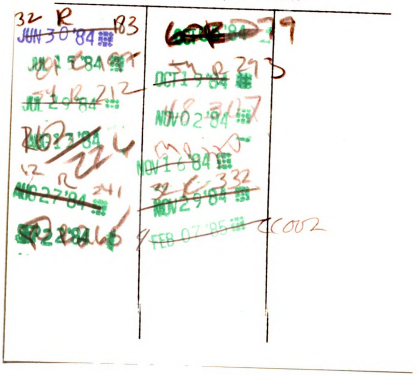

MEASUREMENT OF COMPOSITION OF GROWTH AND MUSCLE PROTEIN DEGRADATION IN CATTLE

By

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ABSTRACT

Measurement of Composition of Growth and Muscle Protein Degradation in Cattle

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Experiment 1

The effect of 2 dietary energy levels on protein and fat gain during the growing (GRO) and finishing (FIN) phase of a feedlot trail was studies with 28 Limousin steers (254 kg starting weight). Steers were randomly allotted into 4 separate treatment (TRT) groups and each TRT group was fed the following diets: TRT-1, 80% high moisture corn-corn silage (HMC-CS) during both GRO and FIN; TRT-2, HMC-CS during GRO and all corn silage (CS) during FIN; TRT-3, CS during GRO and FIN; TRT-4, CS during GRO and HMC-CS during FIN. Protein gains were greater during GRO ($P < .05$) for TRT 1 with the trend to be greater for TRT 2 than TRT 3 and 4, and during FIN and TOT for TRT 1 and 4 than TRT 2 and TRT 3. Fat gains were greater ($P < .05$) for TRT 1 and TRT 2 than

TRT 3 during GRO, during FIN ($P < .05$) for TRT 1 and 4 than TRT 2, and during TOT for TRT 1 and 4 than TRT 3. ADG were highest ($P < .01$) for TRT 1 during the GRO, TRT 1 and 4 during FIN and TRT 1 during TOT.

Experiment II

Two genetically different types of steer calves were used in a two-year study to evaluate the effect of frame size on protein and fat deposition in cattle. ADG were greater ($P < .05$) for LG cattle during GRO and TOT while daily DM intake was less for SM cattle ($P < .01$) during the GRO, FIN and TOT. Feed/gain was not different between frame types. LG cattle gained more protein/day during GRO, FIN ($P < .05$) and TOT ($P < .01$) than SM cattle. However, there was no difference in daily fat gain between frame types.

Experiment III

Eight steers of two genetic types (four each) were used to evaluate the effect of frame size on turnover rates of muscle proteins using 3-methylhistidine as an index. Analysis for creatinine (CRT) and 3-methylhistidine (3MH) was performed.

LG cattle excreted more ($P < .05$) 3MH and CRT during the trial than SM. 3MH excretion slightly decreased while CRT excretion increased with time for both treatments. 3MH and CRT excretion, expressed on a per unit of body weight basis, removed a frame effect. However, a decrease with time was seen with both CRT and 3MH/BW. The 3MH:CRT ratio tended to decline with age, while no frame effect was observed. The FBR and FSR was slightly lower for SM than for LG cattle with the FGR tending to be larger for SM than for LG.

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LITERATURE REVIEW

GROWTH OF MAJOR BODY TISSUES

From the standpoint of animal production, the growth phenomenon from the time of conception to maturity, is an important aspect of animal agriculture. The major attempt in the study of growth of the animal is to produce carcasses that have muscle combined with desirable amounts of carcass fat and minimum bone. Growth according to Fowler (1968) can be looked at in two aspects. The first is measured as an increase in weight or mass. Hammond (1952) defined growth as an increase in bodyweight until mature size is attained. Growth may also be described as an increase in mass of a body in definite intervals of time (Schlose, 1911). It is generally agreed however, that growth is more than just weight or mass gain. The second aspect involves changes in the form and composition of gain resulting from differential growth rates of the component parts of gain. At this time, no one definition of growth seems totally acceptable, but animal scientists generally prefer the description by Maynard and Loosli (1969). They defined true growth as an increase in muscles, bones and organs and should be distinguished from any increase resulting from fat deposition in adipose tissue. According

to their definition any increase in water, protein or ash content of tissue would constitute growth. In beef cattle we are primarily concerned with the growth and accretion of muscle and bone plus accretion of fat, since these three tissues are the major components of the marketable carcass.

Live weight change is easily measured by expressing it as weight gain per unit of time. However, the relative growth rate of specific tissues is more difficult to measure. In the past no reliable methods for obtaining quantitative measurements of tissues in live animals were used. The most common methods for obtaining tissue growth patterns have employed serial slaughter techniques by killing random samples of animals over a range of live weights.

A normal growth curve for live weight in cattle follows the configuration shown in Figure 1. The calf at birth, if all nutritional requirements are met, will grow along a sigmoidal shaped curve showing acceleration at around puberty and deceleration as maturity is approached.

The growth of muscle, bone and fat tissue does not coincide with the whole animal growth curve. As the animal grows, besides having a weight increase, there are adjustments in rates of growth of the tissues. The order of tissue growth is synonymous in all species (Brody, 1945). Tissues of most importance toward survival of the animal are formed before birth. Thus, the order of tissue

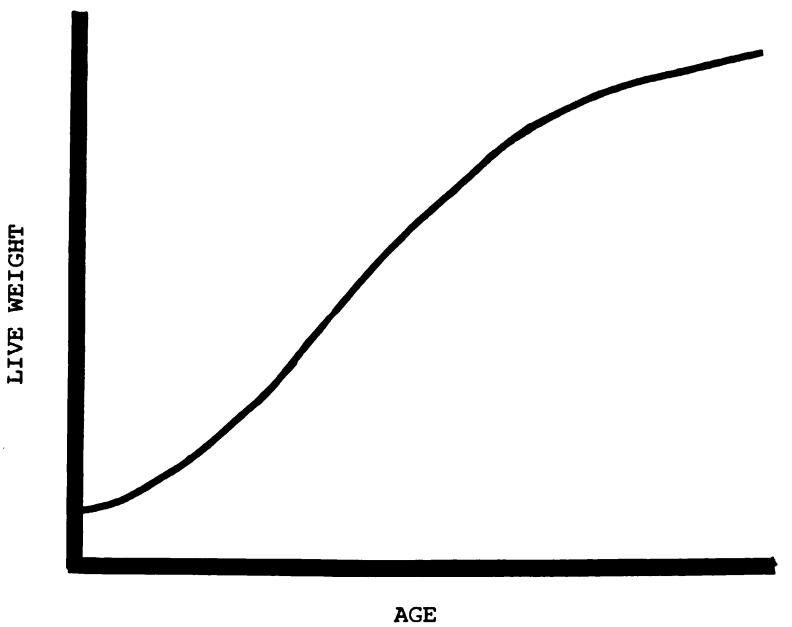

Figure 1. A typical growth curve.

growth follows a sequential trend with tissues of more physiological importance being first to develop. The order of development follows an outward trend starting with the central nervous system, progressing to bone, tendon, muscle, intermuscular fat and subcutaneous fat (Palsson and Verges, 1952a). However in the case of limited nutrient supply the tissues are affected in reverse order.

Due to the early development and growth of bone (McMeekan, 1959) and the later development and growth of muscle, the muscle to bone ratio at birth may be as low as 2:1. If growth patterns of the tissues are examined, the rates of growth postnatally do differ (Berg and Butterfield, 1968). Postnatal bone growth is at a slow steady rate (Johnson, 1974; Weiss et al., 1971) but muscle growth is relatively fast. This ultimately causes the muscle to bone ratio to increase from birth to maturity. Fat comprises a relatively small amount of the tissue in a calf at birth, but eventually it's growth rate increases so that in terms of absolute amounts, it approaches or even surpasses that of muscle tissue. Muscle comprises a high percentage at birth, rises slightly, and then begins to decrease in percentage at the fattening phase takes over.

Bone must reach a level of development during the prenatal life which allows it to function at birth; bone would therefore be called an early developing tissue. Muscle must also function at birth but not to the extent needed at

maturity therefore muscle would be a intermediate developing tissue. Fat on the other hand has minimal utility at birth and is a late developing tissue type (Berg and Butterfield, 1968; Berg et al., 1978 a,b,c,d). Research in sheep (Wallace, 1948; Palsson and Verges, 1952b) and swine (McMeekan, 1940) were similar to observations seen in cattle.

These patterns of growth have been shown by many workers, including Callow (1948) and Berg and Butterfield (1968). The problem of partitioning growth amoung body parts, still remains Huxley (1932) defines the size relationship between the whole and its parts, mathematically by the use of the allometric equation of $y=ax^b$, where y is the size of an organ or part, x is body size and b is the growth coefficient of the organ or part. This equation was found to give a reasonable quantitative description is based on the assumption that relative changes in component parts during growth are more dependent on the absolute size of the whole rather than on the time taken to reach that size.

The growth coefficient b represents the ratio of the percentage post-natal growth of y to the whole x and it therefore enables relative maturity to be expressed. The size of b is high when y represents a late maturing tissue, and low when y represents an early maturing tissue.

The allometric equation was used to compare the growth of muscle, bone and fat relative to muscle plus bone by Elsley et al. (1964) for lambs and pigs, Berg and Butterfield (1966), Mukhoty and Berg (1971) and Berg et al. (1978a) for cattle. The growth coefficient for bone in beef cattle was found to be low, less than 1.0;, for muscle intermediate, greater than 1.0;, and for fat high, from 1.5 to 2.0. The coefficients would substantiate that during post-natal growth, bone grows at a low rate, muscle at an intermediate rate and fat at a high rate.

The relative growth rates have been compared between muscle, bone and fat, however the mechanics of the individualtissue growth should also be explored in this review.

Bone develops either directly from connective tissue or is performed as cartilage which undergoes gradual ossification. The pattern and rate of ossification may vary from bone to bone depending on the strength required for that bone to function. Skull bones and consequently the volume of the cranial cavity, grow rapidly. Long bones grow in length at the epiphyseal plates. Primary growth in long bones postnatally is associated with increasing diameters due to the function of the periosteum, the connective sheath surrounding the diaphysis of the bone, laying down bone around outside of the shaft. As new bone is synthesized on the outside, degradatin and resorption occurs on the inside to keep a fairly constant thickness.

Muscle tissue development and growth is quite different from bone or fat tissue. Mesoderm, the middle third of the primary germ layer in the embryonic stage gives rise to skeletal muscle tissue. During the embryonic stage, mesoderm cells referred to as undefined intermediate myogenic precursor cells, differentiate into presumptive myoblasts (Allen et al., 1979). These mononucleated presumptive myoblasts are replicating cells within the myogenic lineage but are unable to fuse or synthesize myofibrilar proteins. The differentiation of a presumptive myoblast through quantal mitosis, forms the mononucleated myoblast. These postmitotic cells contain all the essential machinery and are capable of synthesizing all contractile proteins found in mature muscle cells. There is however no DNA synthesis or mitotic cell division, so that once this stage is attained the quantity of genetic machinery will be altered only through satelite cell incorporation (Young et al., 1978; Young et al., 1979). The mononucleated myoblasts fuse with other mononucleated myoblasts to form new mulinucleated myotubes or may even fuse into existing myotubes. The myotubes accumulate myofibrilar proteins and are called myofibers or skeletal muscle cells (Stromer et al., 1974; Allen et al., 1979; Young and Allen, 1979).

On the basis of muscle fiber numbers, Burleigh (1976) suggested a two-phase pattern for muscle growth from embryonic to adult development. During the first phase,

hyperplasia predominates where presumptive myoblasts or cells locked into being muscle cells are actively replicating for a major portion of the embryonic development. During the second phase, hypertrophy and some hyperplasia is occurring where the amount of protein per cell increases and cell replication is very slow.

Winick and Nobel (1965) added a third phase where hypertrophy is dominate and hyperplasia is negligible. During early muscle growth prior to fusion, hyperplasia is primarily responsible for the growth of muscle. After fusion, hypertrophy is the major contribution to the growth (Goldspink, 1972), with satellite cells likely increasing the DNA per muscle (Eversole, 1978; Young et al., 1978; Young et al., 1979).

Fat cells arise from mesenchymal cells which are able to synthesize DNA but not triglycerides. Although the precise pathway of histogenesis has not been clearly established, it is thought that mesenchymal cells differentiate into fibroblasts which in turn differentiate into preadipocytes. The preadipocytes form into lobular, gland like masses of epithelioid cells that develop into brown adipose tissue or directly into ordinary white adipose tissue. Palsson (1955) stated that during the early development phase, adipose tissue has the lowest priority for nutrients, the reason for it's later maturity. The

actual accretion of fat during the postnatal growth and development phase of the animal is dependent on the intake of energy over the required for maintenance.

At the cellular level, increases in adipose tissue are achieved by either adipocyte hyperplasia or hypertrophy. Bell (1909) and more recently Simon (1965) reported the preadipocyte cell loses its capacity to divide when it begins to acculuate lipid droplets. This, however, is difficult to study since lipid accululation is initiated before the cell can be recognized as an adipocyte or fat cell. It is however generally agreed that during early development both hyperplasia and hypertrophy of adipocytes does occur. Dietary restriction does affect hypertrophy of the adipocyte (Lee et al., 1973a,b) if compared at a constant age, but by looking at weight constant comparisions no effects are seen. There was however an effect of dietary restriction on adipocyte number in several adipose depots. Lee et al. (1973a,b) indicated that dietary restriction may affect adipocyte number in intramuscular depot but not in subcutaneous depots. This may suggest that adipocyte differentiations may be complete at birth in the subcutaneous depot, but not in the intramuscular location. This then reinforces the previous discussion that adipose tissue growth occurs from both hyperplasia and hypertrophy depending on the tissue depot.

BODY COMPOSITION METHODS FOR MEASURING GROWTH

A variety of techniques have been used in attempting to measure changes (Haecker, 1920; Hankins and Howe, 1946; Garrett et al., 1959; Byers, 1979) in body composition during growth and development. The most common method of determining composition of gain is to determine the body composition of the animals at two or more points in their growth curve. Determining body composition of an animal can be done by two very general methods. The animals are either killed and the carcasses analyzed or *in vivo* techniques are used.

Serial Slaughter Techniques

Serial slaughter techniques, have been the most common methods to evaluate composition and growth of animal tissue. In order to use a serial slaughter method several animals must be used. Since the animal is sacrificed, one is unable to evaluate growth of that animal at a later time in its growth and development. A second animal must be slaughtered at a later point and by difference one can measure growth. One must assume that both animals were initially of the identical body composition. Serial slaughter techniques are very expensive since many animals are needed to obtain any statistically significant differences.

Whole Animal Chemical Analysis

One of the most reliable and accurate techniques for determination of body composition is the physical separation of tissues and then chemical analysis of the tissues. Early workers like Haecker (1920), Moulton et al. (1922) and others analyzed the total empty body of cattle by summing separate analyses for different parts and tissues. This technique is not widely used today primarily because it is laborious, and involves economic loss of valuable carcass products.

Whole body analysis using physical separation of tissues has yielded a great deal of information on the body composition of cattle but cannot be a routine process. It should be an end point against which other less precise and indirect methods of predicting carcass composition can be tested.

9-10-11 Rib Section Separation

Hopper (1944) suggested the rib cut as a predictor for composition of the whole body. This was subsequently developed into a prediction formula for carcass composition by Hankins and Howe (1946). This technique, based on physical separation of the 9-10-11th rib cut into muscle, fat and bone, has had widespread use for prediction purposes in cattle experiments. The rib cut was chosen for its easy accessibility and high correlations relative to other cuts.

The relation of muscle and fat in the rib cut to the muscle and fat in the carcass were quite good with correlation coefficients of 0.85 and 0.93 respectively. However, there is considerable room for error, particularly between dissectors and levels of fatness in the separating procedures.

Specific Gravity

The discovery of the principle of density is credited to the Greek scientist Archimedes around 200 B.C. It is based on the fact that a body displaces a volume equal to its own. Estimation of density can be made from the Archimedean principle. By weighing in air and weighing in water, carcass density can be determined. For practical purposes the carcass can be considered a two-component system, fat and fat-free tissue. Fat has a density of 0.90 and muscle about 1.0. A 0.002 change in density is about equal to a 1 per cent change in carcass fatness (Kraybill et al. 1952).

The application of bovin carcass density as a predictor of carcass fatness was first reported by Kraybill et al. (1952). Earlier work has been carried out in humans (Behnke et al., 1942; Morales et al., 1945; Brozek and Keys, 1951), rats (Da Costa and Clayton, 1950), guinea pigs (Rathburn and Pace, 1945) and pigs (Brown et al., 1951). Kraybill et al., (1952) were concerned with using density as a predictor of

the fat content of the live animal. No direct measure of body fat was determined and carcass fat was estimated both from the 9-10-11th rib cut using the prediction equation of Hopper (1944), and from body water content using antipyrine method (Soberman et al., 1949).

This information, along with the relationships among the major chemical components of the bovine established by Reid et al. (1955) was used to develop a comparative slaughter feeding trial procedure in which carcass specific gravity was the key to resolve body composition (Garrett et al., 1959; Lofgreen and Otagaki, 1960; Meyer et al., 1960; Bieber et al., 1961). This procedure was useful in research where an estimate of body composition and energy storage is essential (Garrett et al., 1959; Lofgreen, 1965; Lofgreen and Garrett, 1968; Garrett and Hinman, 1969).

The use of specific gravity or density in the prediction of carcass composition was discussed by Pearson et al. (1968) and Jones et al. (1978), and results from its use were presented by Garrett (1968). According to Garrett (1968), the standard errors of estimate are too high to be very precise in predicting the composition of individual carcasses but in experiments where replication is possible, the use of specific gravity can demonstrate differences between groups in body composition.

Live Animal In Vivo Prediction

The search for accurate indicators of carcass composition on the live animal is a continuing process which has so far achieved little success. Such techniques would make it possible to follow changes in composition in experimental animals during growth without the necessity of slaughter. Breeders would find accurate live animal evaluation a great benefit when breeding and selecting superior lines of cattle.

The objective in live animal appraisal should thus be to estimate both weight and composition of the carcass or even the weight of muscle in the carcass. The percentage of live weight which is muscle tissue is the important consideration.

Potassium 40 Counting

One method that has been used to predict body composition in live animals is the whole-body counting of a naturally occurring radioactive isotope of potassium, K40. This has been used as a possible method for predicting the amount of muscle mass in the live animal (Zobrisky et al., 1959; Lohman et al., 1966; Frahm et al., 1971; Domingo et al., 1972; Clark et al., 1976). ^{40}K has a long life (3×10^9 years) and it occurs at a relatively constant proportion of total potassium. Potassium is found in the body, primarily within the cells. Muscle tissue contains a high proportion

of the total potassium of the body. Lohman and Norton (1968) found the potassium distribution in the body of cattle to be 53.4 per cent in the standard trimmed lean, 12.4 per cent in the skeleton and 16.4 per cent in the gastrointestinal tract and contents. Neutral fat should have no potassium in it but some will be present in fat depot because of blood and connective tissue. It is therefore reasoned that the amount of potassium in the animal's body might relate directly to the amount of muscle tissue and that it should be possible to measure the amount of K by measuring the ^{40}K isotope. There are rather small amounts of K in the body thus ^{40}K measurement is difficult.

Some of the technical problems in the measurement of ^{40}K are associated with equipment calibration, background interference, repeatability and accuracy of measurement. Size and shape of animals being measured may affect the readings.

There are other problems of a biological nature where we are not certain measured K in the body actually means anything. Organs and tissues may have different concentrations and thus different contribution to the total body potassium. There is also some evidence that the K concentration decreases in cattle and distribution is altered as live weight increases and the animal ages.

In spite of the problems with ^{40}K counting, the critical assessment of the usefulness will depend on how well it can predict total muscle mass. Experiments to date have shown reasonably good accuracy. It is a nondestructive technique and if accuracy can be achieved it would find a useful place in the research scheme.

Antipyrine Dilution

Soberman et al. (1949) presented a method for measuring the total water content of the body in vivo based on the dilution of anitpyrine after its intravenous injection. Kraybill et al. (1951) used this same chemical dilution technique to estimate body fat in 30 beef cattle from the measurement of total body water, with the assumption that the fat-free body contains 73.2 per cent of water. Fat and body water estimates were compared using specific gravity, 9-10-11 rib section and the antipyrine dilution technique. Kraybill et al. (1953) also looked at the application of antipyrine dilution in estimating body fat in swine showing similar agreements using specific gravity, fat separation, back fat thickness and antipyrine dilution.

Whiting et al. (1960) discussed some problems in the use of antipyrine, pointing out that although several early experiments showed very good relationships between estimates from the antipyrine technique and other body composition techniques (Kraybill et al., 1951; Kraybill et al., 1953;

Wellington et al., 1956) others have shown unpredictable occurrences of impossible values (Swanson and Neathery, 1956; Garrett et al., 1959). This indicates that there is some differences in the behaviour of various antipyrines in how they equilibrate with the body metabolic pools, for example, with rumen and tissue fluids.

Isotopic Dilution

The most common isotopes used in techniques to determine body composition are isotopes of hydrogen; deuterium and tritium. Water containing one of these two isotopes, either deuterium oxide (D_2O) or tritiated water (TOH) are commonly used for tracing water pools in animals to determine body composition.

Deuterium, a stable isotope of hydrogen of atomic mass two is found in nature mixed with hydrogen of mass one in the ratio of 15 parts deuterium to 100,000 parts hydrogen. In all natural substances in which hydrogen occurs this ratio is essentially the same (Pinson, 1952). When deuterium combines with oxygen to form deuterium oxide (D_2O) a molecule of molecular weight 20 is obtained. Deuterium oxide has a specific gravity approximately 11 per cent greater than of pure H_2O and it is this physical difference in density which has been employed in early measuring of deuterium content of water using the falling drop technique (Schloerb et al., 1951; Hytten et al., (1962). When mixed with H_2O the deuterium in D_2O readily exchanges with the hydrogen of H_2O.

Tritium is the radioactive isotope of hydrogen of mass three. It has a physical half-life of about 11 years decaying with a soft beta particle to helium. Again tritium acts very similar to H_2O with more mass than the pure H_2O. The radioactivity is its most marked physical characterisic from the standpoint of its toxicity within biological systems and also from the standpoint of its assay in the study of such systems.

Hevesy and Hofer (1934) introduced and pioneered the isotope-dilution technique by measuring and tracing the fate of heavy water in the human body. Moore (1946) later discussed total body water measurements, using deuterium oxide, and total body solids, using ^{23}Na and ^{42}K. Gest et al., (1947) and Radin (1947) presented equations for isotope-dilution analyses and discussed the validity of these procedures. The use of these procedures for animal response measurements in terms of animal growth and the differential measurement of body weight gains as pounds of fat, muscle, bone and water for body calorimetry (Reid et al., 1955; Garrett et al., 1959), and application for total body composition with farm animals have been discussed (Pearson, 1965, Reid et at., 1955).

The use of deuterium oxide as a marker for body water was most popular between 1950 and 1958, it is interesting to note that the decline in its use coincided with the rapid development of liquid scintillation techniques that greatly

facilitated the counting of tritiated water. When first introduced, (Pace et al., 1947) tritiated water (TOH) had little advantage over D_2O because both isotopes were difficult to measure. In the case of tritiated water, the marker was converted to gas and was counted in an appropriate instrument such as an electronic reed vibrometer. Thus, at first, the pattern of research with tritiated water duplicated that of deuterium oxide. First work with deuterium oxide and tritiated water centered around the characteristics of the compound as it pertains to the animal body and the total body water determination. Pinson (1952) looked at the properties of deuterium and tritium as tracers of hydrogen in water exchange studies. Edelman (1952) looked at the use of deuterium oxide equilibration in body water and Haigh and Schnieden (1956) looked at total body water in rats.

Most of the experiments using either deuterium oxide or tritiated water looked at total body water to estimate body composition (Till and Downes, 1962; Panaretto, 1963; Panaretto and Till, 1963; Panaretto, 1964). Prediction of body composition from deuterium oxide and tritium space in sheep by Searle (1970a,b). Foot and Greenhalgh (1970), Farrell and Reardon (1972), Trigg et al. (1974) and in cattle by Little and Morris (1972), Crabtree et al.(1974) all approached the ruminant water space as a single pool model. This combines the gut water with the empty body

water in to the total body water pool. The water in the digestive tract is not related to any carcass or empty body components and introduces a large and variable error into prediction of empty body components from estimates of total body water by isotope dilution. Byers (1979) developed a procedure for the separation of gut water from empty body water using a two pool system. Using tracer kinetics formulated by Shipley and Clark (1972), Byers used a deuterium oxide dilution curve and separates it into two pools. This two pool system has shown to be highly correlated to chemical analysis (r^2 = 0.965) and with specific gravity (r^2 = 0.952) by Byers, 1979.

Along with the development of the two pool system which produces a more precise and accurate prediction of body composition in live animals, methods for analysis of deuterium oxide have also improved. Using vacuum sublimation to isolate the water and deuterium oxide, and an infrared spectrophotometer to analyze the concentration of D_2O water (Byers, 1979 and Zweens et al., 1980), analyzing for deuterium has become less cumbersome. This along with the fact that deuterium oxide is not radioactive has increased interest in this isotope in determing body composition <u>in vivo</u>.

