CONTINUED)

VARIABLE NUMBER[a]

1234	5	6 7	8	9	10	11	12	13	14	15	16	17	18	19	20	
326	0202	510	0965	6300	4315	0920	1060	407	469	6849	1460	1682	281	271	1430	1
326	0219	450	1070	5830	3830	0920	1080	355	416	6569	1578	1852	213	229	1410	1
326	0230	535	1250	6240	4080	0940	1180	346	434	6538	1506	1891	265	274	1390	1
326	0237	465	1105	5500	3610	0770	1090	331	469	6564	1400	1982	229	283	1460	1
326	0240	595	1314	6690	4360	1160	1120	389	376	6517	1734	1674	264	310	1640	1
326	0253	485	0852	5665	3530	1085	1035	341	325	6231	1915	1827	241	237	1300	1
326	0256	515	1065	6350	4280	0925	1137	376	463	6740	1457	1790	238	265	1490	1
326	0260	450	0935	5310	3600	0700	1000	360	514	6780	1318	1883	239	240	1360	1
326	0265	480	1314	5920	4100	0690	1120	366	594	6926	1166	1892	268	315	1250	1
326	0282	434	1100	5110	3460	0620	1050	330	558	6771	1213	2055	307	310	2020	1
327	0294	450	0870	5705	4015	0645	1065	277	622	7038	1131	1867	204	235	1500	1
327	0305	430	1020	5525	3910	0610	0995	293	641	7077	1104	1801	214	203	1420	1
327	0312	470	0968	5800	4095	0815	1080	379	602	7060	1405	1862	213	197	1700	1
327	0318	440	0918	5515	3855	0640	1050	367	602	6990	1160	1904	182	190	1490	1
327	0322	430	0919	5320	3730	0610	0960	389	611	7011	1147	1804	198	204	1700	1
327	0344	375	0810	4800	3240	0585	0985	229	554	6750	1219	2052	183	198	1590	1
327	0357	400	0919	5320	3720	0575	1010	368	647	6929	1081	1898	163	164	1060	1
327	0364	330	0700	4250	2815	0560	0855	229	503	6624	1318	2012	201	183	1290	1
327	0368	405	0915	5100	3415	0600	1060	222	569	6696	1176	2078	193	198	1140	1
327	0369	330	0745	4520	3040	0450	1015	300	574	6726	0906	2246	234	249	1370	1

APPENDIX II. RAW DATA (CONTINUED)

VARIABLE NUMBER[a]

1	2	3	4	5	6	7	8	18	19	20	21	22	23	
3	3	3	1	0346	601	12	1001	221	342	0994	1625	600	569	1
3	3	3	2	0618	627	13	0932	210	319	0584	1800	538	606	1
3	3	3	3	0864	630	13	0870	269	411	0876	1575	525	538	1
3	3	3	3	0916	576	13	0841	227	481	0888	1900	569	575	1
3	3	3	3	0927	646	18	1082	229	272	0618	1525	575	619	1
3	3	3	3	0928	576	01	0886	224	292	0776	1800	625	656	1
3	3	3	3	0953	560	11	0932	234	453	0918	1875	481	531	1
3	3	3	3	0964	576	09	0946	208	319	0702	1550	575	575	1
3	3	3	3	1140	646	13	1013	223	375	0626	1900	581	562	1
3	3	3	3	1289	573	17	0847	250	333	0647	1725	619	644	1
3	3	4	1	0349	596	10	1003	202	319	0650	1675	594	612	1
3	3	4	1	0665	591	09	0930	233	264	0800	1875	575	594	1
3	3	4	1	0676	601	12	1340	200	231	0654	1175	581	612	1
3	3	4	1	0808	555	13	0822	234	369	0597	1250	712	731	1
3	3	4	1	0915	534	12	0857	171	133	0542	1575	631	650	1
3	3	4	1	0946	602	08	1022	194	278	0490	1375	606	638	1
3	3	4	1	0991	617	15	0980	250	328	0696	2025	744	731	1
3	3	4	1	1101	556	09	0865	212	292	0620	1400	744	731	1
3	3	4	1	1304	586	08	1083	170	139	0571	2200	725	712	1