INFLUENCE OF CATTLE TYPE AND ENERGY LEVEL ON PERFORMANCE AND COMPOSITION OF GROWTH

The practice of growing out is widely utilized in the beef cattle industry to allow feeder cattle of small to medium frame size to develop more protein in their carcass. There has been, however, some controversy in the animal science community as to whether composition of gain can be modified or altered with changes in nutrition. The concept that composition of growth is constant along with composition at given weights, (Ried et al., 1968b) accepted by some, dictates that the composition of growth cannot be modified. Early research (Haecker, 1920; and Moulton et al., 1922) and then more recently (Callow, 1961; Henrickson et al., 1965; Byers, 1977, 1978, Harpster et al., 1978) documented that substantial modification in composition of growth can occur with changes in dietary energy levels. Whether or not cattle of all mature frame sizes will respond in composition of gain and efficiency to varying levels of energy is of primary concern.

The small framed or compact cattle were in great demand in the 50's and 60's while research during that time clearly indicated a much greater ability to gain for large framed

cattle (Stanley and McCall, 1945; Stonaker et al., 1952). It was also shown, however, that the feed conversion efficiencies did not necessarily coincide with the increased rates of gain in large frame cattle (Stonaker et al., 1952).

Klosterman and Parker (1976) compared Angus and Charolais when fed either corn silage or corn grain rations to a similar slaughter condition. Angus steers fed either high grain or high silage diets did not differ in carcass quality grade when fed to equal weights. However, Charolais steer fed a high silage diet graded two-thirds of a grade lower than those fed a high grain diet. Klosterman and Parker (1976) concluded that lower energy rations were of benefit more to early maturing cattle which consume more feed per unit of body weight.

Crickenberger et al. (1978) using Angus, Chianina crossbred, and Holstein steers fed either high silage or high grain rations, looked at energy level and frame size effects on performance and carcass traits. They found that high grain fed cattle, compared to high silage cattle, had higher daily carcass gains, fatter carcasses and lower cutability. Daily protein and fat gains for high grain fed cattle were greater than high silage fed cattle. Daily protein gain tended to be greater and daily fat gain less for the larger cattle types compared to average or small framed cattle.

Byers and Rompala (1979) showed similar results in that larger framed cattle on a high energy diet gained more protein per day than small framed cattle. They indicated that the "nutritional niches" for small and large size cattle where rates of protein and fat deposition allow optimal composition of growth are provided by moderate energy diets for small mature size cattle and high energy diets for small mature size cattle and high energy diets for large framed cattle.

Byers (1980) in a summary of their research on cattle feeding systems to regulate composition of gain in cattle, indicates that at least for Hereford steers, daily rates of protein growth approached maximum rates of 120g/day at about 1.0 kg daily empty body weight gain. Other recent research documenting similar responses between rate and composition of growth include studies of Woody (1978) and Garrett (1979). Both Woody (1978) and Garrett (1979) reported maximum rates of protein growth at between 0.95 and 1.00 kg daily empty body weight gain may have a very strong relationship with maximum daily protein gain and may be one of the controlling factors.

Smith et al. (1977) compared various biological types of cattle on a wide range of feeding regimes. Cattle were either classified as small type, at least five-eights British, Jersey, or Red Poll, or classified as large type,

one-half Brown Swiss, Charolais, Chianina, Gelbvieh, Limousin, Maine Anjou and large domestic dairy breeds. Five feeding regimes ranging from 2.18 Mcal ME/kg to 3.11 Mcal ME/kg were examined.

There were two or three slaughter groups per feeding regime from which regressions were developed to standardize data at a weight and composition-constant endpoint. Live weight gains were as expected from the energy density of the rations; however feed efficiency measured on a pen basis did not differ among regimes or types. Composition of gain was markedly altered by regime as was the effect of all measures of fat. Those cattle receiving the highest energy did show the most fat gain of any feeding regime in the trial.

Byers and Parker (1979) conducted a study comparing cattle of varying mature size fed differing levels of nutrition. Cattle were fed either high energy or moderate energy diets. They reported that cattle on the high plane of nutrition deposited protein at 34 to 48 per cent and fat at 76 to 93 per cent faster rates than cattle fed the moderate plane of nutrition. This documented a modification in composition of gain resulting in a 19 per cent increase in percent fat in tissue gained with the high plane of nutrition. Large size cattle gained faster and were more efficient than smaller cattle on the shelled corn, high energy diet.

Prior et al. (1977) studied the impact of dietary protein and energy on performance and carcass characteristics of empty body gain and carcass gain in 56 Hereford steers. Using a serial slaughter technique to determine body composition, slaughtering for steers per treatment at 341, 454, and 545 kg. The composition of empty body and carcass gain for a given weight was not affected by ration.

Koch et al. (1979) presented data to indicate that fat deposition was linear with time for all groups, although the rates depended on energy intake. The rates were 8.21, 2.08 and 1.29 kg/month for the high, medium and low planes of nutrition, respectively. The results suggested that steers are not programmed to synthesize protein first and then fat only with the remaining energy; rather the level of energy intake and the age determines how the ingested energy is partitioned into protein and fat synthesis. Other research would tend to agree with this suggestion (Guenther et al., 1965; Klosterman et al., 1965; Jesse et al., 1976; Perry and Beeson, 1976). Thus, the process of protein accretion and fat accretion occur simultaneously during early growth, whereas in later growth, the rate of protein accretion becomes negligible (Bergen, 1974).

It has become evident through the review of the literature that there is considerable variation in results from studies concerning the effect of energy and frame size

on composition of gain, and performance in cattle. Early research documented the increase in fat accumulation with higher energy levels (Haecker, 1920; Moulton et al., 1922), as well as more recent research (Callow, 1961; Henrickson et al., 1965; Byers et al., 1976; Byers, 1977, 1978a; and Harpster, 1978) also concluding that there is an increase in fat storage at similar carcass weights with high planes of nutrition. Jesse et al. (1976a), Guenther et al. (1965), and Reid et al. (1968) reported no effect of energy levels on carcass composition. All this has aided in confusing the nutritionist about this area of research. At least a portion of the inconsistency can likely be attributed to cattle frame sizes compared, portion of the growth curves examined, degree of differences in energy intake levels, slaughter schedule, body composition techniques and trial design.

PROTEIN TURNOVER IN THE INTACT TISSUE

The measurement of protein turnover is frequently based on the uptake or release of labeled amino acids (Waterlow, 1970). One way in which protein turnover can be studied is by injecting a single dose of labeled amino acid into the organism. Since the turnover of the free amino acids in the body is rapid, the administered labeled amino acid will enter rapidly into tissue free amino acid pool and reach the peak value within a few minutes and then fall off rapidly to negligible levels (Waterlow, 1969). It is therefore possible in theory, to measure the rate of protein synthesis and protein breakdown (turnover) by observing the proportion of the administered labeled amino acid which is incorporated into tissue (synthesis rate) or the rate of disappearance from the tissue (degradation rate). If the organism is at a steady state in regards to protein turnover, the synthesis rate is equal to the degradation rate. If the tissue is gaining or losing protein, the rates of synthesis, rates of breakdown and the change in protein content of the tissue are all interrelated. The interrelation can be expressed by the simple equation: PROTEIN ACCRETION = PROTEIN SYNTHESIS - PROTEIN DEGRADATION. If two of the three components are known then the third can be calculated.

The measurement of protein turnover in the whole animal or an intact cell is complicated by the fact that some amino acids are reutilized. Reutilization of amino acids can occur within a tissue, intracellular; or between tissues, intercellular. There are also instances when proteins are secreted by one tissue and utilized as an amino acid source by another. With the reutilization of the amino acids occurring, the consequences of using a labeled amino acid as a tracer can lead to erroneous data in the determination of protein synthesis or breakdown *in vivo*.

Haverberg (1975) reviewed some of the situations that may occur that cause errors in estimating turnover are:

A). After the labeled amino acid has been incorporated into the tissue protein; it is released and can be reincorporated into the tissue. This would lead to a lowered measure of the rate of loss of the label from the tissue protein thus underestimating the rate of breakdown.

B). The labeled amino acid can be incorporated into the protein of one tissue and released where upon it can be incorporated into protein of another tissue. In this instance, the extent of labeling of the later tissue protein would be dependent on the extent of both intracellular and intercellular recycling of the label.

C). The entering labeled amino acid can be diluted out by blood and by the intracellular fluids.

The extent of reutilization of amino acids is dependent on age, diet, hormones, and various conditions that may affect the muscle protein turnover. Individual amino acids are reutilized at different rates and extents. It has been generally assumed that the shorter the experimental half-life obtained or the faster the estimate of protein breakdown is, the closer the value is to the "true" rate of protein degradation. In determining to what extent an experimental half-life approximates the actual rate of degradation, estimates of amino acid reutilization are made and the error from the reutilization is calculated. It appears that by the use of non-reutilizable amino acids, unambiguous protein degradation rates may beobtained.

Measurement of turnover of tissue proteins in vivo has been reviewed by Neuberger and Richards (1964), Waterlow (1969), Schimke (1970), Waterlow (1970). With the relative lack of confidence in the available methodology multiple techniques have been used to look at degradation and synthesis rates of proteins.

To illustrate some of the diversity in results of protein degradation studies a comparison of data can be used. Estimates of whole body protein turnover in adult man, using isotope compartment models, vary from 100 gm per day (Wu and Snyderman, 1950) to 300 gm per day (Waterlow, 1969; Grumer et al., 1961; Kassenaar et al., 1960). There are enormous discrepancies when comparing degradation rates by pulse

dosage of animals with a labeled amino acid and by measuring the rate of loss of label from injected labeled protein. Fashakin and Hegsted (1970) found that rats given ^{14}C-amino acids, lost the label from plasma with a half-life of about 18 days, whereas, injection of rats with pre-labeled plasma proteins yielded half-lives of 3 days or less (Anker, 1960). Similarly, Swick and Ip (1974) found that the rate of decay of radioactivity in albumin was 30% slower after administration of ^{14}C guanidino-arginine than that obtained with ^{14}C-carbonate. The longer estimates are due to extensive reutilization of the labeled amino acid.

Gan and Jeffay (1967) gave rats continuous infusion of labeled lysine or tyrosine in order to demonstrate and estimate the degree of intracelluclar amino acid recycling. When the plasma levels became constant, they estimated the specific activities of each of these amino acids in the plasma and the tissues. In the liver, the free amino acid pool achieved a specific activity no greater than 50% of the systemic plasma levels, and when protein degradation was accelerated by a short period of fasting, the label in the liver free amino acid pool fell to about 10% of the plasma level. Muscle showed less extensive dilution with recycled amino acids and was not greatly affected by fasting. This extensive recycling in liver has been confirmed by Stephen and Waterlow (1966), Waterlow and Stephen (1968) and by Schimke (1970), all of whom compared the rate of loss of

labeled ^{14}C-guanidino-arginine from liver protein with loss of U-^{14}C-arginine. This method of avoiding recycling by using ^{14}C-guanidino-arginine depends on the exchange of the guanidino group of free arginine during the urea cycle. There are several drawbacks to the use of ^{14}C-guanidino-arginine.

Some labeled free arginine will obviously escape so that recycling of the ^{14}C-label is not completely abolished. There is also the fact that the use of ^{14}C-guanidino-arginine depends on the presence of the urea cycle in the liver, any change in the activity of the area cycle will alter reutilization of the label. This dependence on the urea cycle in the liver makes the use of ^{14}C-guanidino-arginine not applicable to other tissues.

In muscle and in most tissues, the reduction of recycling can be overcome by using ^{14}C-carbonate which labels primarily aspartate and glutamate (Manchester and Young, 1959). This method is based on the uptake and release of $^{14}CO_2$ by the dicarboxylic acids (Swick, 1958) and has been used by Millward (1970a, 1970b), Perry (1974) and Swick and Song (1974) to look at protein turnover in liver and muscle The reaction depends on transamination, occurring in most tissue and is therefore of more general use.

There are labeled amino acids used to estimate muscle protein degradation. Young et al., (1971) used ^{14}C-aspartic acid for the purposes of the in vivo labelng of muscle proteins. Use of this label largely overcomes the problem

of amino acid reutilization and hence allows a more accurate estimate of rate of protein degradation in muscle cells. Swick and Song (1974) indicated that labeled glutamate and alanine can be used also to evaluate rates of protein degradation, but, reutilization of their labels does occur. Tyrosine, because of low metabolism in muscle, and phenylalanine have also been used to evaluate protein turnover in muscle however all labeled amino acid do have, to some extent, the problem of reutilization.

An alternative to the single pulse method that does not provide absolute measurements of turnover of proteins, but rank them in order of magnitude, is the double-isotope technique described by Arias et al. (1969). In this procedure, two isotopic forms of an amino acid, usually ^{3}H- and ^{14}C-leucine, are used to establish two time points on the curve describing degradation of the protein. ^{14}C-leucine is first administered to an animal, then some days later ^{3}H-leucine is given to the same animal just before it is killed. Proteins with rapid turnover rates such as those which are synthesized rapidly (high ^{3}H) and degraded rapidly (low ^{14}C) will have high $^{3}H/^{14}C$ ratios. Glass and Doyle (1972) used this procedure to give rate constants of degradation however they found that the interval separating the two isotope injections must be varied depending upon the range of half-lives being examined. Pool (1971) was quick to point out however that occur due to reutilization even in this procedure.

In a situation in which turnover is in a steady state, degradation is equal to synthesis. This situation is not always occurring, so that measurement of synthesis rates are sometimes necessary. To obtain such an estimate after administration of a labeled amino acid, it is necessary to know the specific activity of the precursor pool. The rise and fall of the specific activity of the pool can then be used to correct for the radioactivity incorporated into the tussue protein. Integration of the precursor pool activity over short intervals must occur in order to obtain a valid picture of the rapidly changing labeling of the precursor pool. An alternative method would be a constant infusion of labeled amino acid, to attain a plateau level in the tissue. This method has been extensively used by Waterlow and Co-workers, (1968). From knowing the level of radioactivity of the plateau, it is possible to calculate rates of synthesis or proteins by observing the uptake of the label into protein.

It is apparent that the merits of both the pulse dose and the continuous infusion depends on the acceptance of the free amino acid pool of the tissues as the exclusive source of amino acids for charging tRNA in protien synthesis. If this is not the case or if it is only approximately so, these methods lose precision in estimating rates of synthesis.

MUSCLE PROTEIN TURNOVER IN SKELETAL MUSCLE

Hormonal Effect

The endocrine system influences muscle turnover not only by the direct actions of hormones on muscle tissue, but also indirectly, by regulating voluntary food intake, and the subsequent distribution of nutrients between the tissues of the body in the fed and fasting state. There are complex interactions between hormones both in the regulation of hormone secretion, and in the way they achieve their effect on the target tissue. Morgan and Wildenthal (1980) listed factors that are important in the regulation of protein turnover. Biological activity and availability of insulin, growth hormone, anabolic steroids, adrenal steroids and thyroid hormones are most important.

Endocrine functions in the very young are not always the same as in the post-weaning animal. The role of individual hormones in determining growth and nutrient distribution may vary at different stages of development (Turner and Munday, 1976). The hormonal control of fetal growth is poorly understood, although it is known not to be dependent on the fetal pituitary hormones. The role of hormones, both in the

adult and in the immature animal, is primarily in protein synthesis stimulation however some hormones are catabolic in nature. Bergen (1975) identified the interaction of hormones as being important at various points in protein synthesis.

Growth Hormone

Early studies of the role of growth hormone in the regulation of muscle protein turnover have been reviewed by several authors (Korner, 1967; Manchester, 1970; Young, 1970; Kostyo and Nutting, 1973; and Kostyo and Reagan, 1976). Administration of exogenous growth hormone to intact sheep has produced a decrease in plasma concentrations of amino acids (Davis et al., 1970) indicating an increased uptake of amino acid by body tissue (Korner, 1967). Diaphram muscle from hypophysectomized rats had a reduced rate of protein synthesis in vitro compared to normal muscle, whereas pretreatment with growth hormone in vivo or addition of the hormone in vitro stimulated both the transport of amino acids and the rate of protein synthesis (Waterlow et al., 1978). Goldberg et al. (1980) showed similar results in skeletal muscle. In muscles from normal rats some workers have found that incubation with growth hormone has no effect (Manchester and Young, 1959; Kostyo

and Schmidt, 1962) while others have found that it stimulates protein synthesis but not amino acid transport (Reeds et al., 1971; Turner et al., 1976).

Growth hormone has been shown to affect amino acid transport, DNA and RNA metabolism and ribosomal aspects of protein synthesis, especially in muscle (Young, 1970; Young and Pluskal, 1977). Hjalmarson et al. (1975) have examined the effect of growth hormone on protein synthesis in the perfused heart. Their data suggests that the rate of protein synthesis was lower in hearts from hypophysectomized rats than in normal or hormonetreated animals. Judging from the increased amount of ribosomal subunits in the hearts of the hypophysectomized rats, the initiation of protein synthesis was impaired. Exogenous growth hormone has been shown to increase nitrogen retention (Davis et al., 1970) and increase carcass protein in sheep (Wagner and Veenhuizen, 1978).

An understanding of the mode of action of growth hormone on muscle protein metabolism is complicated by the existence of growth-promoting polypeptides under growth hormone control. These appear to be somatomedin A and C, non-suppressible insulin-like activity and multiplication stimulating activity; exerting an insulinlike action on their target tissue (Zapf et al., 1978). Furthermore, insulin and nutritional state may modulate somatomedin generation (Yeh and Aloia, 1978; Phillips and Vassilouplou-Sellin, 1979).

Insulin

A difficulty that is faced in understanding the mode of action of insulin on tissue protein metabolism is that this hormone exerts a large number of diverse effects in cells. Studies on insulin effects upon skeletal muscle have been carried out with much variation in both technique and results (Wool and Krahl, 1959; Jefferson et al., 1974; Frayn and Maycock, 1979). But, of the hormones involved in the regulation of protein turnover in muscle, more is known about insulin than any other (Cahill, 1970; 1971, 1976; Manchester, 1976; Goldfine, 1978). Insulin regulates the uptake of glucose and also of many amino acids into muscle by stimulation of the A transport system. Insulin also regulates a number of intracellular processes, including protein metabolism. It is not known precisely how insulin is involved but a theory is that the insulin-receptor complex generates a second message at the cell surface which carries out all subsequent intracellular effects. Alternatively, it is possible that insulin itself enters the target cells (Goldfine, 1978) and that the hormone or product of insulin interacts directly with intracellular structures and protein synthetic machinery.

The stimulation of muscle protein synthesis, by insulin, observed in vitro was confirmed in vivo. Hay and Waterlow (1967) showed that in whole animals, the rate of muscle

protein synthesis was depressed in experimental diabetes. Kurihara and Wool (1968) and Sender and Garlick (1973) reported similar results in their trials. Goldstein and Reddy (1967) showed an enhanced entry of amino acids into cells but not a direct effect on the synthesis of protein. Jefferson et al. (1974) showed a stimulating effect of insulin on protein synthesis and also showed a suppression of protein breakdown. Albertse et al. (1979) presented data to indicate a dramatic decrease in protein synthesis with diabetic rats when insulin levels were depressed. Millward et al. (1976b) observed in muscle of rats that RNA activity fell to very low levels when treated with streptozotocin. Wool et al. (1968) established that insulin is involved in the regulation of peptide chain initiation. These workers demonstrated a decrease in polysomes and an increase in monomeric ribosomes in skeletal muscle of alloxan diabetic rats.

Generally insulin has been shown to increase muscle protein synthesis however, a preliminary study by Chrystie et al. (1977) indicated that exogenous insulin did not increase protein synthesis but decreased breakdown of protein in fetal lambs. This may be another of the many functions of insulin.

Anabolic Steroids

There has been relatively little investigation into the mode of action of endogenous anabolic steroids or their derivatives on growth of skeletal muscle and tissues, other than sex organs and accessory sex tissues (Young and Pluskal, 1977). Since skeletal muscle is more developed in the male than in the female, it is supposed that androgens might be responsible for myotrophic or anabolic action. Although more is known about the mechanism of action of the estrogens (Chan and O'Malley, 1978), the sex steroids appear to affect similar biochemical processes in their target tissues. However, specifically whether the androgens and estrogens bring about their myotrophic effects in similar or dissimilar ways is uncertain.

Grigsby et al. (1976) showed an increase in protein synthesis in rabbits given exogenous testosterone, but, an increased plasma insulin would indicate no direct effect by testosterone. Some effects of androgens on muscle however are known. Young (1980) reviewed androgen effects indicating that testosterone increases muscle weight and protein mass, increases nitrogen retention, binds to cytosol protein receptors and that RNA synthesis increases in muscle via an effect of nuclear chromatin. Mayer and Rosen (1977) concluded that androgens interfere with the binding of glucocorticoids to receptors in muscle cytosol.

This is supported by the known antagonistic effect of anabolic steroids on the catabolic effects of glucocorticoids on muscle protein metabolism (Young and Pluskal, 1977).

Glucocorticoids

Glucocorticoid hormones have long been known to have a general action on protein metabolism which is opposite that of insulin, decreasing the protein content of the carcass and increasing that of liver (Munro, 1964). Their overall effect is that of growth suppression and muscle wasting (Long et al., 1940). With respect to corticosteroids, they can directly inhibit DNA and protein synthesis and cell replication (Loeb, 1976; Baxter, 1978). Based on reviews (Loeb, 1976; and Baxter, 1978), glucocorticoids can inhibit growth hormone production and perhaps somatomedin production. It seems that this is not the only mechanism for their adverse effect on body protein gain. It is possible that steroids may affect uptake of substrates, such as glucose (Munck, 1971), which in turn affect growth or they may induce synthesis of inhibitory proteins that block synthesis of RNA. Young et al. (1968) showed that after injection of hydrocortisone, ribosomes isolated from rat skeletal muscle were less aggregated than those from control rats. The exact mechanism for glucocorticoid actions is not known. However it is known glucocorticoids result in reduction of muscle protein.

Thyroid Hormones

An endocrine factor that is increasingly attracting more interest in the area of muscle protein metabolism is the iodothyronines. It is known that thyroidectomized rats have decreased protein synthesis in the gastrocnemius muscle in rats (Flaim et al., 1978b) and that treatment with thyroid hormone replacement enhanced synthesis. Young and Munro (1978) observed a reduced rate of muscle protein breakdown in thyroidectomized rats, and with thyroxine replacement there was an increase in muscle protein breakdown rate. Turnover of muscle proteins is more rapid in the presence of thyroid hormone. Since a net gain of muscle protein is achieved, and since both synthesis and degradation is enhanced, protein synthesis must be enhanced more than breakdown.

Little is known about the mechanism of thyroid hormone action on protein metabolism. Thyroid hormones bind to several subcellular constituents, in particular, saturable nuclear binding sites have been demonstrated in several tissues (Sterling, 1979; Oppenheimer, 1979). The existence of T_3-binding proteins in the cytosol is firmly established but their function has not been defined (Sterling, 1979). It has been suggested that they may help maintain a readily

available intracellular pool of hormone. Oppenheimer (1979) reviewed the possible mechanisms of thyroid hormone and suggests that the T_3-nuclear receptor complex may stimulate directly or indirectly the formation of a diversity of mRNA sequences.

Nutritional Effect

Skeletal muscle has long been known to be a source of substrates for energy metabolism and of amino acids for essential protein synthesis in other tissue and organs. The studies of energy balance in fasting man (Cahill, 1970; Young and Scrimshaw, 1971) serve as good examples of the contribution of muscle protein and extent of adaptation during reduced energy intake. In the fasting obese adult, as glycogen stores become depleted, muscle protein becomes the source of glucogenic precursors from which glucose needed by the brain can be synthesized. The loss of proteins oxidized for energy clearly could not be tolerated for long. However, the brain switches from glucose as an energy source to ketones as its major fuel thus reducing the oxidation of muscle protein. A similar adaptation can be produced by malnutrition, since a malnourished child loses less nitrogen than a child who is well nourished (Kerr et al., 1973). The provision of amino acids for gluconeogenesis and subsequent adaptation which occur must result from changes in the relative rates of muscle protein synthesis and breakdown.

The changes in RNA concentration in muscle in response to various dietary treatments have been documented by many workers. Young and Alexis (1968) reported a loss of RNA from muscles in protein-deficient rats and Howarth (1972) clearly demonstrated that protein synthesis had priority over DNA synthesis when muscle growth was impaired during a protein-deficient feeding period. As dietary levels decreased from 24 per cent crude protein to 18, 12 or 6 per cent crude protein, weight gains and gastrocnemius muscle weights decreased. There was a greater decrease in DNA with RNA being intermediate to DNA and protein.

Hill et al. (1970) and Cheek and Hill (1970) showed a reduction in protein synthesis during a protein-deficient diet while DNA synthesis is primarily affected with a calorie-deficient diet. Perhaps the most detailed study is that of Spence and Hansen-Smith (1978), who investigated the effect of severe undernutrition on 13 rat muscles and showed a fall in the RNA/protein ratio along with muscle responses.

Millward et al. (1976a) presented data to indicate that during starvation in young and adult rats, synthesis rates decreased initially stopping growth. Then as synthesis rates continue to fall breakdown rates also decline. After muscle takes over as the source of amino acids, taking over from the liver and the gastro-intestinal tract, breakdown rates show a progressive increase. In the

adult rat the fractional breakdown rate of muscle, after 4 days of starvation are nearly twice the initial rate (Millward and Waterlow, 1978). Winick and Noble (1966) reported that the effect of calorie and protein restriction on cell number and/or cell size was dependent on the phase of growth. Rats subjected to early malnutrition had impeded cell growth and division. They concluded that undernutrition affects the permanent suppression of DNA synthesis more than RNA or protein synthesis and that it caused permanent growth retardation in animals when DNA accumulation is rapid.

N^{τ}-METHYLHISTIDINE AS AN INDEX FOR MUSCLE DEGRADATION

It is generally known that the skeletal musculature is a major tissue in whole body protein metabolism (Young, 1970). Skeletal muscle represents a large reserve of protein that can be made available during periods of dietary stress. Nevertheless, there is little information on the mechanisms responsible for the maintenance of protein content in skeletal muscle and the contribution of protein turnover in muscles to the overall body protein metabolism under various nutritional, hormonal and nervous conditions, and in response to stress (Millward et al., 1976; Young, 1970; Young and Munro, 1978). There has been a growing interest in recent years in the study of protein

catabolism. The techniques used in these investigations have been primarily isotopic. However, the reutilization of the radioactively-labelled tracer amino acid, arising from protein catabolism, for protein synthesis yields underestimates of protein degradation rates (Poole, 1971; Glass and Doyle, 1972). An ideal index of protein degradation would be an amino acid which is reutilized and reincorporated into proteins.

Asatoor and Armstrong (1966) were first to suggest the measurement of the rate of excretion of 3-methylhistidine could provide an estimated of the turn-over of myofibrillar degradation.

3-Methylhistidine was first identified as a normal component of human urine in 1954 (Tallan et al., 1954), while Block and Hubbard (1962) found the amino acid in the urine of rabbits. The occurrence of 3-methylhistidine as a component amino acid in skeletal-muscle myofibrillar proteins of rabbits, was first demonstrated by Johnson et al. (1967) and by Asatoor and Armstrong (1967). Elzinga et al. (1973) later reported the positions of 3-methylhistidine in the amino acid sequence of actin of rabbit skeletal muscle. Further studies have demonstrated its presence in muscle proteins of other species (Haverberg et al., 1974; Haverberg et al., 1975; Holbrook et al., 1979; Nishizawa et al., 1979). It has been shown that the proportion of histidine molecules methylated in myosin

varies from one muscle of myosin species to another, whereas the 3-methylhistidine content of actin is constant (Johnson et al., 1969; Kuehl and Adenstein, 1970). Huszar and Elzinga (1976) succeeded in isolating from both rabbit and bovine cardiac myosin a tryptic peptide that contains the non-methylated histidine.

3-Methylhistidine is found in both actin and myosin and available evidence indicates that the methyl group is attached to free histidine after the formation of histidyl-tRNA (Haverberg, 1975t; Ward and Buttery, 1978). Hardy and co-workers (1970) demonstrated that S-adenosylmethionine was an effective methyl donor for the formation of 3-methylhistidine in muscle protein in rabbit skeletal muscle homogenates. This was confirmed by Krysik et al. (1971) in studies of the methylation of chick muscle proteins in vitro. Young et al. (1970, 1972) studied binding in vitro and in vivo of various labelled amino acids to tRNA and were unable to demonstrate a His(3Me)tRNA. In conclusion, there is good evidence demonstrating that the methylation step occurs after the formation of histidyl-tRNA.

The fate of administered 3-methylhistidine has been examined by Cowgill and Freeburg (1957). They found that most (90%) of the radioactivity following administration of ^{14}C methyl-3-MeHis to rats, rabbit, and chicken appears in the urine. Paper chromatography of the urine showed that unchanged 3-methylhistidine accounted for a major

proportion of radioactivity. Young et al. (1972) examined the fate of orally or intravenously administered ^{14}C-methyl N$^{\tau}$-methylhistidine in rats and confirmed that there was a quantitative excretion of the label in the urine. Only trace levels of radioactivity appeared in the feces. This was later confirmed in humans (Long et al., 1975), in rabbits (Harris et al., 1977), in cattle (Harris and Milne, 1979; Harris and Milne, 1981) but does not seem to be true in sheep (Harris and Milne, 1977; Harris and Milne, 1980) or in swine (Milne and Harris, 1978; Harris and Milne, 1981). The physiological factors which control the accumulation of 3-methylhistidine in swine and sheep are not exactly known. However, in swine, the intravenous dose of labelled 3-MeHis is largely retained, if not exclusively, in muscle where a considerable amount of non-proteinbound 3-methylhistidine exist (Harris and Milne, 1981). In sheep, Harris and Milne (1980a,b) reported evidence of a 3-methyhistidine dipeptide, balenine, present in muscle which ties up large amounts of 3-methylhistidine released during muscle protein degradation.

Because both actin and myosin are present generally in eukaryotic cells, the quantitative contribution by skeletal muscle 3-methylhistidine to the total urinary output of the amino acid is important to know if it is to be used as an index of muscle protein degradation. The assessment of the protein-bound 3-methylhistidine content of tissues and

organs of rats (Haverberg, 1975; Nishizawa et al., 1977), and in cattle (Nishizawa et al., 1979; Harris and Milne, 1981) has shown that 93 to 84 percent of the protein-bound 3-methylhistidine pool exist in skeletal muscle. These researchers all showed the mixed proteins, in all of the organs examined, contained detectable levels of protein-bound 3-MeHis. However, the total amount in these tissues is quite small compared with that of skeletal muscle mass.

Much of the skepticism surrounding the use of 3-methylhistidine as an index for muscle protein degradation involves the unknown contribution of the tissues and organs to the urinary-excreted pool. The contribution of each tissue source to 3-MeHis in urine depends on its turnover rate as well as its size. Millward et al. (1980) indicates that non-muscle sources of 3-methylhistidine may account for a considerable proportion of urinary excretion of the amino acid. They looked at rats in the steady state and then made the assumption that the methylation rate (synthesis) should be equal to the rate of release of 3-methylhistidine (degradation). Millward et al. (1980) used two different experimental approaches to evaluate the validity of the use of 3-MeHis as an index for skeletal-muscle degradation. In the first experiment, synthesis of the protein-bound 3-methylhistidine from S-adenosylmethionine was measured in skin at 2.61% day^{-1}, in muscle at 1.08% day^{-1} and in the gastro-intestinal tract at

9.57% day^{-1}, assuming the breakdown rates would be identical. In the second experiment, [^{14}C-CH_3] methionine was injected into rats and the radioactivity of 3-MeHis in urine was determined. They then analyzed the dilution curve, identifying three exponentials and equating them to muscle, skin and gastro-intestinal tract. By calculating the rate constants, the contribution of each tissue to urinary excretion was then determined, with muscle contributing 24.9% skin 6.8%, and gastro intestine 9.8% of the total excretion.

Harris (1981) however, points out errors and inconsistencies in the data published by Millward et al. (1980). First, the pool size of 3-methylhistidine in the gastro-intestinal tract and its contribution to the total amount excreted were overestimated by a factor of 3. This would change the contribution from 9.8% to 3.1% of the total daily excretion. Second, the fractional synthesis rate of 1.08% day^{-1} measured in the muscle of 250 gram rats was 27% that measured in mixed muscle protein of the same animals. In contrast, Millward and colleagues reported elsewhere (Bates et al., 1980) that actin and mixed muscle protein had fractional synthesis rates that were almost identical when measured in 100-150 gram rats in the same way as employed by Millward et al. (1980). No explanation of this large relative difference was offered by Millward

et al. (1980), nor was any reference of Bates et al. (1980) quoted. Harris claims that the results are ambiguous and that present evidence supports the view that most of the 3-methylhistidine in urine originates in skeletal muscle tissue and as a result, remains a suitable index of muscle protein breakdown in vivo.

Nishizawa et al. (1977) reported from studies of the comparative turnover of 3-MeHis-containing proteins in intestine, skin, and muscle, that intestine and skin may contribute about 17% of the total urinary 3-MeHis output. The accuracy, however, of this estimate is uncertain because the labelling technique used may be confounded by the reutilization of labelled methionine, which serves as the label donor for formation of labelled 3-methylhistidine (Young and Munro, 1978).

Bates et al. (1979) presented data which suggest that skin and intestine are rapidly turning over pools of 3-methylhistidine and could make up substantial contribution to 3-methylhistidine excreted in urine. Again, using similar experimental techniques to Millward et al. (1980), they state that skeletal muscle may contribute as little as 25% of the total 3-methylhistidine excreted. Harris et al. (1977) however, measured the fractional breakdown rate of mixed muscle proteins and calculated that 90% of 3-methylhistidine in urine originated in muscle, assuming actin and myosin have similar rates of turnover. This

contribution of muscle to 3-methylhistidine in urine becomes approximately 80% if correction is made for the rate of actin breakdown being 88% of mixed muscle protein (Lobley and Livie, 1979) and assuming that most of the 3-methylhistidine from muscle originates in actin (Haverberg et al., 1974).

Some studies have been conducted utilizing 3-methylhistidine as an index for skeletal muscle breakdown. Young et al., (1973) first discussed the potential use of 3-methylhistidine excretion as an index of progressive reduction in muscle protein catabolism during starvation in obese human patients. The excretion decreased progressively with starvation. These findings suggested that the reduced output of urinary 3-methylhistidine coincided with decrease urinary N output due to an adaptation and decrease in catabolism of muscle proteins as starvation progressed.

Haverberg et al. (1975) using rats, showed a marked and progressive decrease in 3-methylhistidine excretion in the protein-depleted group. The group restricted in both dietary protein and energy, showed an initial small increase in daily 3-MeHis output, but then a decrease in excretion. These results were confirmed by Funabiki et al. (1976) and Nishizawa et al. (1978), in subsequent studies. Ogata et al. (1978) and Nishizawa et al. (1978) both showed an increase in 3-methylhistidine excretion initially in

starved rats, indicating an increase in degradation of muscle. Ogata et al. (1978) did not see a decrease in excretion, however rats were starved for 72 hours then fed for 24 hours. Nishizawa et al. (1978) did see a decrease in excretion of 3-methylhistidine after 4 days of starvation. Omstedt et al. (1978) used 3-methylhistidine excretion as an index for muscle degradation in evaluating protein quality. They found that when higher quality protein was fed to rats depending on the protein uptake, an increase in urinary output of 3-methylhistidine occurred.

Nishizawa et al. (1979) attempted to evaluate and determine the fractional catabolic rate of myofibrillar proteins of skeletal muscle in cattle during growth, using urinary 3-methylhistidine excretion. He concluded that the catabolic rate of muscle protein was approximately 1.22%/day.

There have been several studies utilizing 3-MeHis excretion clinically to evaluate burn victims (Bilmazes et al., 1978) and Duchenne muscular dystrophy patients (Ballard et al., 1979). These studies indicate that with severe burns the excretion of 3-methylhistidine is increased (Bilmazes et al., 1978) and decreased in patients suffering from musclar dystrophy (Ballard et al., 1979).

In another report, children suffering from severe protein-energy malnutrition and children undernourished (Nagabhushan et al., 1978) showed decreased protein

degradation with 3-methylhistidine excretion decreased during malnutrition and undernutrition and increased considerably after treatment.

OBJECTIVES

1. To determine if an energy level effect on composition of gain does occur in cattle.
2. To determine the differences in composition of gain in cattle varying in frame size.
3. To determine the validity in using 3-methylhistidine as an index to measure muscle protein degradation in cattle.
4. To determine if a frame size effect for degradation of muscle protein does occur in cattle.

MATERIALS AND METHOD

PROTEIN ACCRETION AND COMPOSITION OF GAIN STUDIES

TRAIL 1 - ENERGY EFFECT

Experimental Animals

A feedlot trail was conducted from December 11, 1978 to October 11, 1979 utilizing a total of 31 7/8 blood to full blood Limousin steers. They were quite uniform in their type, frame and condition. There was a two month spread in birth dates ranging from March 8, 1978 to May 14, 1978.

Experimental Design and Rations

The experimental design is outlined in table 1. It consisted of a growing phase and a finishing phase with the switch occurring when the cattle weighed approximately 340 kg. The average initial weight was approximately 254 kg. All cattle were terminated at a constant weight of 522 kg.

All cattle were allotted to one of four treatments with 7 head allotted to each treatment. Three steers initially and four steers at the midterm switching point were slaughtered to determine body composition. Therefore, 24 steers were on trial for the entire experiment.

Table 1. Experimental Design For Studying the Energy Effect on Composition of Gain (Trial 1)

Treatments	1	2	3	4
Growing Phase[a] (250 kg-340 kg)				
Energy Level[b]	HIGH	HIGH	LOW	LOW
Days on Feed	70	70	102	103
Finishing Phase[a] (340 kg-522 kg)				
Energy Level[b]	HIGH	LOW	LOW	HIGH
Days on Feed	160	211	198	139

[a]Diet during growing phase balanced at 13% C.P.
Diet during finishing phase balanced at 11% C.P.

[b]High energy diet = 80% HMC (DM Basis).
Low energy diet = All corn silage.

Rations are shown on table 2 and are divided into high energy and low energy diets by phase. Cattle assigned to the high energy ration were initially offered an all corn silage ration (dry matter basis). The corn portion of the diet was increased to 35% high moisture corn the first week then increased to 60% the second week. By the third week steers were on the full-feed 20% silagecorn ration. This procedure was used to adjust steers to the high grain ration both initially and at the midterm switching point.

Trial 1 utilized four treatments to evaluate the energy effect on composition of gain. Treatment 1 steers were fed the high grain diet during both the growing and finishing phase (HH). Treatment 2 steers were fed the high grain diet during the growing phase and the high silage diet during the finishing phase (HL). Treatment 3 steers received the high silage diet during both the growing and finishing phase (LL) while treatment 4 received the high silage diet during the growing phase and the high grain diet during the finishing phase (LH).

Management Procedures

Within 24 hours of arrival to the MSU Beef Cattle Research Center, all steers were given identification tags, and vaccinated for pasteurella, IBR, BVD, AND PI_3. All cattle were injected with 2 million I.U. of vitamin A.

Table 2. Rations Fed to Limousin Steers in Feedlot Study (Trial 1)

Item	Int. ref. no.	Growing		Finishing	
		High Silage % DM	High Grain % DM	High Silage % DM	High Grain % DM
Corn, aerial pt, w-ears, w-husks, ensiled, well-eared mx. 50% mn. 30% dry matter	3-08-153	87.00	20.00	92.00	20.00
Corn, dent, yellow grain, gr 2 US	4-02-931		72.90		78.00
Soybean, seeds, meal solv-extd	5-04-604	12.14	5.85	7.08	1.38
Limestone, grnd	6-02-632		0.96		0.33
Phosphate, deflourinated, grnd	6-01-780	0.57		0.63	
Trace Mineral Salt		0.25	0.25	0.25	0.25
Vitamin A[a]		0.02	0.02	0.02	0.02
Vitamin D[b]		0.02	0.02	0.02	0.02
Percent of ration dry matter:					
Crude Protein[c]		13.00	13.00	11.00	11.00
Calcium[c]		0.46	0.46	0.46	0.46
Phosphorous[c]		0.34	0.34	0.34	0.36

[a] 30,000 IU vitamin A per gram.

[b] 3,000 IU vitamin D per gram.

[c] Calculated from average nutrient composition (NRC, 1976).

Rations were mixed immediately prior to feeding in a horizontal batch mixer. The complete ration was then transported by conveyor belt to the appropriate pen feedbunk. Cattle were fed ad libitum once daily in sufficient amounts so that bunks were nearly cleaned up at feeding time. Daily feed records were maintained and periodically, the unconsumed feed was removed, weighed and the amount recorded.

All cattle were group-fed and housed in concrete lots which were partially covered, and bedded with straw. Approximately one-half of the floor space of each pen was covered by a roof. Automatic waterers supplied the cattle with adequate amounts of water.

Slaughter Procedures and Carcass Evaluation

Initially three cattle were selected to be representative of those cattle placed on feed in the trial and body composition was determined using the 9-10-11th rib section technique. Animals were slaughtered at a packing plant location 25 miles from the Beef Cattle Research Center.

Intermediate slaughter calves were also selected to represent the average calf in the treatment pen. One steer per treatment, a total of four, was slaughtered and body composition was determined using the 9-10-11 rib section analysis technique. Animals were slaughtered at the MSU Meats Laboratory.

Cattle were all slaughtered at the termination of the trial at the MSU Meats Lab. Body composition was determined on the carcass using the specific gravity technique and the 9-10-11 rib section analysis. Hot carcass weights were obtained along with carcass data after the carcasses had been chilled a minimum of 24 hours.

Body Composition Determination

Body composition of the experimental animals was determined by either specific gravity, 9-10-11 rib section analysis or deuterium oxide dilution and in some cases all three methods were used as a comparison of techniques. Initial, midterm and final kill steers all had body composition determined by the 9-10-11 rib section and D_2O dilution techniques. In addition, all final kill steers had body composition determined by the specific gravity technique. The 24 steers that were on test the entire trial were infused with D_2O on six occasions: initially, finally, midterm switching point, half way through the growing phase and twice during the finishing phase. The object of the six body composition determinations using D_2O was to monitor composition of growth at 45-70 kg intervals.

For all steers slaughtered, 9-10-11 rib analysis and D_2O dilution were performed to compare techniques in estimating composition. The specific gravity technique was performed on final kill steers and compared with both 9-10-11 rib section and deuterium oxide dilution techniques.

Correlations between the three body composition techniques were then calculated.

Specific Gravity

The application of carcass density in determining body composition is the primary factor in the specific gravity method (Garrett and Hinman, 1969). Carcass weights are obtained on the chilled carcass in the air and under water. A galvanized steel tank, 112 cm in diameter and 183 cm in height, was fitted with a triple-beam pan Toledo balance. The tank was filled to near capacity with water, and crushed ice was added to maintain the temperature of the water at 10°C or lower. The front and rear quarter of one side of each carcass was suspended from the balance and immersed underwater and the weight recorded. It is necessary to remove any air pockets in the carcass before recording the weight. Carcass and water temperatures were obtained periodically and recorded for later calculations of correction factors. The following equations were developed by Garrett and Hinman (1969) to estimate carcass composition using the density of the carcass:

$$\% \text{ carcass fat} = 587.86 - 530.45 \times SG$$

$$\% \text{ carcass protein} = (20.0 \times SG - 18.57)(6.25)$$

$$SG = \frac{\text{CW in air}}{(\text{CW in air} - \text{CW in } H_2O)(\text{Correction for } H_2O \text{ \& Carc-temp})}$$

9-10-11 Rib Section

The estimation of body composition by analysis of the 9-10-11 rib cut from one side of each carcass used in trial 1, was developed and outlined by Hankins and Howe (1946). Rib sections were removed from the carcasses and further processed at the MSU Meats Lab. Ribs were subsequently separated into bone and soft tissue. The soft tissue portion was ground through a Hobart meat grinder (0.47 cm screen) five times and thoroughly mixed and a subsample (1 kg) was frozen for subsequent analyses. Rib tissue was analyzed for moisture by drying approximately a 6 to 7 g sample at 100°C for 24 hours. Ether extract was determined on dried samples with the Goldfisch procedure. Total N was determined on a 1 g wet sample using a techicon Auto-Kjeldahl System. The following equations were used to estimate carcass composition from the rib cut composition:

$$\% \text{ carcass Protein} = .66(\text{rib protein } (\%)) + 5.98$$

$$\% \text{ carcass Fat} = .77\ (\text{rib fat } (\%)) + 2.82$$

Using the equations developed by Garrett and Hinman (1969), empty body composition was calculated:

$$\text{Empty Body Protein } (\%) = .7772(\text{carcass protein } (\%)) + 4.456$$

$$\text{Empty Body Fat } (\%) = .9246\ (\text{carcass fat } (\%)) - 0.647$$

Deuterium Oxide Dilution

The idea of using isotopic dilution to determine the body composition of live animals is not new. Byers (1979) developed a procedure that separates the empty body water from gut water thus, improving the accuracy of the procedure.

Deuterium oxide (99.8% D_2O) was used as a tracer for tracing body water in beef cattle. The D_2O was made to physiological osmolarity by adding 9g NaCl/1000 ml. Ten grams of physiological D_2O per 45 kg of body weight were infused to produce an initial blood concentration of 400-600 ppm. A 30-45 cm piece of polyethylene surgical tubing (PE200), inside diameter of 1.4 mm, was passed through a 12 gauge needle placed in the jugular vein in an animal that had its head restrained in a head gate. A one-way stainless steel stopcock was attached to a catheter in the vein. Following infusion of the D_2O, the stopcock and catheter was flushed with 50 ml of physiological saline to insure that all D_2O was washed into the animal's circulation.

All blood samples (12 cc each) collected were placed in heparinized test tubes to avoid clotting of blood. Following the collection of an initial sample, preweighed syringes (50 or 60 cc) containing the desired amount of physiological D_2O, were infused in single dose as fast as possible. The catheter was flushed by withdrawing the

discarding 20-30 ml of blood immediately prior to collecting the first sample and 10 ml before the later samples. Samples were collected at time intervals sufficient to produce a dilution curve to calculate pool size. The time intervals were t_o or initial, prior to infusion to ascertain any background levels of D_2O. Then samples were collected at 20, 30, 40, 50 and 80 min., at 4-6 hours and 1,2, and 3 days after infusion. All animals were infused in the morning prior to feeding and weighed each day samples were collected and averaged to attain the live weight used for calculations.

Samples were analyzed using methods by Byers (1979). Blood was transferred to 100 ml volumetric flasks, then attached to condensers (cold finger) which were partially submerged in super-cooled (-80°C) methanol. Vacuum (5 mm Hg or less) was then applied to the collection apparatus in order to lyophilize the blood sample (3 hours recovery time). Water, collected from the blood in the cold finger, was thawed and analyzed in an infrared spectrophotometer.

A Wilks Scientific Miran I Fixed Filter Infrared Analyzer was used to measure absorbance. A 4.0 micron filter was used to provide the wavelength that coincides with maximum absorbance of D_2O and minimum absorbance for H_2O. Precision sealed calcium fluoride cells with 0.2 mm spacers were used for all samples. The 0.2 mm pathlength reduces the amount of energy put through and electronic noise, and increases sensitivity of the equipment.

Since the absorptivity of D_2O bands exhibit temperature dependency, a temperature controlled cell holder was used. Fluid was circulated through the cell holder from a temperature controlled bath to maintain a proper temperature (24.5°C).

Aliquots of the H_2O-D_2O solution were injected into the CaF_2 cell of the I.R. analyzer which analyzed the concentration of D_2O in the water to the nearest 1 ppm.

After the water samples were analyzed in the I.R. spectrophotometer, the dilution curve was graphically analyzed. Using the two-pool open system kinetics from Shipley and Clark (1972), pool size estimates were calculated. Figure 2 illustrates the model for the two-pool open system. The overall equations for the system are:

$$SA_t = SA_o(1)e^{-k_1 t} + SA_o(Z)e^{-k_z t}$$

Normalized General Equation-fraction of dose:

$$q_a/q_{ao} = H_1 e^{-g_1 t} + H_2 e^{-g_2 t}$$

Where: q_a = quantity of tracer in pool a

q_{ao} = dose placed in pool a at zero time

H_1 = (intercept 1) (Q_a)

H_2 = (intercept 2) (Q_a)

Needed equations to solve for rate constants:

$$K_{aa} = H_1 g_1 + H_2 g_2$$

$$K_{aa} + K_{bb} = g_1 + g_2$$

$$K_{aa} K_{bb} - K_{ab} K_{ba} = g_1 g_2$$

$K_{ab} = K_{bb}$, since pool a is the only outlet for pool b

K_{aa}, K_{bb} = overall turnover for pools A and B respectively

$K_{oa} = K_{aa} - K_{ba}$

Flow Rates:

$F_{ba} = K_{ba} Q_a$

$F_{ab} = F_{aa} = K_{aa} Q_a$

$F_{oa} = K_{ab} Q_b$

So: $Q_b = F_{ab} / K_{ab}$

Pool Sizes:

Pool A = $Q_a = 1 / SA_{ao}$

Pool B = $Q_b = F_{ab} / K_{ab}$

2-pool open system example calculation:

Live weight - 390 kg

Dose - 99.50 grams

Time following infusion (min)	D_2O, PPM (mg/kg)
15	437.0
26	388.5
35	371.3
45	366.9
502	306.9
1496	289.0
3196	258.0
4596	232.0

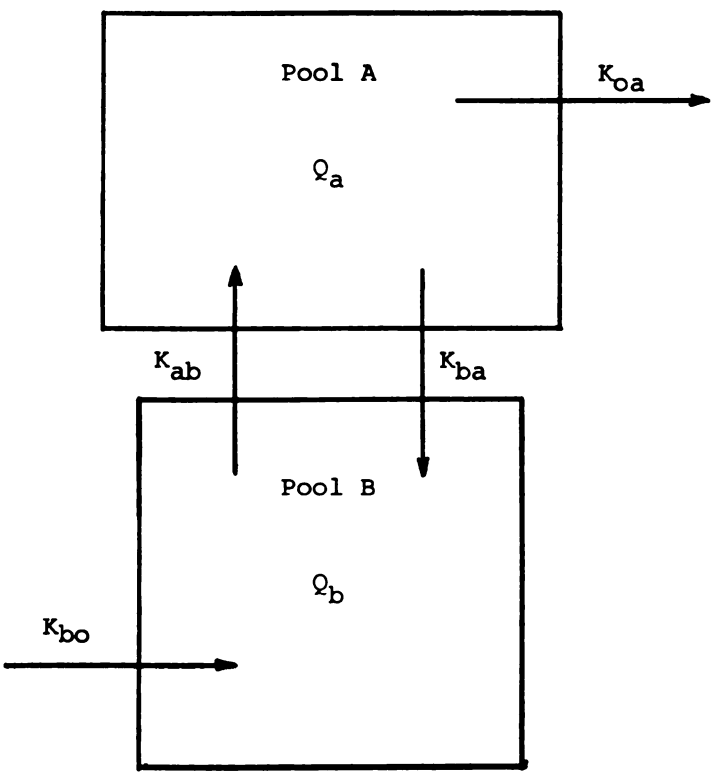

Figure 2. Model for a 2-pool open system.