APPENDIX II. RAW DATA (CONTINUED)

VARIABLE NUMBER[a]

1	2	3	4	5	25	26	27	28	29	30	31	32	33	34	35	39	40	
1	1	1	1	0005	806	445	244	101	439	182	5519	3033	1257	0802	0750	431	415	2
1	1	1	1	0008	902	513	255	119	432	201	5689	2826	1315	0842	0820	425	409	2
1	1	1	1	0009	894	480	280	123	390	172	5372	3131	1377	0858	0825	425	409	2
1	1	1	1	0014	978	513	308	119	430	166	5242	3150	1220	0845	0820	421	405	2
1	1	1	1	0015	884	502	258	114	429	194	5676	2920	1293	0908	0890	418	402	2
1	1	1	1	0021	635	363	180	080	459	205	5810	2841	1266	0698	0665	392	376	2
1	1	1	1	0022	362	488	256	103	475	191	5664	2964	1193	0832	0810	389	373	2
1	1	1	1	0023	792	431	237	103	417	182	5441	2989	1304	0752	0720	388	372	2
1	1	1	1	0026	640	378	166	074	511	227	5908	2598	1156	0748	0715	382	366	2
1	1	1	2	0051	997	531	323	130	409	164	5327	3242	1303	0970	0945	437	421	2
1	1	1	2	0053	733	389	222	115	339	175	5306	3024	1564	0720	0678	430	414	2
1	1	1	2	0056	811	435	249	108	404	175	5361	3066	1328	0862	0845	422	406	2
1	1	1	2	0057	806	406	272	118	343	149	5038	3371	1468	0825	0820	419	403	2
1	1	1	2	0059	853	494	230	119	416	215	5788	2696	1392	0965	0940	414	398	2
1	1	1	2	0061	900	454	301	132	343	151	5043	3345	1471	0855	0825	410	394	2
1	1	1	2	0062	784	442	198	124	356	223	5639	2525	1584	0832	0800	408	392	2
2	3	2		0112	820	373	306	126	297	122	4548	3726	1530	0810	0775			2
2	3	2		0124	821	325	370	109	297	088	3953	4508	1330	0768	0751			2
2	3	2		0141	745	360	253	116	310	142	4835	3398	1557	0755	0735			2
2	3	2		0154	852	367	365	104	354	101	4306	4277	1217	0750	0760			2
2	3	2		0160	864	361	380	107	337	095	4181	4393	1242	0763	0750			2
2	3	2		0166	833	375	320	121	311	117	4498	3845	1448	0800	0760			2
2	3	2		0197	860	416	426	102	409	098	4335	4431	1059	0830	0790			2
2	3	2		0202	918	429	355	120	358	121	4676	3869	1306	0838	0810			2
2	3	2		0219	971	417	428	109	382	097	4296	4411	1125	0875	0855			2
2	3	2		0226	906	345	428	112	308	081	3809	4720	1235	0925	0875			2
2	3	2		0232	719	369	209	126	292	176	5132	2909	1450	0675	0670			2
2	3	2		0255	876	367	389	103	355	094	4195	4457	1182	0798	0785			2
2	3	2		0274	904	383	406	098	391	094	4231	4489	1082	0915	0875			2
2	3	2		0276	880	352	332	119	296	090	3996	4457	1351	0878	0785			2
2	3	2		0278	915	434	356	110	395	122	4740	3890	1199	0850	0825			2
2	3	2		0301	828	382	332	099	384	115	4611	4010	1200	0768	0750			2
2	3	2		0746	839	413	290	114	363	138	4917	3567	1328	0825	0780			2

APPENDIX II. RAW DATA (CONTINUED)

VARIABLE NUMBER[a]