By using a mathematical process called "curve peeling", the semi-log regression of D_2O (ppm) was plotted against time following total equilibration to provide the intercept and slope of the line. Predicted D_2O levels from this regression for early samples are subtracted from actual concentrations at these time and the semi-log regression of these differences vs time gives rise to the $intercept_1$ and $slope_1$. Equations are shown in figure 3.

Specific activity for late pool:

$$SA_t = 318.35e^{-,0000677t}$$

Specific activity "peeled" for early pool:

$$SA_t = 168.08e^{-.02954t}$$

Total function describing the SA of pool a:

$$SA_t = 168.08e^{-.02954t} + 318.35e^{-.0000677t}$$

Dividing the intercepts by dose:

$$\frac{168.08 \text{ mg/kg}}{99500 \text{ mg}} = .001689 \text{ / kg water}$$

$$\frac{318.35 \text{ mg/kg}}{99500 \text{ mg}} = .003199 \text{ / kg water}$$

$SA_{(to)}$ values as fraction of dose infused.

$$SA_{at} = .001689e^{-.02954t} + .003199e^{-.0000677t}$$

Solving for compartment sizes, rate constants and flow rates:

$$Q_a = \frac{1}{SA_{ao}} = \frac{1}{.004888} = 204.58 \text{ kg}$$

where SA_{ao} = combined intercepts of SA_a curve, at t_o exponents = 1

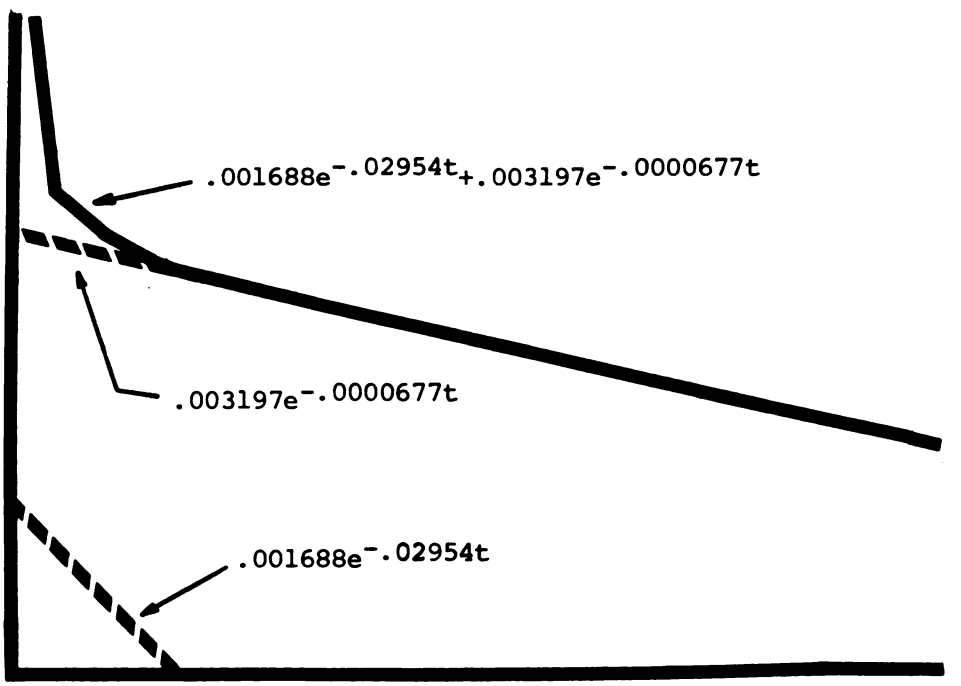

Figure 3. Kinetic equations for pool separation.

Normalized equation:

$$H_1 = .001698(204.58) = 0.3455$$

$$H_2 = .003199)204.58) = 0.6545$$

$$q_a / q_{ao} = .3455e^{-.02954t} + .645e^{-.0000677t}$$

$$K_{aa} = H_1 g_q + H_2 g_2 = (.3455)(.02954) + (.6545)(.0000677) = .0102515$$

$$K_{bb} = g_1 + g_2 - K_{aa} = .02954 + .0000677 - .0102515 = .0193562$$

$$K_{ab} = K_{bb} = .0193562$$

$$K_{ba} = \frac{(K_{aa})(K_{bb}) - (g_1)(g_2}{K_{bb}} = \frac{(.0102515)(.0193562) - (.02954)(.0000677)}{.0193562}$$

$$K_{oa} = K_{aa} - K_{ba} = .0102515 - .0101482 = .0001033$$

Flow Rates:

$$F_{ba} = K_{ba}Q_a = (.0101482)(204.69) = 2.077 \text{ kg/min}$$

$$F_{ab} = F_{aa} = K_{aa}Q_a = (.0102515)(204.69) = 2.09838 \text{ kg/min}$$

$$F_{oa} = K_{oa}Q_a = (.0001033)(204.69) = .02114 \text{ kg/min}$$

Since $F_{ab} = K_{ab}Q_b$

$$Q_b = F_{ab}/K_{ab} = \frac{2.09838 \text{ kg/min}}{.0183562} = 108.40 \text{ kg}$$

Pool A = 204.58 kg

Pool B = 108.41 kg

Empty Body Water, kg = 1.038 (pool A) - 17.918

Gastro-Intestinal weight, kg = .832(pool A)-17.918

Empty Body Weight, kg = Live weight - (Pool B(.83)-.31)

Empty Body Protein, kg = .3017(EB Water)

Empty Body Mineral, kg = .0689(EB Water)

Empty Body Fat, kg = Live weight - (G.I. weight + EB H_2o/.7296)

Statistical Analysis

In feedlot trial 1, analysis of variance (Snedecor and Cochran, 1967) was used to examine main effects and interactions for all variables. Mutiple analysis of variance was then used when appropriate, and contrasts were designed for comparing selected treatment combinations of primary interest. If $P \leq .20$, the level of statistical significance was reported. $P < .05$ was determined as being significant, while $P < .01$ was reported as highly significant.

TRIAL 2 AND TRIAL 3 - FRAME SIZE EFFECT

Experimental Animals

Feeding trials were conducted from April 22, 1980 to October 7, 1980 and from November 5, 1980 to April 21, 1981 utilizing 20 cattle of two frame sizes. Steers representing the small mature frame-size were primarily Hereford, Angus, or Hereford X Angus cattle. Simmental or Charolais crossbred steers were used as representatives for

large mature frame-size cattle. Cattle for trial 2 were purchased in March of 1980 to begin that experiment in April. Cattle for trial 3 were (USH and AHSC) steers from the Lake City Breeding Project described by Harpster (1978). Average initial weights for trial 2 were 307 and 322 kg for small frame and large from respectively and the average initial weights were 206 and 288 kg for small and large frame steers respectively for trial 3. The combined average initial weights, for trial 2 and trial 3, were 260 kg for the small frame and 302 kg for the large frame cattle.

Experimental Design and Rations

The experimental design and rations fed in trial 2 and 3 are presented in tables 3 and 4, respectively. The design of the two trials were the same in both cases (Table 3). Large frame and small frame cattle were used to evaluate the effect of frame size on composition of gain. Trial 2, consisted of 6 small and 6 large frame steers while, trial 3 utilized only 4 steers for each treatment. In both trial 2 and trial 3, cattle were fed a high grain diet (between 70 and 80% concentrate) with crude proteins of 13 and 14 percent of the rationdry matter respectively. The roughage in the ration for trial 2 was pelletted corn cob meal and for the ration in trial 3, chopped alfalfa hay (Table 4).

Table 3. Experimental Design for Studying the Frame Size Effect on Composition of Gain (Trial 2 and 3)

Treatments	1	2
Growing Phase[a]	SMALL	LARGE
Finishing Phase	SMALL	LARGE

[a]Year 1 and Year 2 diets were isocaloric.
Year 1 diet balanced at 13% C.P.
Year 2 diet balanced at 14% C.P.

Table 4. Rations Fed to Steers Varying in Frame Size (Trials 2 and 3)

Item	Int. ref. no.	Trial 1 % DM	Trial 2 % DM
Corn cob pellets (#4, Andersons)		15.00	
Alfalfa, hay, S-C, mature	1-00-071		10.00
Corn, dent, yellow, grain, gr 2 US, cracked	4-02-931	69.46	78.76
Soybean, seeds, meal solv-extd.	5-04-604	9.50	10.00
Sugarcane, molasses	4-04-696	5.00	
Limestone, grnd	6-02-632	0.70	0.95
Trace Mineral Salt		0.30	0.25
Vitamin A[a]			0.02
Vitamin D[b]			0.02
Percent of ration dry matter:			
Crude Protein[a]		13.00	14.00
Calcium[b]		0.40	0.46
Phosphorous[c]		0.36	0.34

[a]30,000 IU vitamin A per gram.

[b]3,000 IU vitamin D per gram.

[c]Calculated from average nutrient composition (NRC, 1976).

Management Procedures

Calves used in these trials were either western bred or calves from the Lake City Breeding project. All calves were processed within 12-24 hours of arrival at the MSU Beef Cattle Research Center. Incoming calves were vaccinated for pasteurella, IBR, BVD, and PI_3 and were given intramuscular injections of vitamins A and D. After a 30 day adjustment period pour-on insecticide was applied to all calves to control grubs and lice. Vitamin A and D injections were given every 60 days to all calves on trial.

Cattle were fed ad libitum in individual feeders once daily. Ration ingredients fed in trial 2 were premixed in batches of 2000 pounds at the feed mill and bagged in paper bags. Mixing of the ration for trial 2 was done every two weeks, according to the comsumption of dry matter by the experimental animals. Rations for trial 3 were mixed daily prior to feeding in a horizontal batch mixer. Daily feed records were maintained and unconsumed feed was periodically removed, weighed and recorded.

All cattle were individually weighed at the beginning of each experiment and every 28 days thereafter until termination. Initial and final weights were determined as an average of weights from four successive days at the beginning and end of the trials.

All cattle were individually penned in 240 cm square stalls in an environmentally controlled metabolism room. Each steer had an individual feeder and access to an automatic waterer.

Slaughter Procedures and Carcass Evaluation

Trial 2, steers were slaughtered when they were estimated to grade high good to low choice. Steers in trial 3 were all slaughtered when it was estimated that 75% of the small frame steers would grade choice.

Cattle in trial 2 were slaughtered at a small packer in Okemos, Michigan in two groups. Five of the small framed steers were slaughtered first then 4 weeks later, the remainder of the steers were slaughtered. Cattle in trial 3 were slaughtered at the MSU Meats Lab all within one week of each other.

Carcass data were collected in a like manner regardless of the trial year and slaughter location. Complete carcass quality and yield data were collected after carcasses had been chilled for a minimum of 24 hours.

Body Composition Determination

Body composition was determined in the experimental animals using the deuterium oxide isotope dilution technique previously discussed. Cattle in both trials were

infused with D_2O to estimate composition at the start of the trial, midway through the trial and at the end of the trial prior to slaughter.

Statistical Analysis

To evaluate the statistical differences among frame sizes, a two way analysis of data was done including frame size and years. The procedure of least squares was used because of the unequal numbers of steers in the two years. A program in the Stat 4 series was used on the Cyber 750 computer at MSU.

MUSCLE PROTEIN DEGRADATION STUDIES

3-METHYLHISTIDINE AS AN INDEX OF MUSCLE BREAKDOWN

Metabolism and Reutilization Study

Experimental Animals, Design and Collection Procedures

Two Charolais crossbred heifers, weighing approximately 323 kg, were used to evaluate the fate of [^{14}C]-3-methylhistine in cattle during the winter of 1979. The heifers were housed in an environmentally controlled metabolism room confined in individual 91 x 244 cm stalls designed to allow urine collection. Cattle used in this trial received a corn-hay ration during the collection period.

The two heifers were each given 10.83 μCi injections intravenously of [^{14}C]-3-methylhistidine. By placing an indwelling Foley catheter through the urethra and into the bladder, urine was quantitatively collected for 120 hours from the heifers. Urine volume was measured to the nearest ml and the quantity recorded. After agitating the urine collected during each time period, sub-samples were taken. Remaining portion of urine collection was properly discarded through the MSU Radioactive Waste Disposal Service.

Scintillation Counting

All radioactive determinations were made using a scintillation counter (Tracor), which had an automatic quench calibration that was performed by the combined external standard channels ratio method. Samples were counted in ACS cocktail (aqueous counting scintillant, Amersham Corp.).

For determination of the CPM of [^{14}C]-3-methylhistidine in the urine, samples were placed in 15 ml of scintillation cocktail at quantities of 0.2 ml undiluted urine or 0.2 ml of urine diluted 1:4 with H_2O. A standard containing 0.05 ml of [^{14}C]-3-MeHis, used in the experiment, added to 15 mls of scintillation fluid was used to determine activity of label and a blank sample containing 0.2 ml water in 15 mls of cocktail was used to determine any background.

3-Methylhistidine Excretion Studies

Experimental Animals, Design, Ration and Collection

Cattle used for this experiment were the same steers utilized in trial 3 and described in a previous section. Of eight steers used, four steers were designated small framed and four steers as large framed, to evaluate first, the excretion of 3-MeHis as an index for muscle protein

breakdown and second, to determine if there was a frame effect on turnover rates in cattle. Cattle were fed a corn-hay ration balanced at 14% crude protein. This ration has been discussed in detail earlier.

Urine collection was conducted by placing the steers in individual 91 X 244 cm stalls designed to allow collection of urine and feces. Collections were in 3 day periods every 35 days, for a total of five collections. Urine was collected in large plastic containers containing enough acid to acidify to pH 2-3; this required approximately 200 ml of 50%, H_2SO_4. The containers were emptied once daily, urine volumes were measured and recorded. Ten percent aliquots were secured, composited for given steers during the collection period and stored at 5°C until the collection period was completed. After each 3 day collection period the composited urine sample was stored at -20°C until analysis.

Urine Preparation and Analysis

Urine samples were centrifuged to remove sediments them deproteinized with equal volumns of 10% SSA (sulfosalicylic acid). After allowing the samples to sit at least 60 minutes in ice, they were centrifuged at 1000X g for 15 minutes. Hydrolysis of urine samples using an equal volume of 12N HCl, was done by heating to 110°C for 20

hours in sealed tubes flushed with nitrogen gas for 30 seconds. Prior to hydrolysis, an internal standard, S-β-(4-Pyridylethyl)-L-cysteine (PEC) was added (1.0 ml of 1mM PEC solution).

After hydrolysis, the hydrolysate was then filtered through No. 2 Whatman filter paper. The filtered hydrolysate was then evaporated to dryness, redissolved in distilled water and re-evaporated to dryness twice. The dried hydrolysate was finally dissolved in 5 ml 0.2 M pyridine, and refiltered through Metricel 0.2 m membrane filter using a milli-pore filtering system.

The pyridine dissolved sample was applied to a column (1.5 x 7.5 cm) containing a cation exchange resin, Dower 50W-X8 with 200-400 mesh designation. The column had 30 ml water passed through to desalt the resin then was equilibrated with 40 ml of 0.2 M pyridine. The acid and neutral amino acids along with creatinine, were eluted with 100 ml of 1 M pyridine and this portion was dried by evaporation. Histidine, lysine, 1-methylhistidine, methyllysine and methylarginines were eluted after the initial 100 ml of 1 M pyridine, with 150 ml of 1 M pyridine solution.

The 3-MeHis fraction collected was evaported to dryness, then dissolved in distilled water and re-evaporated, twice. The sample was then dissolved in 5ml of 0.01 N HCl and analyzed on an amino acid analyser (Dionex) using a Pico IV buffer system with lithium citrate buffers. A program was modified to elute all amino acids initially except the 3-MeHis and PEC fraction, which were eluted last.

Creatinine Analysis

Creatinine determinations for the urine were done using Folin's Method of the Jaffe reaction as described in Hawk's Physiological Chemistry (1965).

RESULTS AND DISCUSSION

Body Composition Estimates

Considerable research involving many diverse techniques has been done in attempts to develop reliable measurements of body composition. In this study three different techniques were utilized to estimate body composition in cattle. Table 5 summarizes the results of comparisons between carcass specific gravity, deuterium oxide dilution and 9-10-11 rib tissue separation techniques in an attempt to evaluate each method.

The range in size of animals used for the comparisons was quite narrow (225 to 525 kg empty body weight) making the probability of getting high correlation coefficients less than if the range were wide.

The formula used to describe the linear regression relationship between two methods being compared was: $y = mx + b$. The y and x variables are supplied by the comparative data point of the methods being regressed. Variable m is the slope of the line and implies that for each change in y, a change in x occurs. The y-intercept is represented as b in the formula and is the point on the y-

Table 5. Relationships Between Carcass Specific Gravity, 9-10-11 Rib Separation and Deuterium Oxide Dilution Techniques for Estimating Body Composition

Variables	Corr. Coef.(r)	y Intercept	Slope	SE
Sp. Gr. vs. D_2O				
Empty Body Weight	.88	87.20	.81	3.27
Empty Body Water	.60	18.53	.91	3.24
Empty Body Protein	.57	-.84	.98	1.18
Empty Body Fat	.67	56.05	.54	3.40
% EB Protein	.73	4.93	.70	.12
% EB Fat	.53	12.99	.50	.71
D_2O vs. Rib[a]				
% EB Protein	.72	4.24	.83	.12
% EB Fat	.71	.78	.77	.60
Sp. Gr. vs. Rib[a]				
% EB Protein	.59	8.90	.53	.13
% EB Fat	.48	11.05	.41	.73

[a]Twenty four animals were used to compare Specific Gravity to D_2O and Rib Section techniques while, 31 animals were used to compare D_2O to Rib Section techniques.

axis when the quantity is zero on the x-axis. Ideally, when comparing the methods with each other, the slope would be 1 and the y-intercept 0. Any deviation from ideal was subject to interpretation.,

Figures 4 through 9 graphically interpret the regression equations for various comparisons between specific gravity and D_2O dilution. Empty body weight was most highly correlated (r=.88), but empty body protein weight had the highest slope (m=.98). Percent empty body fat had both the lowest correlation coefficient (r=.53) and the lowest slope (m=.50). Our data would indicate, by comparing y intercepts and slope, that specific gravity and D_2O dilution are similar in their predictions of empty body water, which should confirm data reported by Byers (1979).

An evaluation of the regression lines would indicate a trend for D_2O dilution to estimate slightly higher empty body weights, along with higher quantities of empty body water and fat. Empty body protein estimates would appear to be quite similar for both methods.

The correlation coefficients are not as high as those shown by Byers (1979), but when comparing weights in a similar range to those used in his experiment with ours, distribution of points follow about the same pattern.

Figure 4. Empty Body Weight - Specific Gravity versus D_2O Dilution.

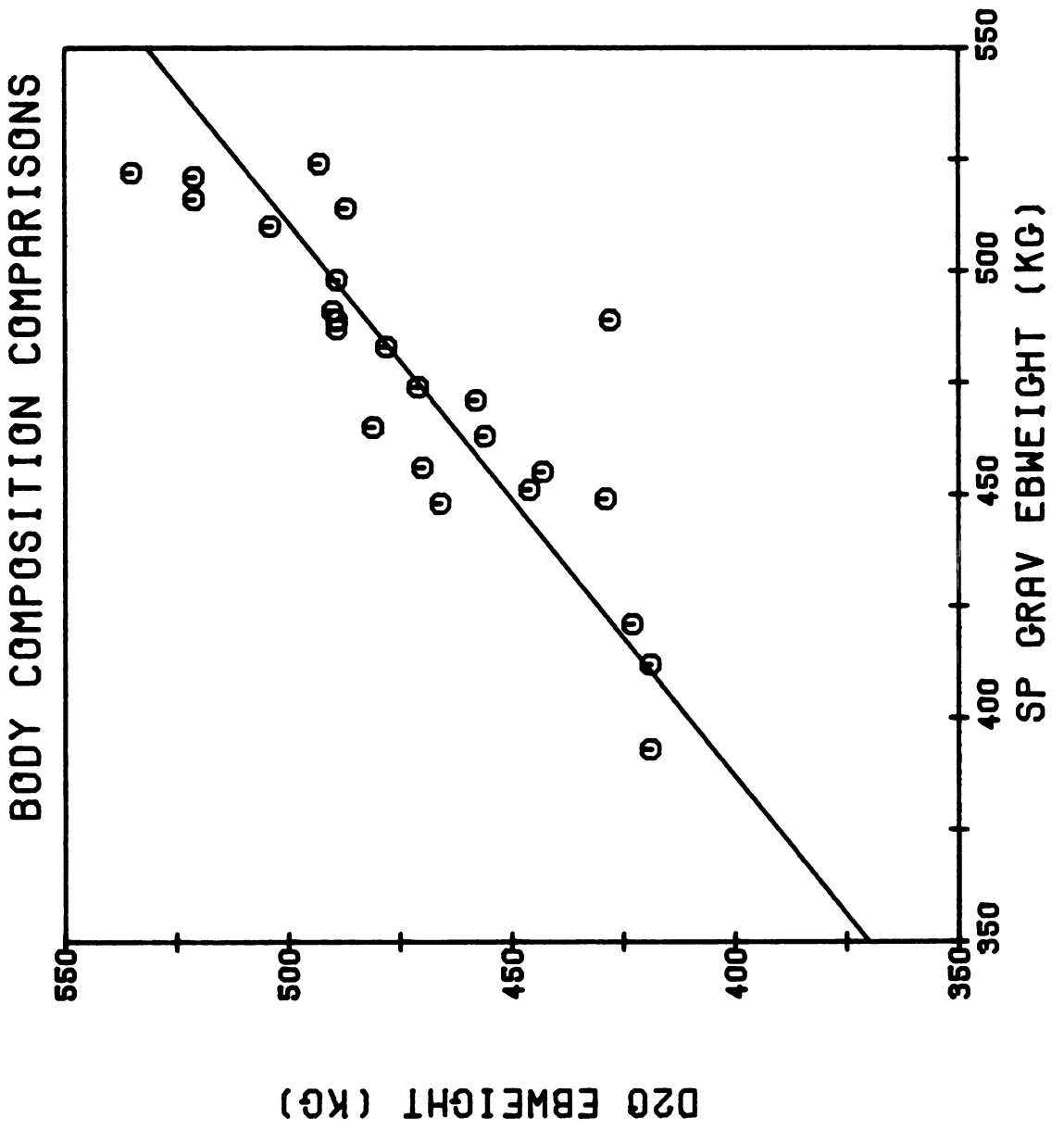
BODY COMPOSITION COMPARISONS
D2O EBWEIGHT (KG)
SP GRAV EBWEIGHT (KG)
550
500
450
400
350
350
400
450
500
550

Figure 4.

Figure 4. Empty Body Weight - Specific Gravity versus D_2O Dilution.

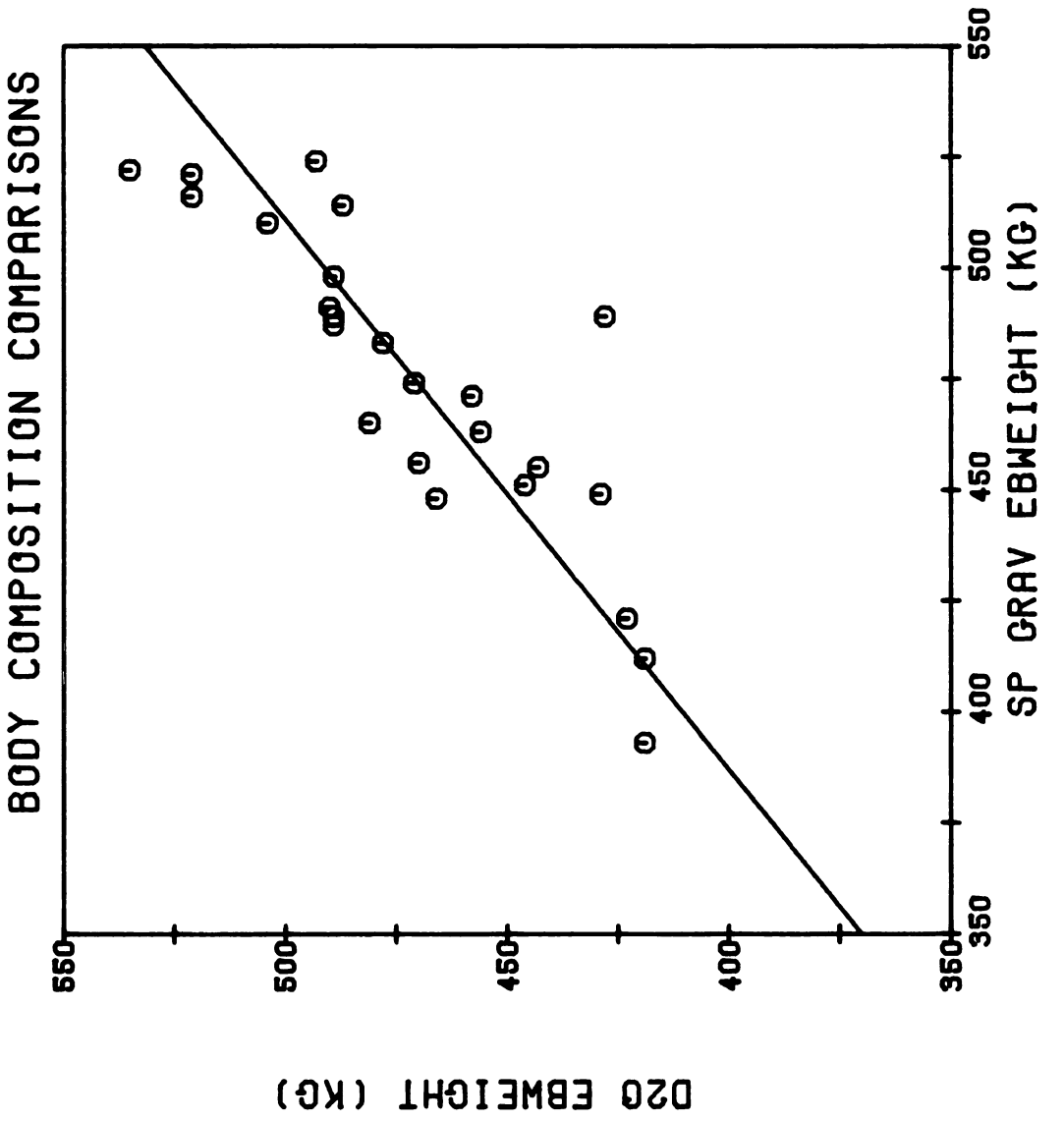
BODY COMPOSITION COMPARISONS
D2O EBWEIGHT (KG)
SP GRAV EBWEIGHT (KG)
550
500
450
400
350
550
500
450
400
350

Figure 4.

Figure 5. Empty Body Water - Specific Gravity versus D_2O Dilution.

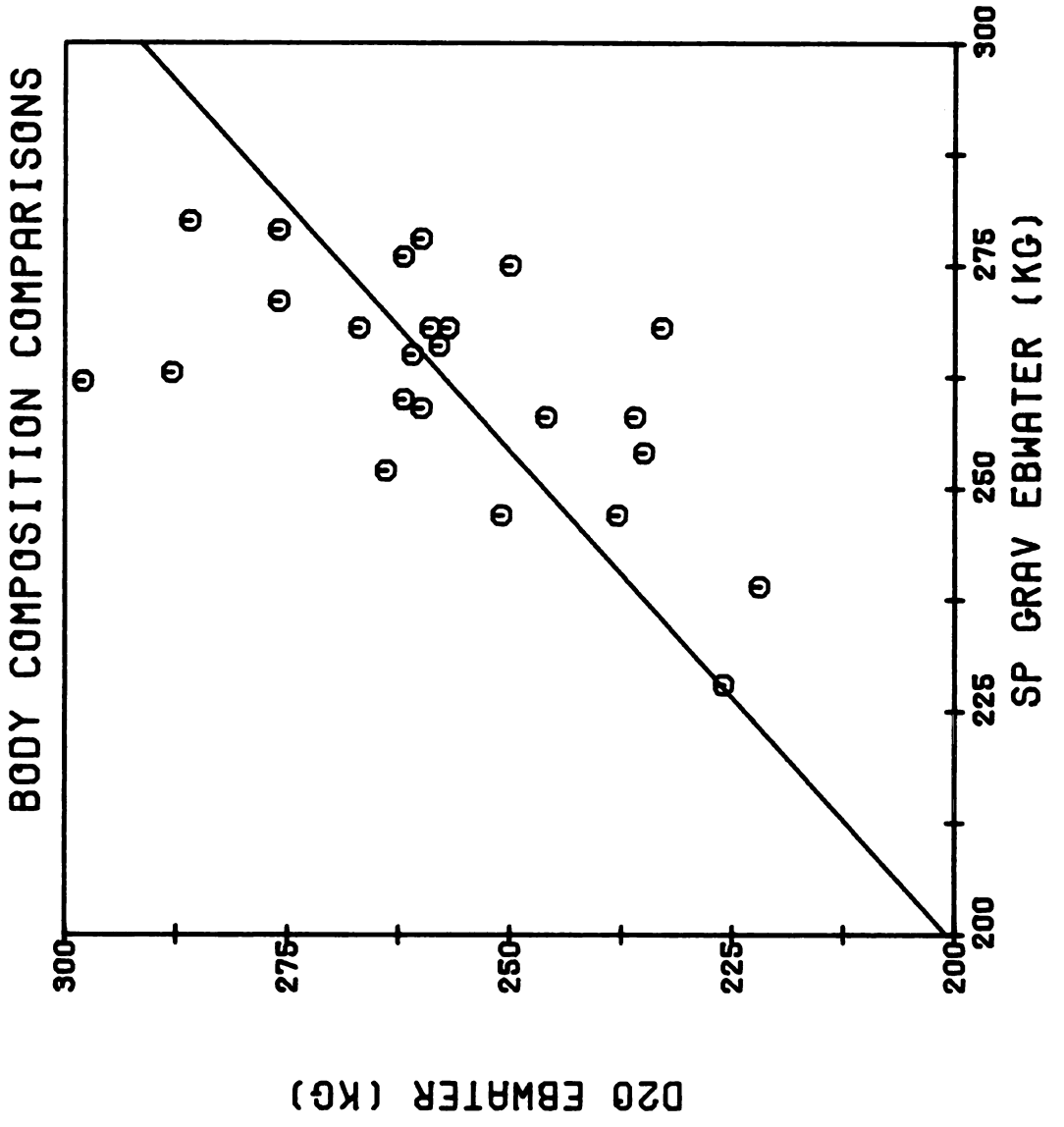
BODY COMPOSITION COMPARISONS
D20 EBWATER (KG)
SP GRAV EBWATER (KG)
300
275
250
225
200
300
275
250
225
200

Figure 5.

Figure 6. Empty Body Protein - Specific Gravity versus D_2O Dilution.

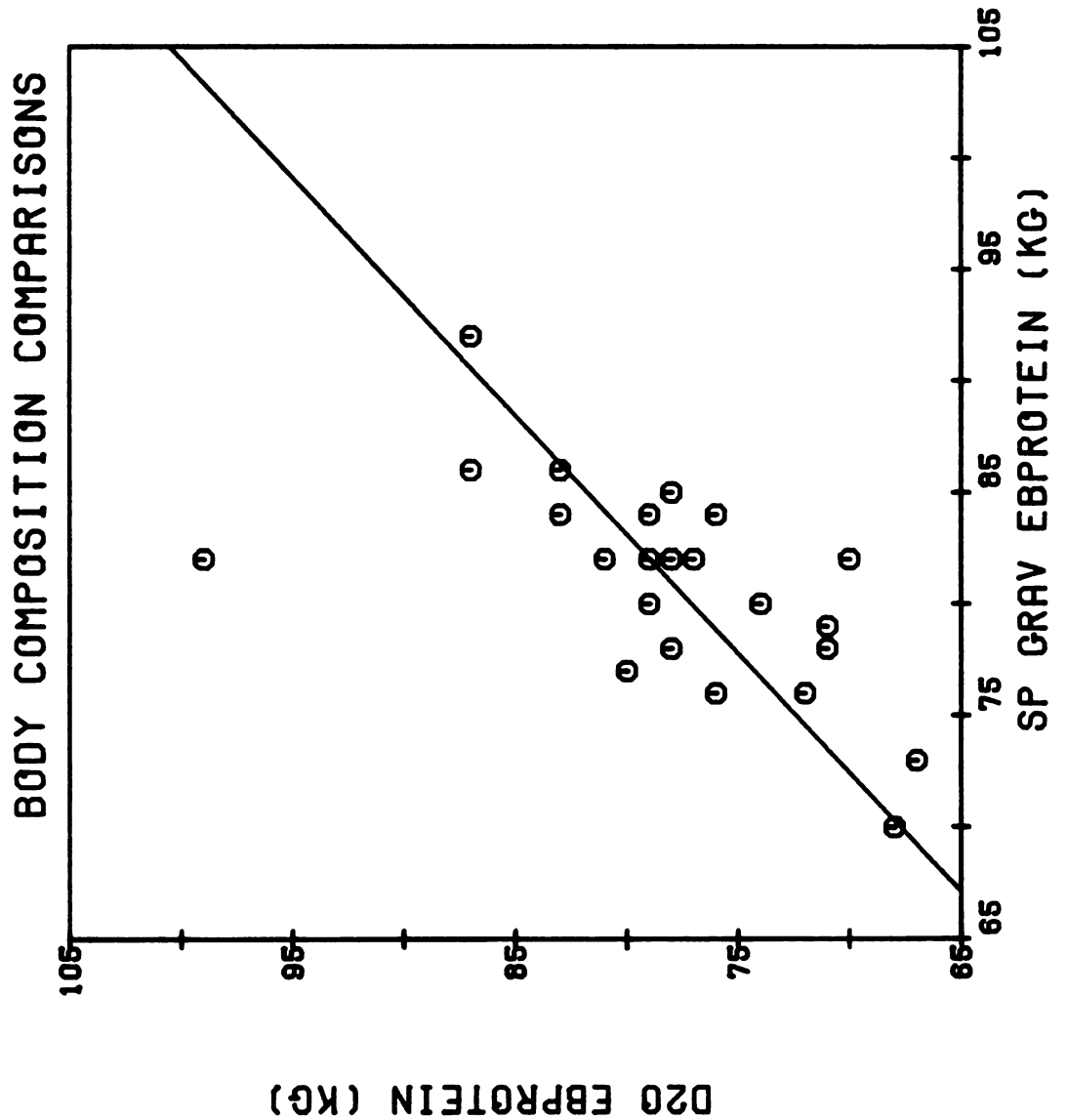
BODY COMPOSITION COMPARISONS
SP GRAV EBPROTEIN (KG)
D2O EBPROTEIN (KG)
105
95
85
75
65

Figure 6.

Figure 7. Empty Body Fat - Specific Gravity versus D_2O Dilution.

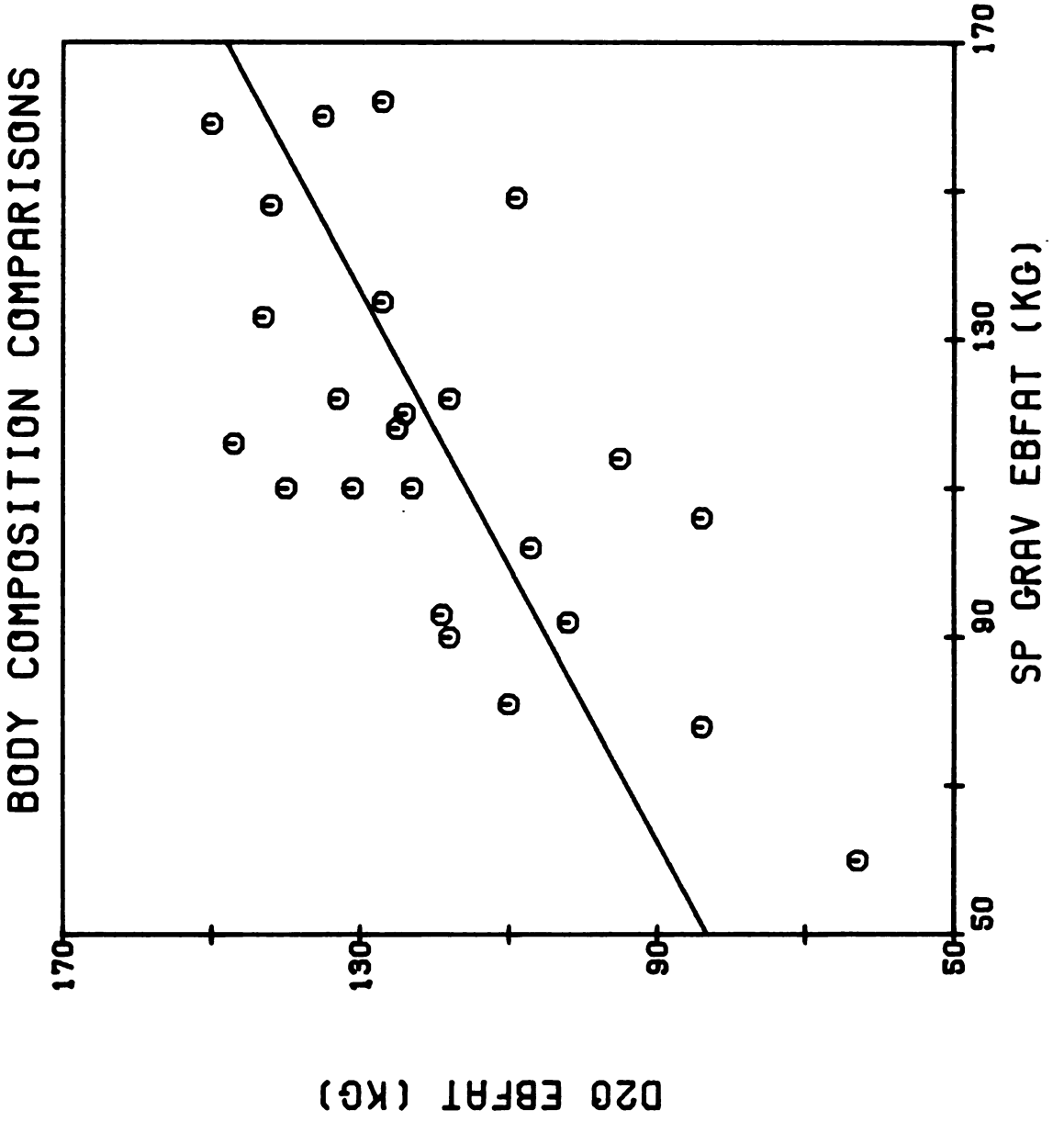
BODY COMPOSITION COMPARISONS
D2O EBFAT (KG)
SP GRAV EBFAT (KG)
170
130
90
50
50
90
130
170

Figure 7.

Figure 8. Percent Empty Body Protein - Specific Gravity versus D_2O Dilution.

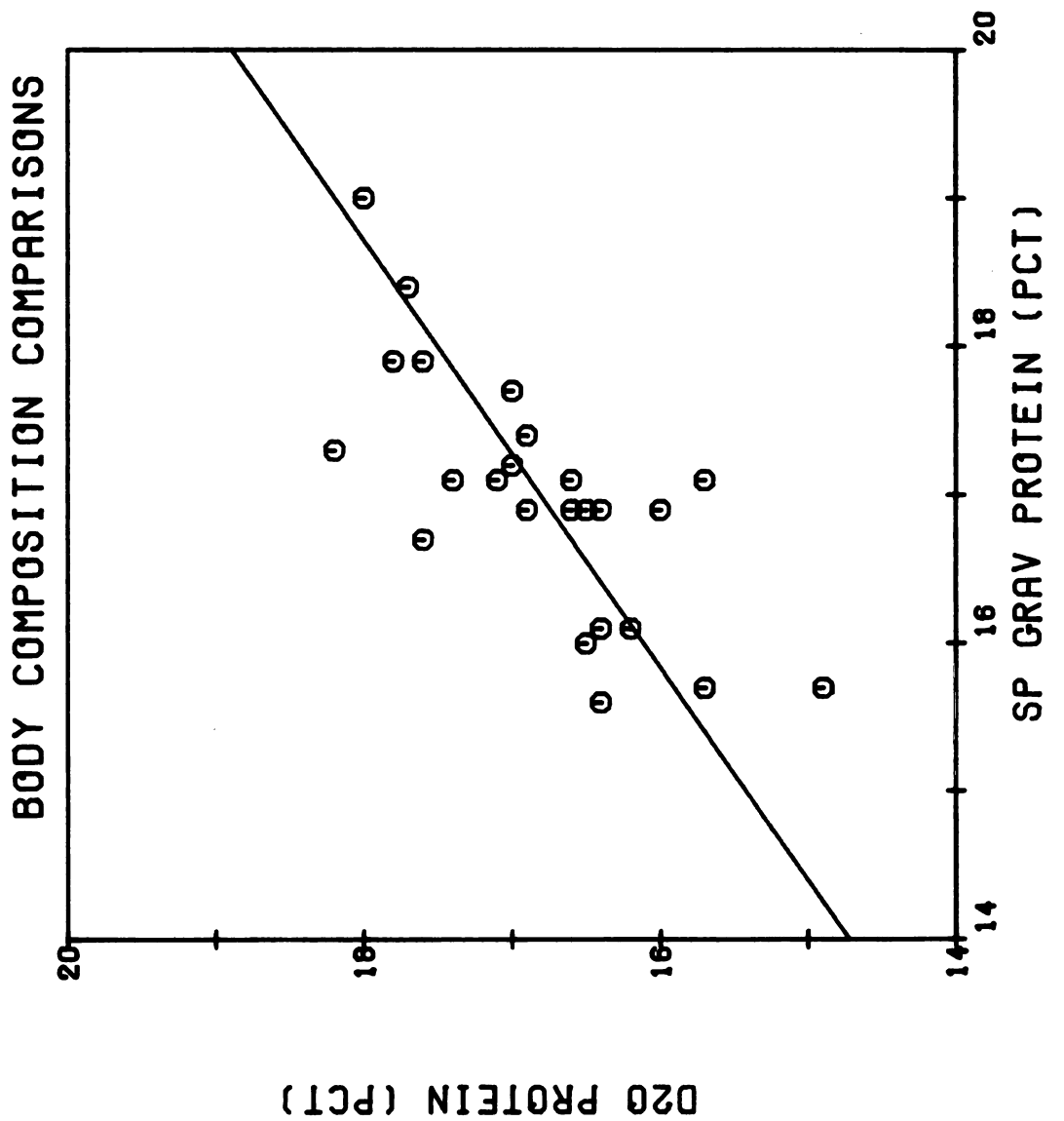
BODY COMPOSITION COMPARISONS
D20 PROTEIN (PCT)
SP GRAV PROTEIN (PCT)
20
18
16
14
20
18
16
14

Figure 8.

Figure 9. Percent Empty Body Fat - Specific Gravity versus D_2O Dilution.

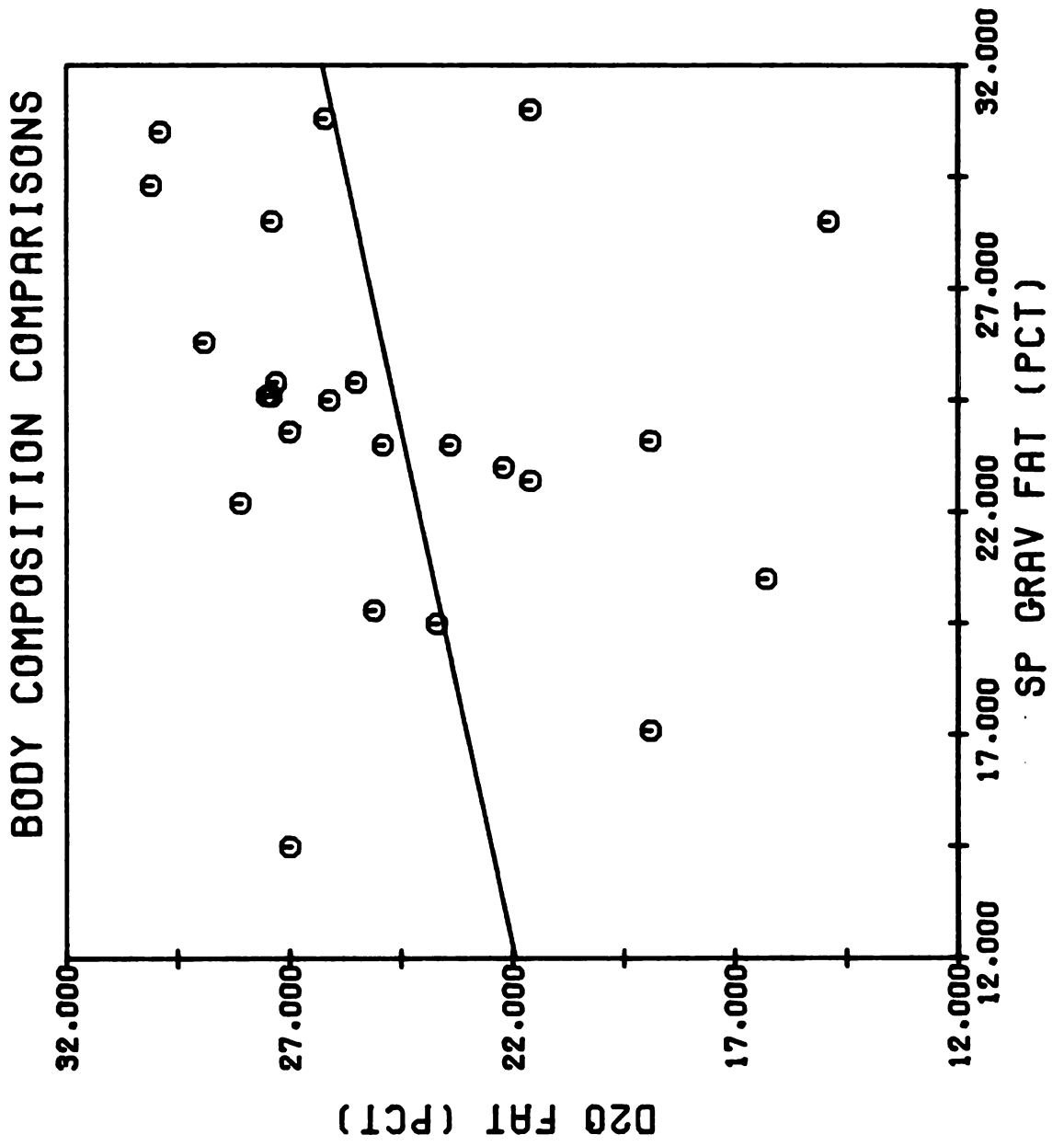
BODY COMPOSITION COMPARISONS
D2O FAT (PCT)
SP GRAV FAT (PCT)
32.000
27.000
22.000
17.000
12.000
12.000
17.000
22.000
27.000
32.000

Figure 9.

Figures 10 and 11 graph the 9-10-11 rib separation estimates against D_2O dilution estimates. Correlation coefficients are high at .72 for % empty body protein and .71 for % empty body fat. The y intercepts and slopes indicate that rib separation estimates higher protein and slightly lower fat contents than the D_2O dilution technique would predict.

The comparison between 9-10-11 rib separation and specific gravity techniques shows a low correlation coefficient Figure 12 illustrates the low value for the slope in the regression equation comparing percent protein in the empty body. The graph would also indicate that the specific gravity method estimates lower % protein values than rib separation. In evaluating the estimations of percent fat in the empty body by the two methods, Figure 13 very graphicaly interprests the regression equation. The wide scatter of points and the low correlation coefficient would indicate a very low relationship between the two techniques for predicting the % empty body fat in the same animal. This conclusion is supported by similar findings in previous unpublished work by the author.

Powell and Huffman (1968), using simple correlation coefficients between the various methods for predicting body composition and chemically determined carcass composition showed that the 9-10-11 rib section tissue separation was most accurate in predicting carcass fat

Figure 10. Percent Empty Body Protein - D_2O Dilution versus Rib Separation.

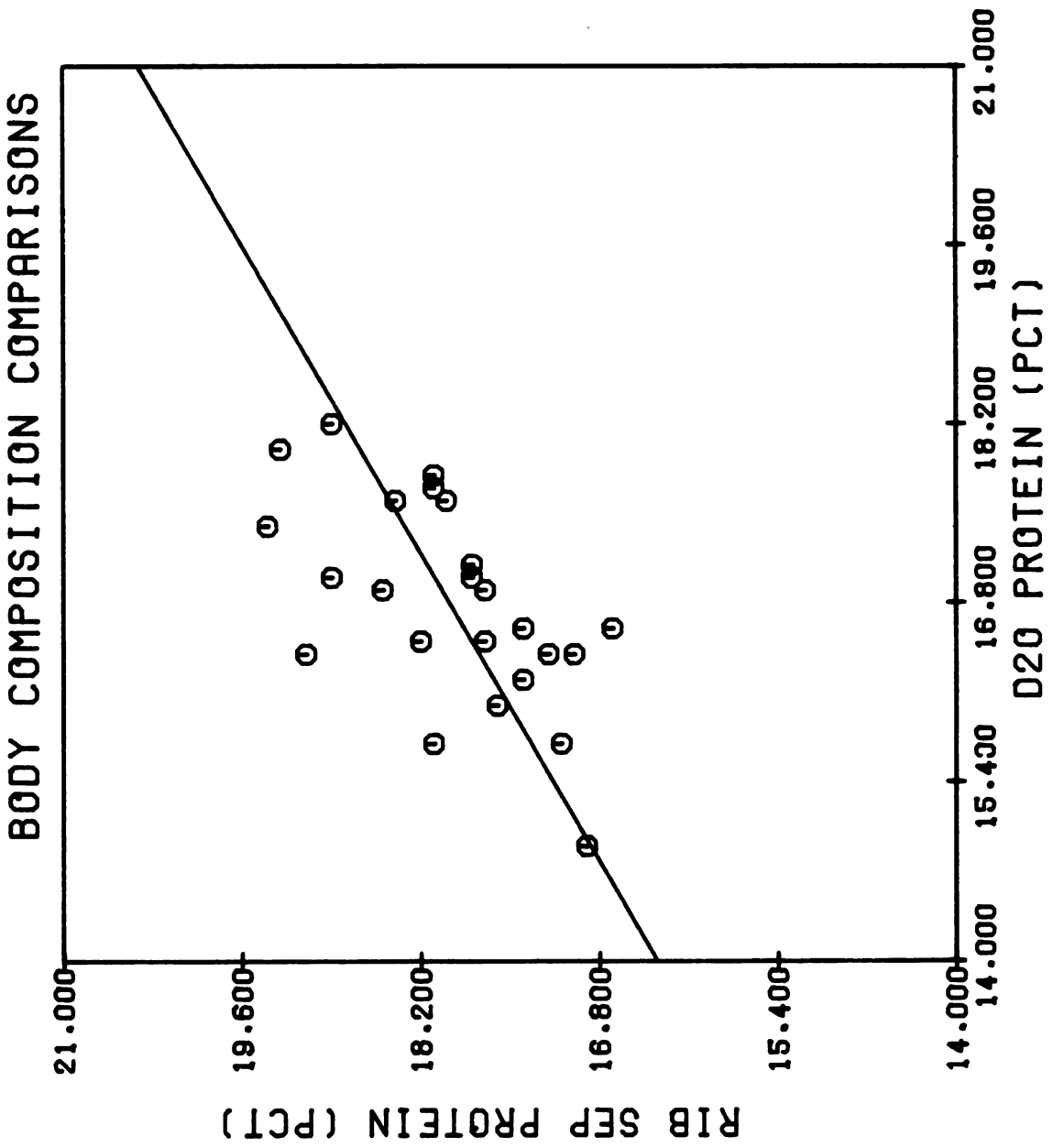
BODY COMPOSITION COMPARISONS
RIB SEP PROTEIN (PCT)
21.000
19.600
18.200
16.800
15.400
14.000
14.000
15.400
16.800
18.200
19.600
21.000
D2O PROTEIN (PCT)

Figure 10.