1	2	3	4	5	34	35	36	37	38	39	40	41	42	43	
3	1	5		2155	1217	1160	1224			673	645	617			?
3	1	5		2158	1281	1275	1255			666	638	610			?
3	1	5		2170	1339	1239	1221			663	635	607			?
3	1	5		2164	1243	1187	1162			576	548	520			?
3	1	5		2165	1238	1216	1183			588	560	532			?
3	1	5		2166	1143	1077	1055			624	596	568			?
3	1	5		2169	1256	1187	1120			599	571	543			?
3	1	5		2183	1348	1317	1250			636	608	580			?
3	1	5		2174	0917	0900	0870			533	505	477			?
3	1	5		2175	1032	0986	0810			521	493	465			?
3	1	5		2173	1199	1154	1140			566	538	510			?
3	1	5		2163	1001	0972	0885			487	459	431			?
3	1	5		2179	0901	0906	0800			461	433	405			?
3	2	0		0221	0832	0729	0645	0557	0526	416	388	358	331	303	?
3	2	0		0226	0848	0732	0674	0596	0523	418	379	349	322	294	?
3	2	0		0229	0850	0800	0710	0620	0575	405	375	345	318	290	?
3	2	0		0238	0861	0768	0698	0622	0580	422	383	353	326	298	?
3	2	0		0242	0785	0727	0665	0587	0552	409	379	349	322	294	?
3	2	0		0251	0902	0775	0742	0676	0609	424	380	361	329	301	?
3	2	0		0263	0827	0780	0718	0663	0605	351	326	298	270	247	?
3	2	0		0264	0720	0620	0558	0500	0461	361	325	297	269	246	?
3	2	0		0266	0795	0694	0625	0551	0455	336	297	269	246	219	?
3	2	0		0278	0815	0860	0670	0628	0557	457	426	382	363	331	?
3	2	0		0204	0795	0743	0711	0637	0613	341	322	294	263	230	?
3	2	0		0214	0890	0830	0785	0725	0653	435	399	371	335	314	?
3	2	0		0217	0715	0738	0676	0635	0586	385	353	326	298	267	?
3	2	0		0223	0860	0840	0792	0752	0681	341	326	298	267	234	?
3	2	0		0241	0830	0790	0705	0641	0603	379	349	322	294	263	?
3	2	0		0254	0840			0715	0667	502	467	440	412	375	?
3	2	0		0255	0742	0683	0598	0543	0467	301	278	255	228	196	?
3	2	0		0262	0740	0710	0656	0581	0510	310	292	264	241	214	?
3	2	0		0279	0888	0767	0689	0625	0564	376	327	299	271	248	?
3	2	0		0285	0752	0685	0638	0551	0515	295	272	249	222	190	?

APPENDIX II. RAW DATA (CONTINUED)

VARIABLE NUMBER[a]

1	2	3	4	5	34	35	36	37	38	39	40	41	42	43	
3	2	6	0	202	937	845	795	704	647	344	329	301	270	237	?
3	2	6	0	219	807	770	692	628	573	359	343	316	288	257	?
3	2	6	0	230	951	928	815	746	672	364	348	321	293	262	?
3	2	6	0	237	867	815	725	642	578	363	347	320	292	261	?
3	2	6	0	240	1065	970	880	789	708	408	378	348	321	293	?
3	2	6	0	253	855	823	733	664	609	349	324	296	268	245	?
3	2	6	0	256	925	900	814	723	649	350	325	297	269	246	?
3	2	6	0	260	832	748	700	631	570	403	373	354	322	294	?
3	2	6	0	265	845	789	737	638	543	319	301	273	250	223	?
3	2	6	0	282	818	706	604	526	437	279	247	220	188	156	?
3	2	7	0	294	906	849	765	693	636	394	363	335	298	279	?
3	2	7	0	305	842	782	717	656	609	387	356	328	291	272	?
3	2	7	0	312	905	827	754	712	659	418	385	358	330	293	?
3	2	7	0	318	855	785	734	681	637	399	368	340	303	284	?
3	2	7	0	322	845	782	707	652	590	418	385	358	333	293	?
3	2	7	0	344	755			655	549	390	355	326	293	256	?
3	2	7	0	357	765			657	545	389	354	325	292	255	?
3	2	7	0	364	685	605	540	487	440	329	294	257	229	195	?
3	2	7	0	368	795			659	530	383	348	319	286	249	?
3	2	7	0	369	685	600	520	445	358	292	249	221	187	160	?