Figure 11. Percent Empty Body Fat - D_2O Dilution versus Rib Separation.

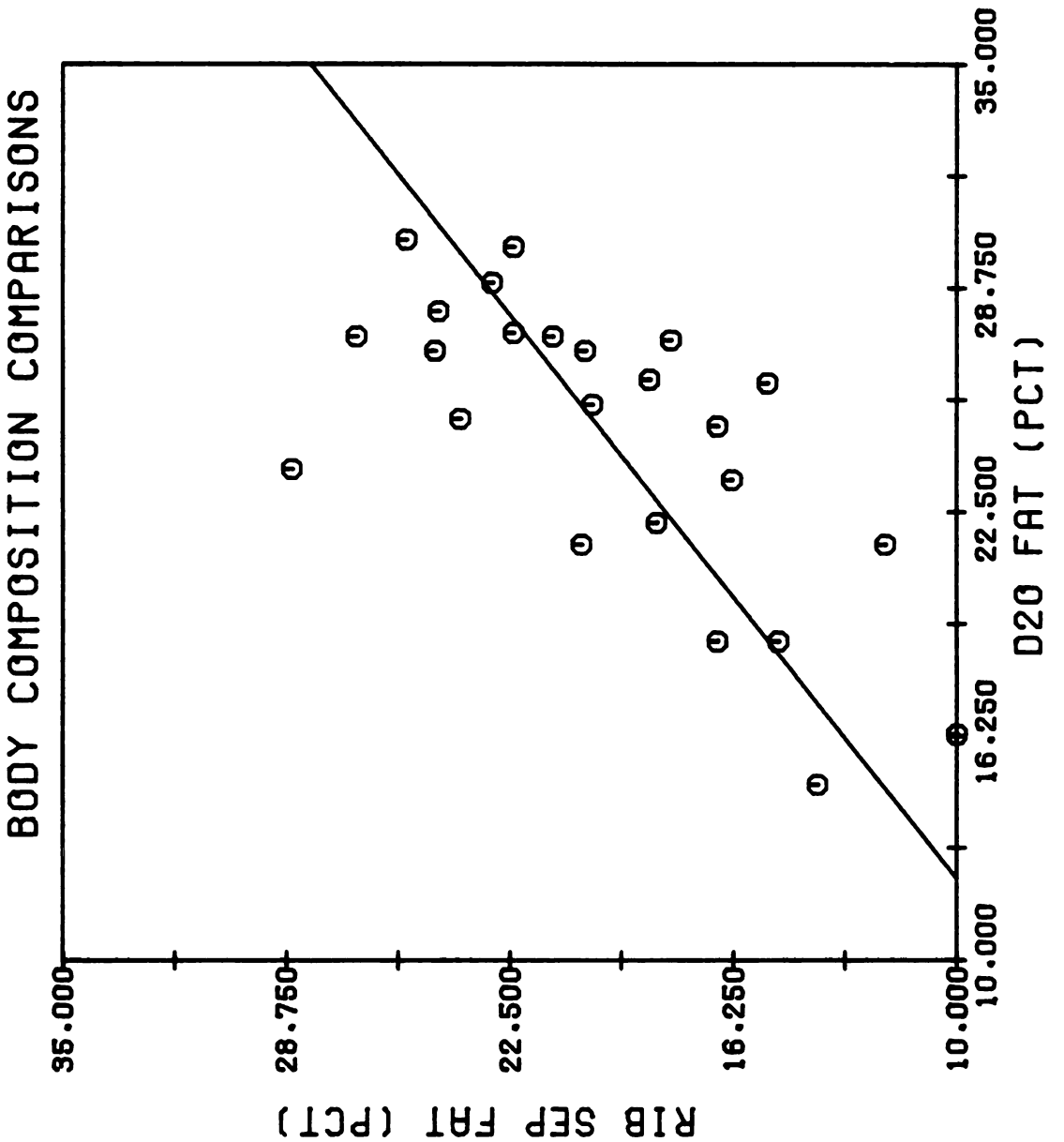
BODY COMPOSITION COMPARISONS
RIB SEP FAT (PCT)
35.000
28.750
22.500
16.250
10.000
10.000
16.250
22.500
28.750
35.000
D2O FAT (PCT)

Figure 11.

Figure 12. Percent Empty Body Protein - Specific Gravity versus Rib Separation.

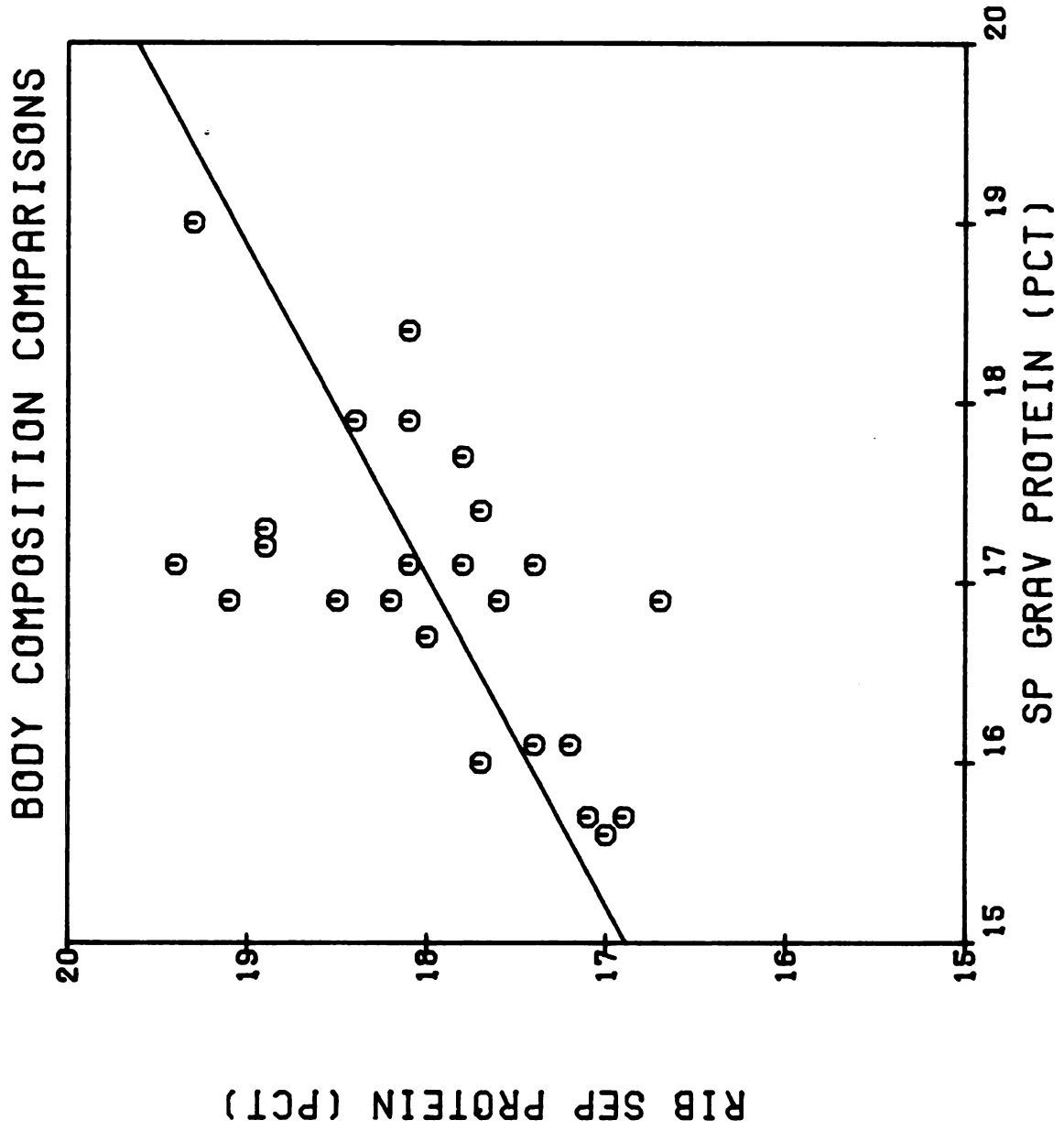
BODY COMPOSITION COMPARISONS
SP GRAV PROTEIN (PCT)
RIB SEP PROTEIN (PCT)
20
19
18
17
16
15

Figure 12.

Figure 13. Percent Empty Body Fat - Specific Gravity versus Rib Separation.

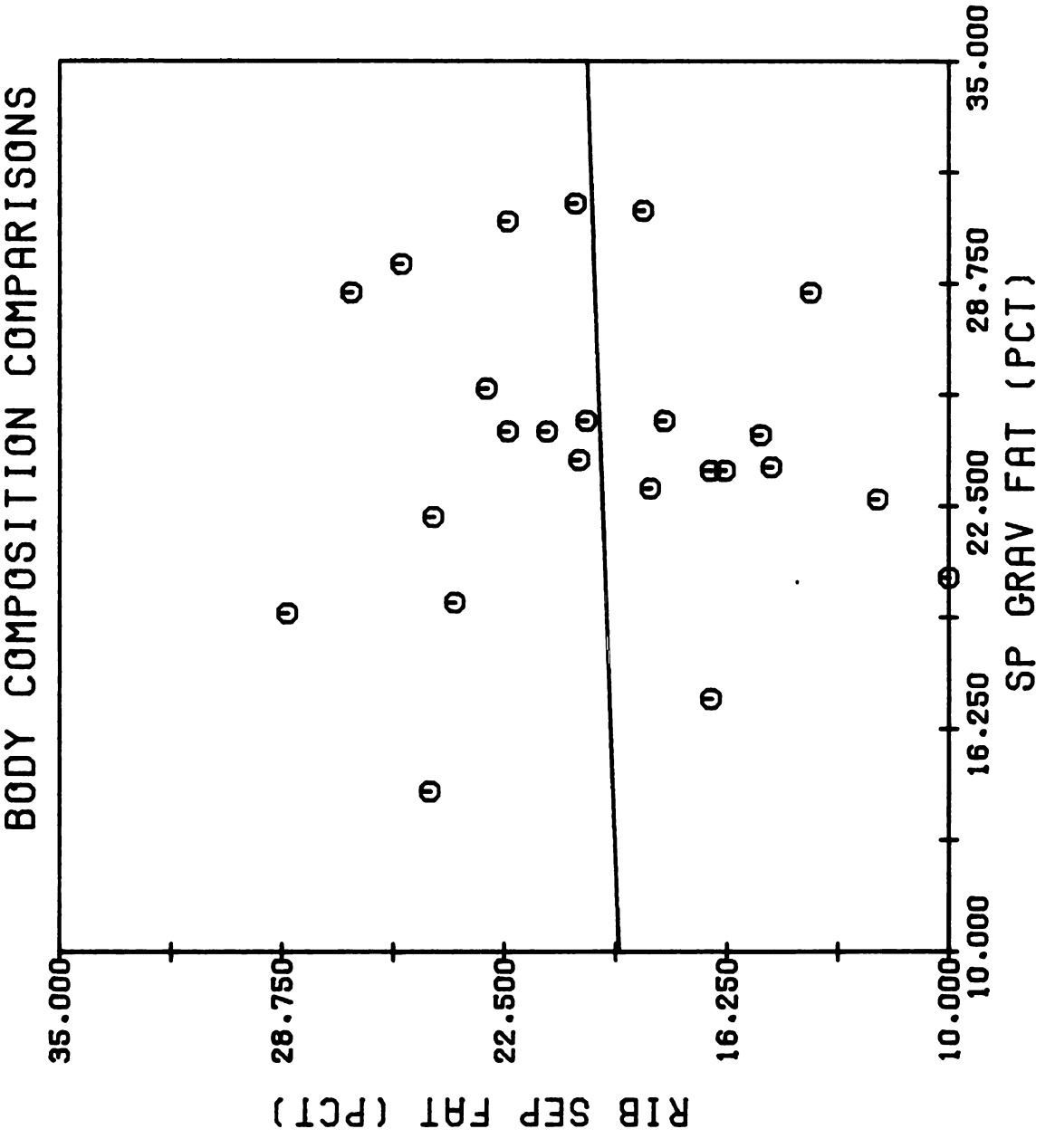
BODY COMPOSITION COMPARISONS
RIB SEP FAT (PCT)
35.000
28.750
22.500
16.250
10.000
10.000
16.250
22.500
28.750
35.000
SP GRAV FAT (PCT)

Figure 13.

(r=.94) and carcass protein (r=-.96). They also indicated that specific gravity was not as good as the rib section method in estimating both fat (r=-.92) or protein (r=.89). Byers (1979) and Garrett and Hinman (1969), however showed a very high correlation between specific gravity and carcass chemical analysis empty body water, which would also indicate accurate protein content predictions.

We found that rib section and specific gravity methods were lowly correlated while rib section and D_2O dilution methods were more highly correlated. This would indicate that if rib section is the most accurate method we used to predict body composition then D_2O dilution ranks second being a more accurate method than specific gravity for the predictions of body composition. It is evident that since the comparisons between the three methods used in this experiment were not highly correlated, mixing of the techniques within a single trial could lead to errors in calculating change in body composition. A single technique should be used and while it may or may not accurately predict body composition, the relative changes over time could still be determined.

The usefulness of the different methods depend on several factors including: want or need for serial kill, discounting of damaged carcasses and equipment and/or facilities to perform each technique properly. Each of the three methods have advantages over the other with the D_2O

dilution method being advantageous in studying growth of cattle when low numbers are necessary and repeated observations are desired on the same animal.

Feeding Trial - Energy Variation

In the past much controversy has arisen surrounding the area of nutritional change in cattle of all frame sizes and their effects on composition of gain. We therefore designed a study to evaluate the effect of various energy levels on the composition of gain in later maturing, large frame cattle.

Feedlot Performance

Summaries of the performance from each energy treatment are reported in Table 6. The performance for each feeding phase and overall are given. In each phase, those cattle fed the high grain ration had the highest rate of gain and the lowest feed requirements per unit of gain. Although the means do not differ significantly between high-low and low-high treatments during the growing phase, the trend is evident for high energy rations to increase average daily gain over low energy rations. Cattle fed the high grain ration throughout the trial had a greater average daily gain for the entire trial ($P < .05$). Cattle fed a high grain ration only during the finishing phase

Table 6. Energy Effect on Feedlot Performance (Trial 1)

	Treatments				
	H-H	H-L	L-L	L-H	SE
Item					
Daily Gain, kg					
Growing	1.35[a]	1.14[ab]	.89[c]	.98[bc]	.148
Finishing	1.11[a]	.84[b]	.94[c]	1.16[a]	.016
Total	1.19[a]	.91[c]	.94[c]	1.08[b]	.016
Average Daily DM Intake, kg					
Growing	6.15	6.41	5.22	5.48	NA
Finishing	6.87	5.76	6.66	7.19	NA
Total	6.70	5.92	6.50	6.85	NA
Feed Efficiency					
Growing	4.64	4.98	5.75	5.64	NA
Finishing	6.19	6.86	7.09	6.20	NA
Total	5.99	6.48	7.03	6.32	NA

[abc]Means within rows with different superscripts differ ($P < .05$).

showed higher average daily gains ($P < .05$) for the total trial than either treatments fed high silage during the finishing phase. These results are consistent with Jesse et al. (1976b), Crickenberger et al. (1978), Danner (1978), and Harpster (1978).

The cattle in the high-low treatment had a negative response to reducing the energy content of their diets. Highlow cattle had the lowest average daily gains (.84 kg/day) during the finishing phase showing similar gains to the low-low treatment cattle for the total experiment (.91 and .94 kg/day respectively).

Body Composition and Carcass Parameters

Empty body compositions, determined by the D_2O dilution technique, of the cattle are shown in Table 7. Cattle for all treatments were infused at similar weights and thus empty body weights show little difference between treatments.

Figure 14 illustrates the changes in empty body water as empty body weight increases. There is little difference in body water content between treatments. However, at a constant empty body weight of 476 kg, treatments receiving high silage during the finishing phase had more empty body water than the high-high treatment fed a high grain ration throughout the trial.

Table 7. Energy Effect on Empty Body Composition, By Period[a] (Trial 1)

Item	Treatments H-H	H-L	L-L	L-H	SE
Empty Body Weight, kg					
Initial	222.82	232.51	222.69	220.92	3.99
Period 1	270.16	269.96	256.32	262.41	4.67
Period 2	313.27	311.74	307.51	319.16	5.88
Period 3	388.58	369.48	364.70	370.11	7.14
Period 4	431.96	408.89	413.26	433.47	6.91
Period 5	476.55	458.21	473.89	476.87	6.96
Empty Body Water, kg					
Initial	136.66	145.95	137.74	140.09	2.40
Period 1	154.04	164.05	153.85	156.17	2.94
Period 2	181.40	181.98	185.68	186.65	3.24
Period 3	211.79	212.08	204.84	218.44	4.90
Period 4	233.16	232.63	228.82	239.14	3.17
Period 5	251.56	253.70	263.18	265.20	3.78
Empty Body Protein, kg					
Initial	41.23	44.03	41.56	42.26	.72
Period 1	46.47	49.49	46.42	47.12	.89
Period 2	54.73	54.90	56.02	56.31	.98
Period 3	63.90	63.99	61.80	65.90	1.20
Period 4	71.41	70.18	69.04	72.15	1.00
Period 5	75.90	76.54	79.40	78.50	1.25
Empty Body Fat, kg					
Initial	35.51	32.46	33.90	28.92	1.74
Period 1	59.03	45.12	45.45	48.36	2.90
Period 2	64.64	62.31	53.02	63.33	2.06
Period 3	98.30	78.80	83.94	70.71	4.59
Period 4	111.43	87.54	99.64	105.71	4.33
Period 5	131.76	110.49	113.17	120.24	4.42

[a]Period 2 is equal to midterm body composition and Period 5 is equal to final body composition.

Figure 14. Energy Effect on Empty Body Water Content.

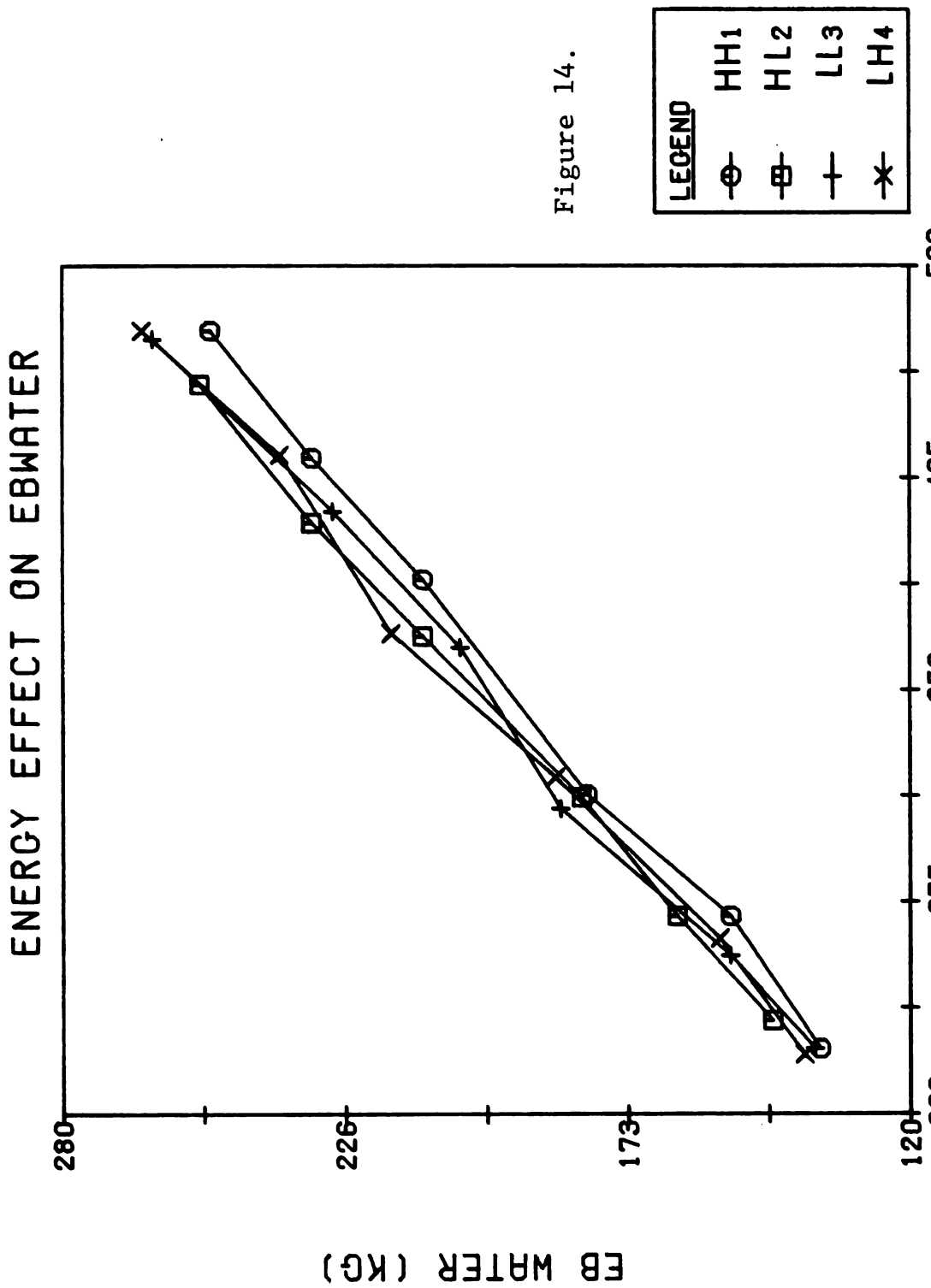
ENERGY EFFECT ON EBWATER
LEGEND
HH1
HL2
LL3
LH4
EB WATER (KG)
280
226
173
120
200
275
350
425
500
EB WEIGHT (KG)

Figure 14.

Figure 15 graphically presents the relationship between empty body protein and empty body weight. This relationship is identical to that of empty body water with empty body weight since empty body protein is calculated from the water. Again, the differences in empty body protein content at given empty body weight are small but, at the termination of the experiment, the treatment receiving a high energy diet throughout the trial had lower protein content than those treatments receiving a low energy diet during the finishing phase. This difference in empty body weight can be attributed in part to differences in days on feed. Cattle fed the high energy diet during the entire trial were on feed 70 days less than treatment group low-low and 51 days less than treatment group highlow. At their rates of protein gain, the treatments low-low and high-low could eventually have a greater amount of empty body protein at the terminal empty body weight and of course less fat.

Figure 16 compares energy treatment effects on quantity of empty body fat at various empty body weights. Cattle fed a high energy diet had more empty body fat present at any empty body weight than those cattle fed a lower energy diet. This is consistent with data from Moulton et al. (1922) and Byers (1980). For the low-high treatment cattle, empty body fat increased dramatically after cattle were switched to the high energy diet.

Figure 13. Energy Effect on Empty Body Protein Content.

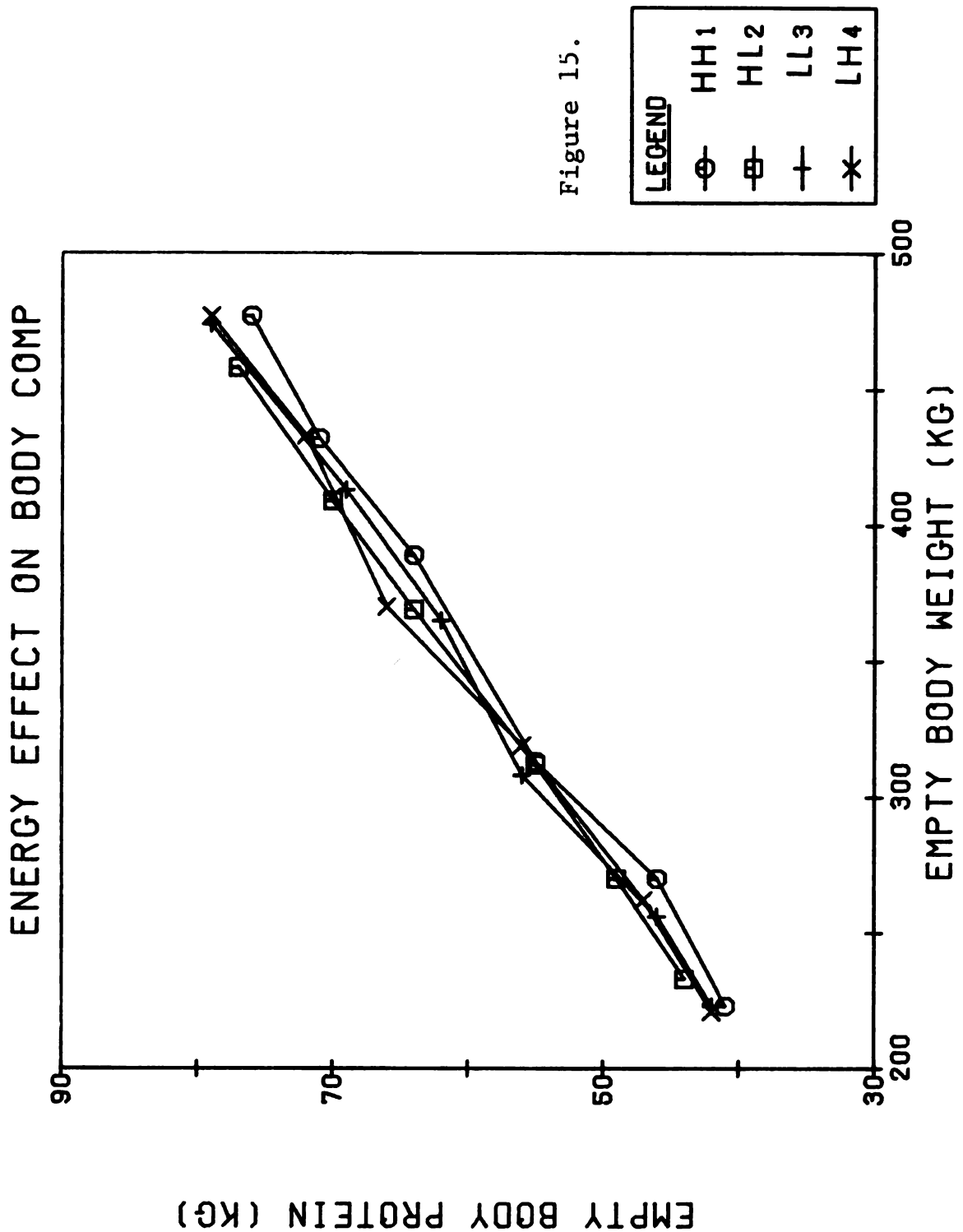
ENERGY EFFECT ON BODY COMP
EMPTY BODY PROTEIN (KG)
EMPTY BODY WEIGHT (KG)
90
70
50
30
200
300
400
500
LEGEND
HH1
HL2
LL3
LH4

Figure 15.

Figure 16. Energy Effect on Empty Body Fat Content.

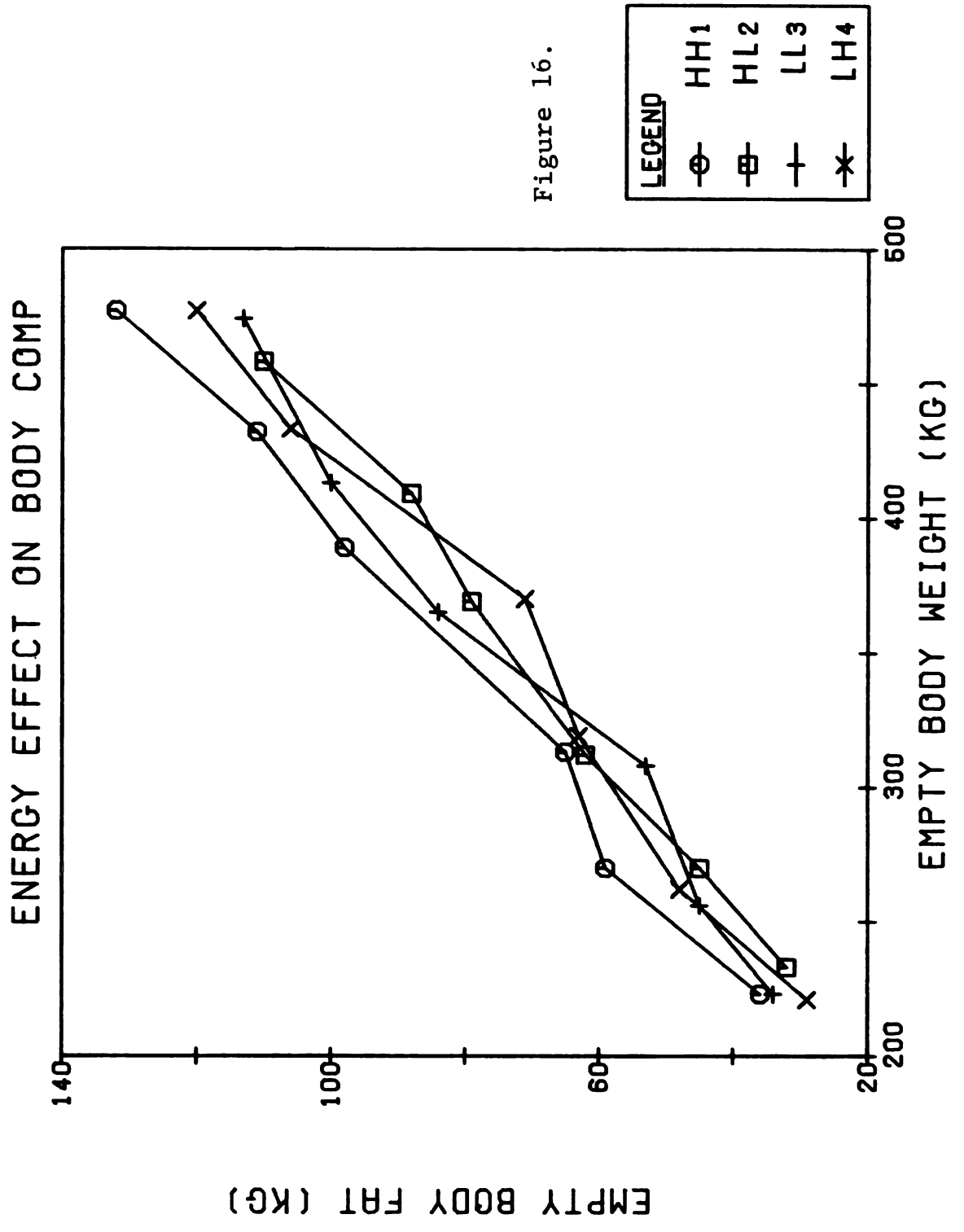
ENERGY EFFECT ON BODY COMP
EMPTY BODY WEIGHT (KG)
EMPTY BODY FAT (KG)
LEGEND
HH1
HL2
LL3
LH4
140
100
60
20
200
300
400
500

Figure 16.

Table 8 summarizes the changes in the percent empty body protein and fat. For all treatments, percentage protein in the empty body decreased with time on feed while percentage fat in the empty body increased from the trial initiation until termination, with the highhigh treatment having higher % empty body fat (27.60%) and lower % empty body protein (15.93%). This would be an expected result of high energy diets and is consistent with Guenther et al. (1965) and Byers and Parker (1979).

Carcass characteristics are presented in Table 9. Energy level had an effect on carcass parameters with high-high and low-high treatments having higher dressing percentages, increased fat thickness, increased kidney pelvic heart fat and increasing yield grades. The high-high treatment had higher quality grades than other energy treatments. This would not be surprising, since higher energy intake has been known to increase fat in the carcass of cattle (Guenther et al., 1965), so one would therefore expect the factors influenced by fat quantity to be higher for treatments on a high energy diet. This data is consistent with and supported by other researchers at this station who determined that at constant weights, high grain fed cattle will have more carcass fat and fat thickness with less of an effect on carcass quality (Crickenberger, 1977; Danner, 1978; Harpster, 1978; and Woody, 1978).

Table 8. Energy Effect on Percentage of Empty Body Protein and Fat (Trial 1)

	Treatments				
	H-H	H-L	L-L	L-H	SE
Item					
Empty Body Protein, %					
Period 1	17.22	18.34	18.14	17.99	.212
Period 2	17.49	17.61	18.24	17.66	.095
Period 3	16.49	17.31	17.02	17.82	.222
Period 4	16.56	17.17	16.76	16.67	.169
Period 5	15.93	16.70	16.76	16.46	.193
Empty Body Fat, %					
Period 1	21.79	16.68	17.61	18.29	.963
Period 2	20.54	20.00	17.14	19.96	.454
Period 3	25.11	21.37	22.67	19.03	1.010
Period 4	25.98	21.37	23.86	24.27	.762
Period 5	27.60	24.06	23.79	25.39	.811

Table 9. Energy Effect on Carcass Characteristics (Trial 1)

	Treatments				
	H-H	H-L	L-L	L-H	SE
Item					
Dressing Percentage	66.03[d]	64.70	62.99	66.13[d]	.266
Adj. Fat Thickness, cm	1.08[d]	.36	.59	.70[d]	.042
Ribeye area, cm^2	94.41	99.25	95.27	99.25	2.197
KPH Fat, %	3.17[d]	2.42	2.33	2.92[d]	.141
Maturity Score[a]	1.67[d]	2.17	2.33	1.50[d]	.121
Marbling Score[b]	10.00	8.17	7.33	8.00	.171
Quality Grade[c]	9.33[d]	8.33	7.67	7.67	.307
Yield Grade	2.50[d]	1.28	1.68	2.07[d]	.104
Hot Carcass Weight, kg	334.76	316.54	326.14	330.67	5.380

[a]Maturity: A-=1; A=2; A+=3.
[b]Marbling score: Slight=8; Slight+=9; Small-=10.
[c]Quality grade: Good-=7; Good=9; Good+=9; Choice-=10.
[d]Means within rows with different superscripts differ significantly ($P < .05$).

Composition of Gain

The effects of energy on daily empty body weight and tissue gains are summarized in Table 10 by period. By evaluating the gains within phases by period, one can observe more subtle changes in rates of empty body gains.

Figure 17 illustrates the changes occuring during the trial, in empty body weight gains. The empty body weights are graphed against days on feed thus, the slopes of the lines are the rates. The graph shows that treatments receiving the high energy diet during the growing phase gained more empty body weight per day than the other treatments. When treatment high-low is switched to a low energy diet, empty body gains decline and are the lowest of all treatments (.69 kg/day). After the switching point at which treatment low-high is changed from a low energy to a high energy diet, an increase in empty body weight gain occurs. This confirms the data of Byers (1980); that suggests that energy levels can alter daily empty body weight gains.

Figures 18 and 19 reflect the changes in daily empty body protein and fat gains between periods. An interesting fact is that an energy effect on protein as well as fat accretion was seen. Energy levels in the diets affect not only fat gains, but also affect protein gains. Patterns are similar for the changes in both fat and protein accretion. Treatment high-high and high-low both have

Table 10. Energy Effect on Daily Empty Body Gains, by Period (Trial 1)

	Treatments				
	H-H	H-L	L-L	L-H	SE
Item					
Empty Body Weight Gains, kg/day					
Period 1	1.35	1.07	.62	.77	.061
Period 2	1.23	1.19	1.06	1.15	.070
Period 3	1.09	.81	.88	1.06	.040
Period 4	.89	.64	.78	1.26	.067
Period 5	1.06	.63	.85	1.06	.079
Empty Body Water Gains, g/day					
Period 1	496.46	516.88	298.35	297.82	2.211
Period 2	781.78	512.56	663.01	622.09	2.543
Period 3	440.44	423.92	294.86	662.26	1.362
Period 4	436.06	331.40	386.76	413.96	1.708
Period 5	438.10	270.13	483.96	635.72	2.275
Empty Body Protein Gains, g/day					
Period 1	149.79	155.94	90.01	89.85	.667
Period 2	235.86	154.64	200.03	187.68	.767
Period 3	132.88	127.90	88.90	199.81	.411
Period 4	153.34	99.99	116.68	124.89	.443
Period 5	106.76	81.49	146.01	155.00	.580
Empty Body Fat Gains, g/day					
Period 1	672.09	361.60	213.99	360.09	.655
Period 2	160.44	491.11	157.76	305.49	.607
Period 3	487.79	232.31	475.63	153.78	1.438
Period 4	267.91	140.96	253.24	699.84	1.533
Period 5	484.12	294.18	190.61	354.39	3.862

Figure 17. Energy Effect on Empty Body Weight Gain.

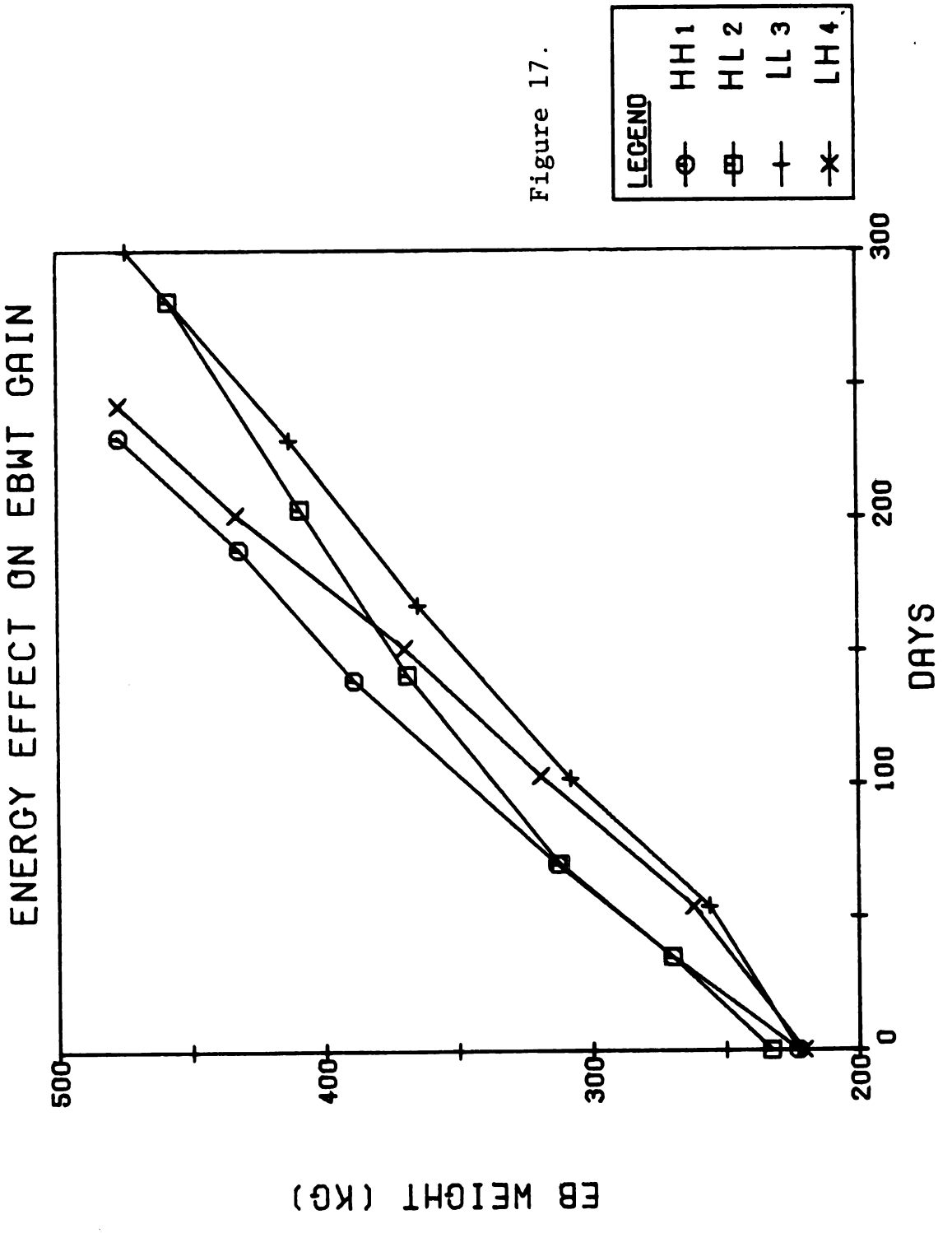
ENERGY EFFECT ON EBWT GAIN
EB WEIGHT (KG)
500
400
300
200
0
100
200
300
DAYS
LEGEND
HH 1
HL 2
LL 3
LH 4

Figure 17.

Figure 18. Energy Effect on Empty Body Protein Gain.

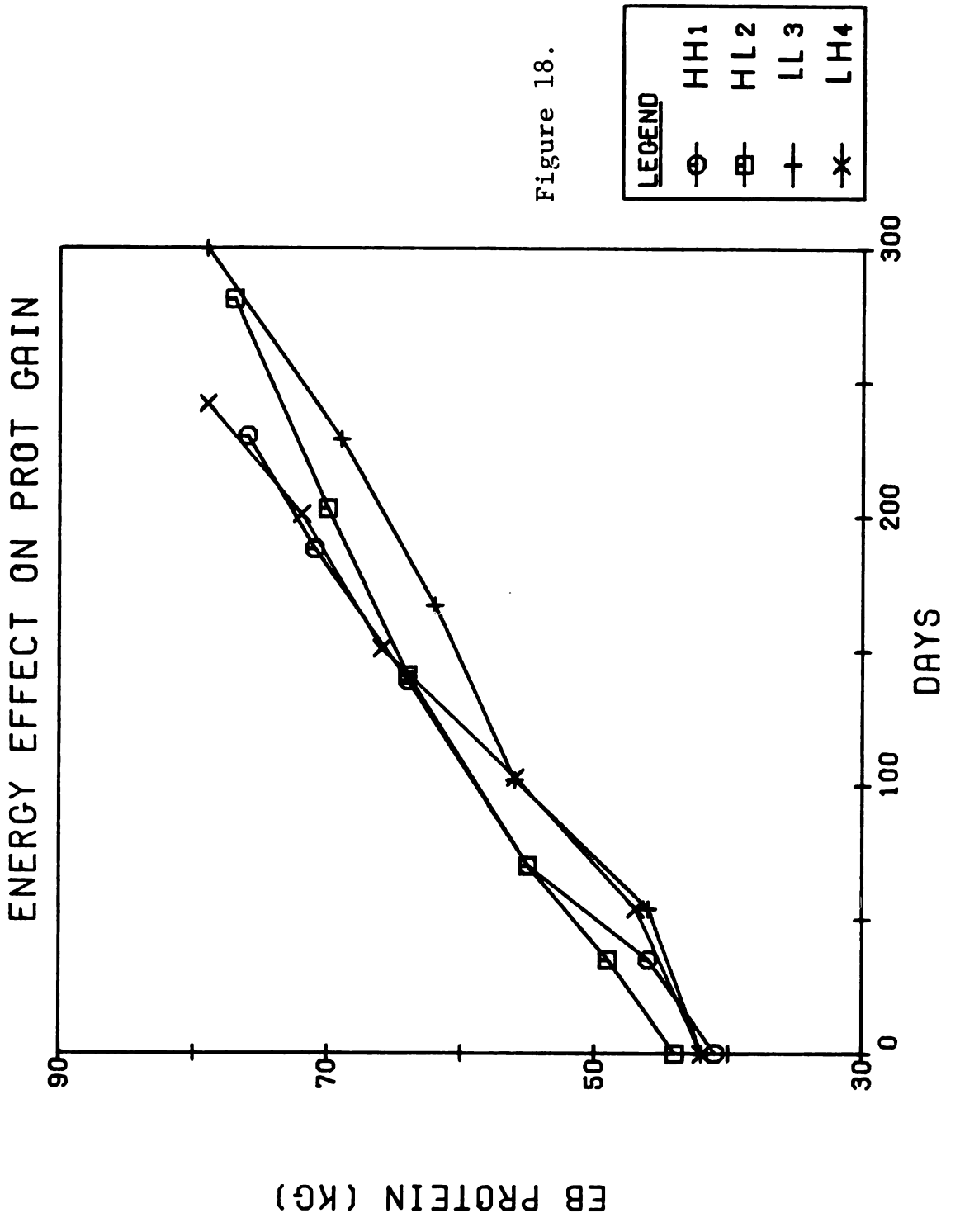
ENERGY EFFECT ON PROT GAIN
LEGEND
HH1
HL2
LL3
LH4
EB PROTEIN (KG)
DAYS
90
70
50
30
0
100
200
300

Figure 18.

Figure 19. Energy Effect on Empty Body Fat Gain.

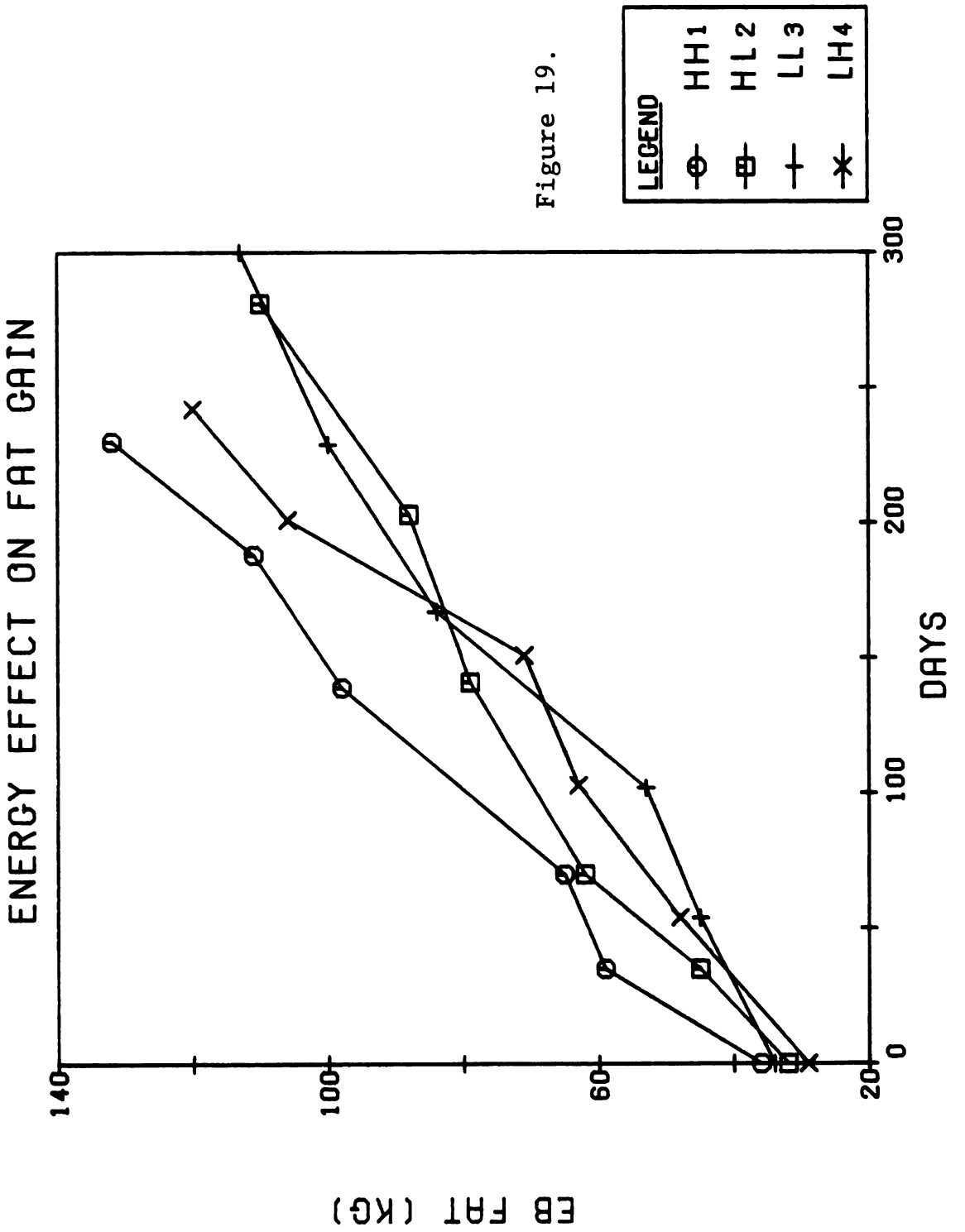
ENERGY EFFECT ON FAT GAIN
EB FAT (KG)
DAYS
LEGEND
HH1
HL2
LL3
LH4
140
100
60
20
0
100
200
300

Figure 19.

higher protein and fat gains during the growing phase, than treatments fed the low energy diet. Beyond the mid-term switching point a change occurs in the rates of tissue accretion for both high-low and low-high treatments. Treatment low-high has increased fat and protein accretion rates to equal and slightly exceed the rates shown by treatment high-high. This effect is mirrored by the high-low treatment, showing decreased rates of fat and protein gain at or below rates for the group of cattle fed the high silage diet the entire trial.

Table 11 shows the treatment effects on empty body gains, dividing the trial by phases. Cattle fed the high energy diet during either the growing or the finishing phase showed greater daily empty body weight gains than low energy treatments during those phases. The treatments high-high and low-high had greater daily empty body weight gains over the entire trial.

Daily empty body water and empty body protein gains show patterns of proportional change since one is calculated from the other. Figure 20 graphically summarizes the changes in daily empty body protein gains between phases. The high energy treatments have higher daily protein gain during both the growing and the finishing phases. Treatment high-low does not differ significantly from low-low but a strong trend is shown in

Table 11. Energy Effect on Daily Empty Body Gains, by Phase (Trial 1)

	Treatments				
	H-H	H-L	L-L	L-H	SE
Item					
Empty Body Weight Gains, kg/day					
Growing	1.29[a]	1.13[a]	.83[b]	.95[b]	.040
Finishing	1.02[a]	.69[b]	.84[c]	1.13[a]	.021
Total	1.10[a]	.80[b]	.84[b]	1.06[a]	.019
Empty Body Water Gains, g/day					
Growing	639.12[a]	514.72[ab]	469.95[bc]	452.08[c]	.024
Finishing	438.48[a]	339.88[b]	391.44[b]	565.12[a]	.018
Total	499.55[a]	383.43[b]	418.14[b]	517.01[a]	.014
Empty Body Protein Gains, g/day					
Growing	192.82[a]	155.29[ab]	141.79[bc]	136.39[c]	.006
Finishing	132.29[a]	102.54[b]	118.10[b]	159.64[a]	.005
Total	150.71[a]	115.68[b]	126.15[b]	149.75[a]	.004
Empty Body Fat Gains, g/day					
Growing	416.26[a]	426.35[a]	187.53[b]	334.11[a]	.023
Finishing	419.49[a]	228.34[b]	303.79[bc]	409.38[ac]	.019
Total	418.51[a]	277.67[b]	264.26[b]	377.35[a]	.016

[abc]Means within rows with different superscripts differ ($P < .05$).