APPENDIX II. RAW DATA (CONTINUED)

VARIABLE NUMBER[a]

1	2	3	4	5	25	26	27	28	29	30	31	32	33	34	35	
3	3	3		0346	859	498	163	184	271	306	5799	1898	2147	1123	1000	2
3	3	3		0618	856	498	190	155	321	262	5819	2217	1812	1120	1005	2
3	3	3		0864	837	455	203	167	272	224	5434	2425	2001	1228	1080	2
3	3	3		0916	830	486	164	163	298	296	5858	1981	1960	1118	0945	2
3	3	3		0927	854	513	259	167	307	199	5382	2708	1748	1173	1075	2
3	3	3		0928	757	441	172	131	337	256	5831	2271	1735	1060	0955	2
3	3	3		0953	753	430	184	128	336	234	5708	2446	1700	1078	0915	2
3	3	3		0964	859	526	156	166	317	337	6122	1811	1929	1090	0975	2
3	3	3		1140	871	464	244	151	307	190	5325	2800	1730	1195	1060	2
3	3	3		1289	887	449	191	145	310	248	5710	2302	1848	1090	0970	2
3	3	4		0349	835	481	180	160	301	267	5761	2151	1913	1100	0985	2
3	3	4		0665	780	449	175	145	310	257	5755	2241	1863	1130	1035	2
3	3	4		0676	926	585	170	156	375	344	6319	1837	1680	1098	1015	2
3	3	4		0808	770	441	165	150	294	267	5733	2144	1949	1083	0950	2
3	3	4		0915	744	405	183	142	285	221	5441	2464	1907	0973	0925	2
3	3	4		0946	875	487	221	152	320	220	5573	2526	1732	1145	1045	2
3	3	4		0991	826	501	153	157	319	327	6068	1856	1899	1183	1065	2
3	3	4		1101	780	480	151	138	348	318	6155	1932	1771	1080	0975	2
3	3	4		1304	844	472	221	139	342	214	5600	2624	1642	1045	0995	2

APPENDIX II. RAW DATA (CONTINUED)

VARIABLE NUMBER[a]

1	2	3	4	5	44	45	46	47	48	49	50	51	52	53	54	
1	1	1	1	0005	0822	1039	0605				172	106	239			3
1	1	1	1	0008	0436	0314	0557				272	338	207			3
1	1	1	1	0009	0388	0477	0298				158	082	234			3
1	1	1	1	0014	0597	0506	0687				118	163	074			3
1	1	1	1	0015	0372	0279	0465				141	076	206			3
1	1	1	1	0021	0760	0682	0837				170	171	169			3
1	1	1	1	0022	0774	0357	1190				160	201	118			3
1	1	1	1	0023	0680	0641	0719				232	078	385			3
1	1	1	1	0026	0380	0344	0416				188	264	111			3
1	1	1	2	0051	0568	0522	0614				125	082	168			3
1	1	1	2	0053	0202	0188	0216				377	461	293			3
1	1	1	2	0056	0344	0197	0492				104	079	129			3
1	1	1	2	0057	0564	0806	0321				202	120	283			3
1	1	1	2	0059	0710	0663	0757				205	167	243			3
1	1	1	2	0061	0302	0313	0290				137	098	176			3
1	1	1	2	0062	0600	0187	1012				428	388	468			3
2	3	2		0112	0335	0430	0201	0261	0448		582	244	468	586	298	3
2	3	2		0124	0353	0288	0310	0329	0484		261	653	113	110	168	3
2	3	2		0141	0271	0200	0322	0203	0358		394	099	226	166	172	3
2	3	2		0154	0334	0206	0367	0531	0230		516	829	475	238	523	3
2	3	2		0160	0512	0175	0380	0482	1010		641	098	690	267	175	3
2	3	2		0166	0248	0309	0257	0200	0226		300	230	286	426	256	3
2	3	2		0197	0849	0437	0582	0900	1477		223	132	181	273	306	3
2	3	2		0202	0388	0142	0292	0224	0331		127	129	161	120	099	3
2	3	2		0219	0249	0325	0593	0294	0338		168	169	164	120	220	3
2	3	2		0226	0253	0221	0241	0235	0316		343	328	402	314	239	3
2	3	2		0232	0352	0318	0313	0330	0448		382	853	360	182	134	3
2	3	2		0255	0442	0554	0485	0245	0486		458	148	376	634	772	3
2	3	2		0274	1162	1838	1520	0459	0829		171	241	118	127	197	3
2	3	2		0276	0567	0687	0506	0381	0693		152	200	123	153	134	3
2	3	2		0278	0678	1128	0640	0316	0626		225	414	098	113	274	3
2	3	2		0301	0286	0376	0259	0252	0256		189	369	132	154	101	3
2	3	2		0746	0334	0483	0211	0198	0444		431	129	667	623	225	3

APPENDIX II. RAW DATA (CONTINUED)

VARIABLE NUMBER[a]

1234	5	44	45	46	47	48	49	50	51	52	53	54	55	
315	3155	0339	0302	0408	0370			153	076	087	297			3
315	3158	0224	0184	0209	0278			353	510	152	397			3
315	3170	0359	0455	0296	0326			235	276	215	215			3
315	3164	0424	0280	0553	0440			158	202	154	119			3
315	3165	0265	0246	0296	0252			103	081	095	134			3
315	3166	0511	0488	0247	0799			087	078	091	092			3
315	3169	0642	0415	0927	0585			151	127	122	204			3
315	3183	0448	0279	0603	0462			132	115	133	148			3
315	3174	0344	0304	0243	0484			098	082	150	061			3
315	3175	0325	0214	0389	0373			127	064	060	258			3
315	3173	0328	0243	0333	0408			139	064	178	176			3
315	3163	0294	0407	0206	0270			298	136	298	460			3
315	3179	0273	0387	0222	0210			104	065	097	150			3
329	0221	0221	0171	0168	0303	0195	0269	046	011	031	023	041	123	3
329	0226	0256	0216	0506	0220	0175	0162	034	018	039	023	022	069	3
329	0229	0325	0238	0514	0409	0170	0293	024	029	015	012	029	037	3
329	0238	0268	0334	0285	0295	0240	0185	034	017	026	023	083	019	3
329	0242	0218	0231	0241	0263	0192	0165	064	025	026	024	214	030	3
329	0251	0458	0391	0997	0245	0439	0219	015	014	01[illegible]	010	011	030	3
329	0263	0796	0429	0586	0460	0475	0030	013	016	005	006	013	023	3
329	0264	0616	0886	0788	0234	0387	0786	021	020	016	030	027	014	3
329	0266	0273	0275	0182	0421	0331	0156	058	022	018	034	190	026	3
329	0278	0400	0306	0767		0219	0306	039	067	014	011	082	022	3
328	0204	0209	0230	0136	0167	0186	0326	031	020	045	050	019	023	3
328	0214	0234		0199	0224	0305	0207	020	059	021	009	009	012	3
328	0217	0242	0185	0212	0152	0392	0271	042	069	011	040	058	031	3
328	0223	0579	0495	0251	0788	0685	0678	018	021	013	012	016	030	3
328	0241	0474	0407	0880	0148	0660	0275	027	025	009	054	017	029	3
328	0254	0269		0216	0308	0257	0295	017	026	026	018	007	007	3
328	0255	0211	0172	0210	0229	0219	0227	048	034	008	017	141	038	3
328	0262	0285	0171	0163	0320	0401	0370	039	058	061	007	060	010	3
328	0279	0411	0403	0767	0312	0290	0283	021	020	019	016	028	021	3
328	0285	0264	0221	0183	0367	0273?	0275?	077?	[illegible]	024?	014	265?	056	3

APPENDIX II. RAW DATA (CONTINUED)

VARIABLE NUMBER[a]

12	34	5	44	45	46	47	48	49	50	51	52	53	54	55	
326		0202	1216	0481	0802	2404	0824	1570	0170	1601	7017	0150	1201	0024	3
326		0219	0352	0430	0558		0204	0215	0280	1700	7035	0560	2300		3
326		0230	0413	0486	0742		0200	0223	0550	2801	8021	1740	3200		3
326		0237	0514	0709	0747	0196	0415	0504	0150	1001	4012	0190	1800		3
326		0240	0708	0571	1264	0598		0400	0260	4401	0017	0300	2800		3
326		0253	0674	0732	0842	0847	0278	0672	0450	1509	5011	0210	8300		3
326		0256	0988	1575	0651	1525	0642	0547	0210	1404	5016	0090	1900		3
326		0260	0441	0739	0332	0366	0269	0501	0200	1901	1032	0200	1900		3
326		0265	0895	0275	0727	0422	2004	1049	0610	2663	2012	0232	1000		3
326		0282	0542	0532	1063	0354	0562	0199	0150	1601	2021	0140	1200		3
327		0294	1059	0434	2198	1666	0335	0664	0220	2200	9023	0160	4100		3
327		0305	1079	1232	0668	1703	0548	1242	0340	3803	9010	0210	6000		3
327		0312	0933	0167	0881	1090	1238	1288	0210	2402	8013	0300	1200		3
327		0318	0852	0982	0925	1213	0783	0357	0160	1801	5019	0110	1500		3
327		0322	0973	0420	1518	0428	2198	0302	0400	4307	0		0320	1600	3
327		0344	0394		0297	0223	0695	0361	0450	3005	2041	0720	3100		3
327		0357	0330	0549	0210	0263	0244	0385	0210	1803	3027	0090	1700		3
327		0364	0232	0222	0346	0198	0141	0253	0250	8407	0100	1000	1400		3
327		0368	0419		0255	0685	0292	0442	0240	4903	2011	0060	2000		3
327		0369	0222	0200	0316	0173	0198		0260	47		0160	15		3

APPENDIX II. RAW DATA (CONTINUED)

VARIABLE NUMBER[a]

1	2	3	4	44	45	46	47	48	50	51	52	53	
3	3	3		0346	0580	0697	0644	0399	432	436	129	730	3
3	3	3		0618	0350	0387	0363	0300	559	317	633	727	3
3	3	3		0864	1683	0421	2596	2031	318	134	330	491	3
3	3	3		0916	0408	0473	0451	0301	262	360	151	276	3
3	3	3		0927	0704	0703	0636	0773	215	352	100	192	3
3	3	3		0928	0371	0292	0431	0390	344	134	138	761	3
3	3	3		0953	0987	0756	1256	0948	106	148	070	099	3
3	3	3		0964	1267	0413	2078	1311	148	166	143	135	3
3	3	3		1140	0820	0347	1273	0841	346	734	090	213	3
3	3	3		1289	0570	0272	0644	0794	223	234	134	302	3
3	3	4		0349	0528	0532	0747	0305	556	401	668	598	3
3	3	4		0665	1072	0431	0701	2084	181	294	086	162	3
3	3	4		0676	0528	0503	0418	0664	705	937	730	448	3
3	3	4		0808	1244	1037	1350	1345	191	185	213	176	3
3	3	4		0915	0370	0260	0172	0678	405	057	584	574	3
3	3	4		0946	1469	0650	1650	2106	335	202	351	453	3
3	3	4		0991	0398	0291	0510	0394	480	665	644	130	3
3	3	4		1101	0672	0305	0320	1391	308	863	262	299	3
3	3	4		1304	0573	0400	0463	0857	625	618	892	364	3