Figure 20. Energy Effect on Daily Protein Gain.

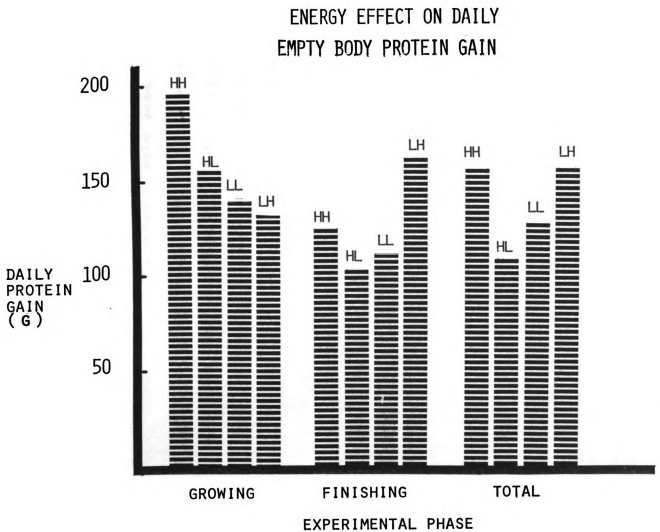
ENERGY EFFECT ON DAILY
EMPTY BODY PROTEIN GAIN
200
150
100
50
DAILY PROTEIN GAIN (G)
HH
HL
LL
LH
HH
HL
LL
LH
HH
HL
LL
LH
GROWING
FINISHING
TOTAL
EXPERIMENTAL PHASE

Figure 20.

favor of higher gains. The treatments that received a high energy diet during at least the finishing phase showed the highest rates of protein gain during the entire trial. A compensatory effect in protein gain does occur for treatment low-high (159.64 g/day) as compared to treatment high-high (132.29 g/day). Although not significant, daily protein gain is higher than that of treatment high-high during the finishing phase.

Figure 21 illustrates the differences in daily fat gains between treatments during the two phases and the total experiment. Energy levels have a marked effect on fat gains with treatments receiving the high energy diets gaining more fat per day than the low energy treatments in both phases. Treatment low-high was not significantly different than the two high energy treatments during the growing phase even though the rate was 80 g/day less. The same situation occurred with the low-low and the low-high treatments during the finishing phase.

Tables 12 and 13 show the effects of energy on empty body tissue gains as a percent of empty body weight gains. Although none were statistically different, cattle fed a high plane of nutrition during an entire feeding trial tended to show decreasing percentages of protein gain and increased percentages of fat gain. This would be in agreement with data from Byers (1979). Cattle fed on a high plane of nutrition during the growing phase then switched

Figure 21. Energy Effect on Daily Fat Gain.

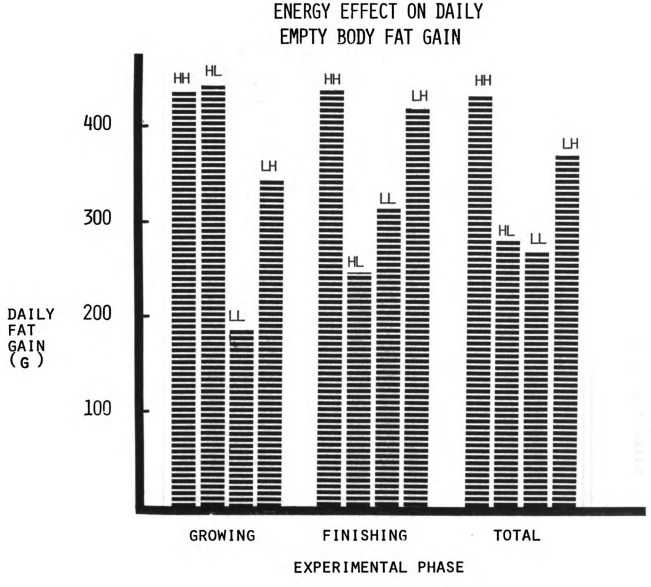
ENERGY EFFECT ON DAILY
EMPTY BODY FAT GAIN
DAILY
FAT
GAIN
(G)
400
300
200
100
HH
HL
LL
LH
HH
HL
LL
LH
HH
HL
LL
LH
GROWING
FINISHING
TOTAL
EXPERIMENTAL PHASE

Figure 21.

Table 12. Energy Effect on Empty Body Tissue Gains as Percentages of Empty Body Weight Gains by Period (Trial 1)

	Treatments				
	H-H	H-L	L-L	L-H	SE
Item					
Empty Body Protein Gains as % of Empty Body Weight Gains					
Period 1	8.22	14.10	14.25	11.79	1.974
Period 2	17.20	12.88	18.61	14.90	.891
Period 3	11.91	15.26	10.40	18.76	1.079
Period 4	9.81	13.98	12.35	15.65	2.335
Period 5	8.47	13.77	16.46	13.95	2.285
Empty Body Fat Gains as % of Empty Body Weight Gains					
Period 1	62.64	35.93	35.26	46.42	8.969
Period 2	21.86	41.48	15.48	27.78	4.048
Period 3	45.91	30.69	52.73	14.78	4.903
Period 4	50.76	42.42	32.46	26.99	5.413
Period 5	56.77	44.02	25.20	36.62	10.224

Table 13. Energy Effect on Empty Body Tissue Gains as Percentages of Empty Body Weight Gains by Phase (Trial 1)

	Treatments				
	H-H	H-L	L-L	L-H	SE
Item					
Empty Body Protein Gains as % of Empty Body Weight Gains					
Growing	14.87	13.72	17.14	14.38	.435
Finishing	12.97	14.83	13.95	14.03	.460
Total	13.68	14.41	15.03	14.16	.334
Empty Body Fat Gains as % of Empty Body Weight Gains					
Growing	32.43	37.65	22.13	34.69	1.974
Finishing	41.07	32.63	36.60	36.24	2.091
Total	37.86	34.55	31.73	35.68	1.519

to a low energy ration showed a reverse effect, actually increasing in the percent of empty body weight gain attributed to protein and decreasing fat contribution to empty body gains. Cattle in treatment low-low showed a pattern similar to high-high. However, the percentage of empty body gain as protein is higher and the fat percentage is lower. Treatment low-high follows a similar pattern of decreasing the protein portion of the empty body gain and increasing the fat.

From the data presented in this section it is clear that energy has an effect on carcass traits, empty body weight gains and empty body tissue gains. Our results do not agree entirely with the theory; that the maximum for protein and muscle growth is genetically set, thus energy intake above needs for protein deposition is stored as fat. This may be the case in part if the maximum rate of protein deposition is attained, but the maximum rate has not been determined. According to Woody (1978), Garrett (1979), and Byers (1980), the daily protein gain is maximized at approximately 1.0 kg daily empty body weight gain for English type breeds. Their daily protein gains were then maximized at 140 g/day. Figure 22 shows that in this trial with exotic cattle a rate of nearly 200 g/day protein gain was attained, per kg/day empty body weight gain. Our data does not show a plateau or maximum daily protein gain that Byers (1980) has reported, however we did observe a

Figure 22. Energy Effect on Protein Content of Gain.

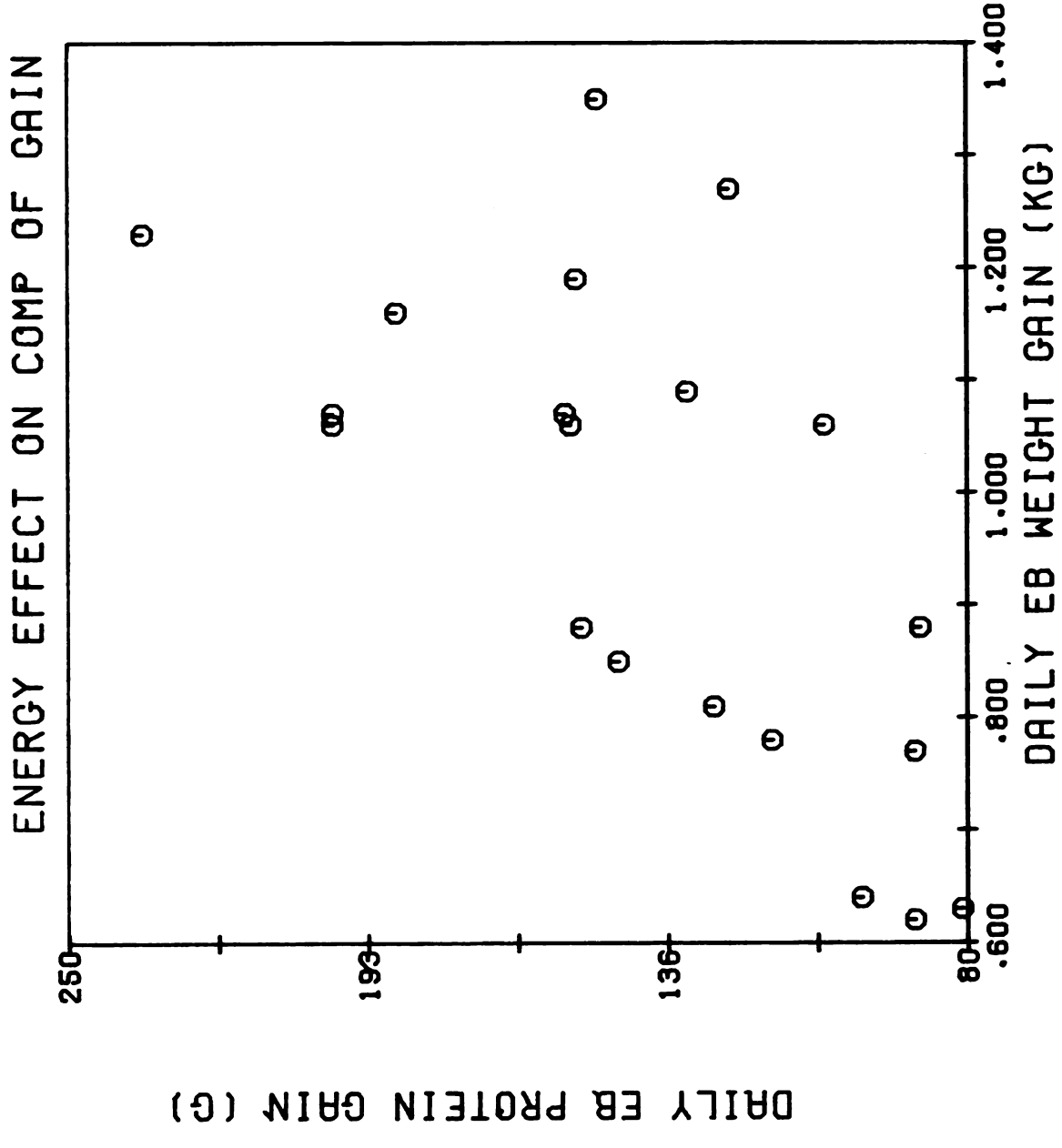
ENERGY EFFECT ON COMP OF GAIN
DAILY EB PROTEIN GAIN (G)
250
193
136
80
.600
.800
1.000
1.200
1.400
DAILY EB WEIGHT GAIN (KG)

Figure 22.

deceleration in the rate of protein gain when daily empty body weight gains were between 1.0 and 1.2 kg/day indicating a biological limit for daily protein growth was being approached.

Figure 23 illustrates the relationship of daily empty body weight gains with daily empty body fat gains. This graph shows that at the point where the rate of protein gain begins to decelerate, the rate of fat gain accelerates. From these two graphs one could project that for later maturing breeds of cattle gaining approximately 1.1 kg/day empty body weight, animals maximize protein gain yet minimize the fat gain. After that point a greater percentage of empty body weight gain will be fat. At empty body gains as low as 0.6 kg/day, fat is being deposited along with protein. This would indicate that at any gain in empty body weight, both protein and fat are being deposited. This would not support the theory of energy in excess of that required for protein deposition is used to deposit fat. High energy groups and the low energy groups deposited fat at high rates even during the growing phase with young cattle. And, at the same time both energy levels deposited over 100 g/day empty body protein. From this it appears that energy is being partitioned between protein and fat synthesis. There does not seem to be a clear cut energy priority from protein synthesis, rather at any energy intake level, a portion is used for protein gain and a portion for fat gain.

Figure 23. Energy Effect on Fat Content of Gain.

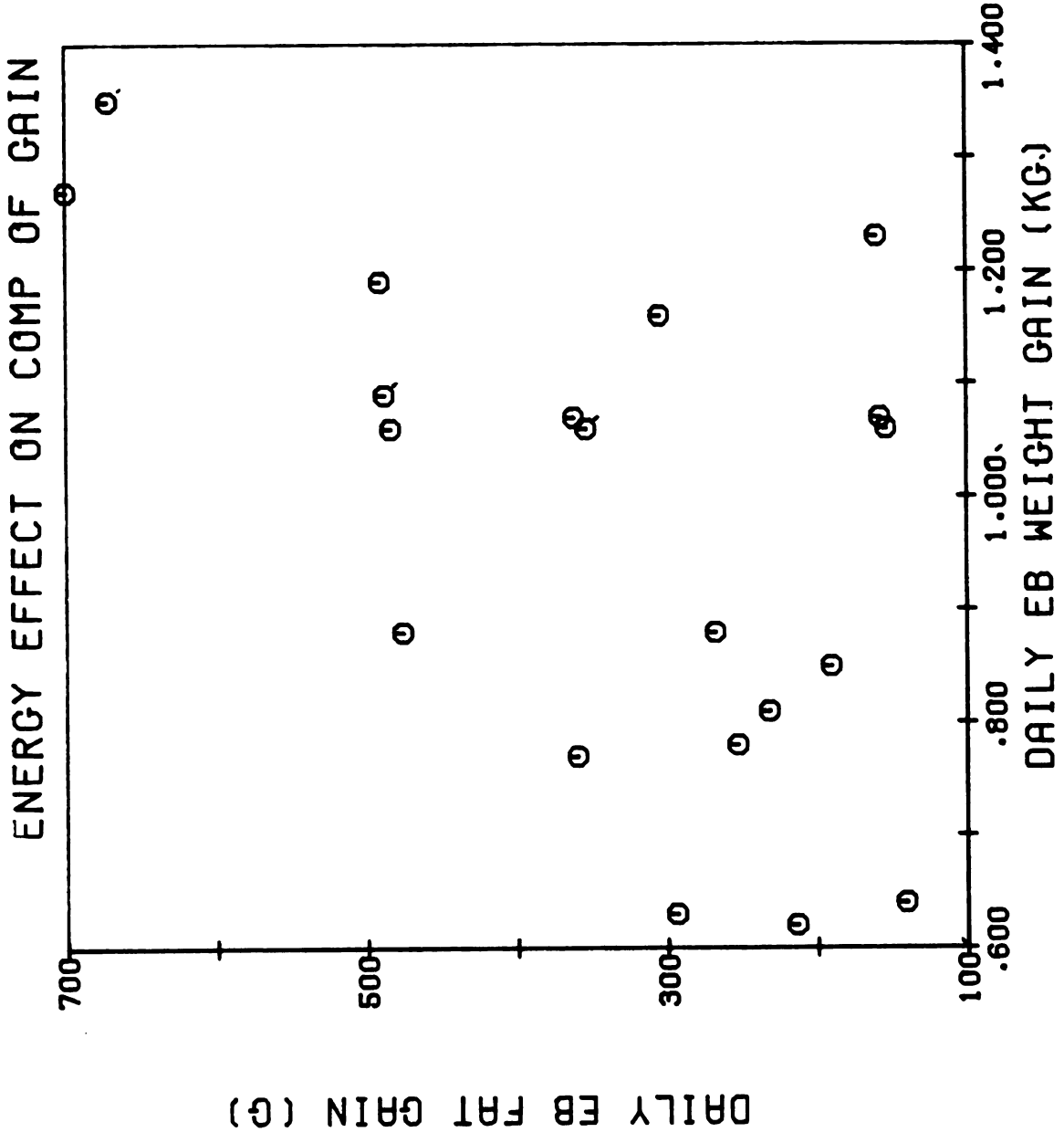
ENERGY EFFECT ON COMP OF GAIN
DAILY EB FAT GAIN (G)
700
500
300
100
.600
.800
1.000
1.200
1.400
DAILY EB WEIGHT GAIN (KG)

Figure 23.

Our observation that composition of gain can be altered by energy levels has not been a consistent observation as indicated in reviews by Marchello and Hale (1976), and Reid et al. (1968). Prior et al. (1977) did show changes in carcass composition with changing energy levels. Their results indicated that the changes in composition were due largely to a greater amount of fat being deposited. Our data shows that the changes in composition were not due just to increased fat with higher energy diets but were also due to an increased protein deposition.

Feeding Trial - Frame Size Variation

A growing of interest in the cattle feeding industry has centered around large frame cattle in the feedlot. The question to be answered is: Do large frame cattle have advantages over small cattle in both protein and fat gain or do large frame cattle gain in very similar patterns to that of small frame cattle. To test this contention, a trial was designed to evaluate the differences in performance and composition of gain for large and small framed cattle.

Animal Performance

Summaries of the performance differences between cattle of two frame sizes are shown in Table 14. The performance for both feeding phases and the overall performance are given. In the growing phase large frame cattle gained more body weight per day and, consumed more dry matter each day than small frame cattle. There was no difference in average daily gains between treatments during the finishing phase. However, small frame cattle consumed less dry matter daily than the large frame cattle. Large frame cattle gained more body weight per day over the total trial than small frame cattle, but the small frame cattle consumed less dry matter each day. Feed efficiency was not different between treatments during either feeding phase and over the total trial. Even though large frame cattle gained more weight per day than small frame cattle they also consumed more feed thus, their feed efficiency was no different from small frame cattle. This is confirmed by Dinius et al. (1976), Prior et al. (1977), and Harpster (1978).

Body Composition and Carcass Parameters

The effects of frame size on empty body composition are shown in Table 15. Cattle used for the large frame treatment were heavier than cattle used for the small frame treatment. This would be difficult to change since the

Table 14. Frame Size Effect on Feeding Performance (Trials 2 and 3)

	Treatments		
	Small	Large	SE
Item			
Daily Gain, kg			
Growing	1.17[a]	1.42[b]	.052
Finishing	.89	1.06	.042
Total	.99[a]	1.20[b]	.039
Average Daily DM Intake, kg			
Growing	6.71[c]	8.13[d]	.238
Finishing	7.47[c]	9.67[d]	.335
Total	7.15[c]	9.10[d]	.260
Feed Efficiency			
Growing	5.71	5.86	.150
Finishing	8.85	9.11	.372
Total	7.36	7.59	.230

[ab]Means within rows with different superscripts differ ($P < .05$).

[cd]Means within rows with different superscripts differ ($P < .01$).

Table 15. Frame Size Effect on Empty Body Composition (Trials 2 and 3)

Item	Treatments Small	Large	SE
Empty Body Weight, kg			
Initial	235.77[a]	267.85[b]	7.307
Midterm	305.95[c]	356.03[d]	8.543
Final	371.56[c]	445.63[d]	9.366
Empty Body Water, kg			
Initial	120.93[c]	149.91[d]	4.387
Midterm	151.22[c]	193.23[d]	4.870
Final	176.26[c]	234.88[d]	5.627
Empty Body Protein, kg			
Initial	36.49[c]	45.23[d]	1.323
Midterm	45.62[c]	58.30[d]	1.469
Final	53.18[c]	70.86[d]	1.698
Empty Body Fat, kg			
Initial	69.79	62.35	4.239
Midterm	98.69	91.19	3.368
Final	129.98	123.71	3.127
Live Body Weight, kg			
Initial	259.50[a]	301.36[b]	8.016
Midterm	340.31[c]	399.05[d]	8.639
Final	413.70[c]	503.97[d]	10.359

[ab]Means within rows with different superscripts differ ($P < .05$).

[cd]Means within rows with different superscripts differ ($P < .01$).

larger cattle generally wean at heavier weights than small frame cattle. Since the large frame cattle were heavier initially, their empty body weights were also greater than small frame cattle. Thus at initial, midterm and final live weights and empty body weights, large frame cattle were significantly heavier.

Figure 24 illustrates the frame effect on empty body water. Large frame cattle had greater amounts of empty body water at all three infusion times and since empty body protein is directly calculated from empty body water we found that large frame cattle had greater amounts of empty body protein than small frame cattle. These relationships are graphically shown in Figure 25. Not surprising, is the fact that there was no difference in absolute amounts of empty body fat between treatments. Figure 26 shows that at any given empty body weight, small framed cattle are fatter. This means that large frame cattle were leaner at any given empty body weight. This is consistent with work reviewed by Berg and Butterfield (1976) and by Prior et al. (1977).

Carcass characteristics are presented in Table 16. There was a frame size effect on some carcass parameters. Small frame cattle had increased fat thickness, less rib eye area and higher yield grades than large frame cattle. There was no difference in quality grades between

Figure 24. Frame Effect on Empty Body Water Content.

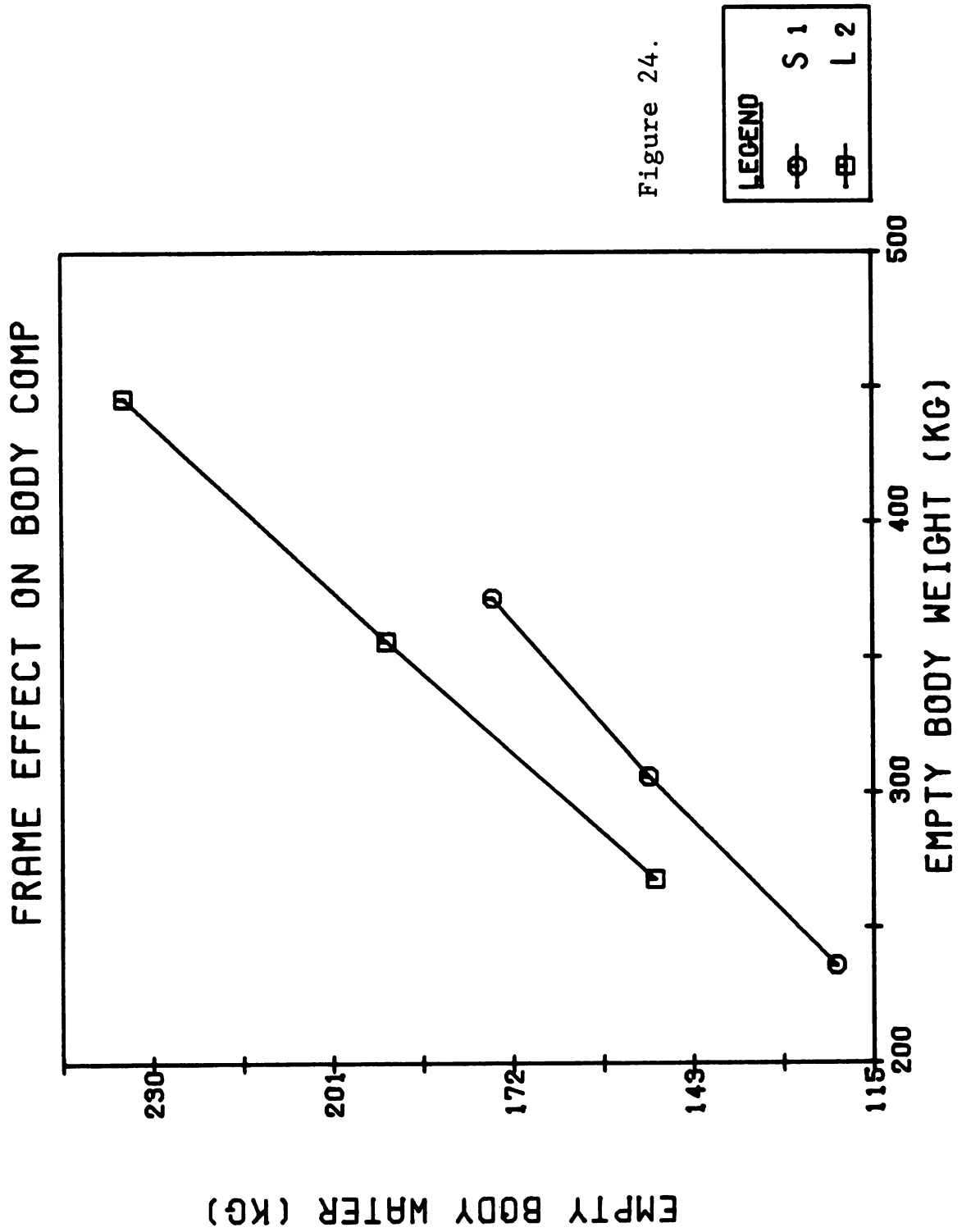
FRAME EFFECT ON BODY COMP
EMPTY BODY WATER (KG)
230
201
172
143
115
200
300
400
500
EMPTY BODY WEIGHT (KG)
LEGEND
S 1
L 2

Figure 24.

Figure 25. Frame Effect on Empty Body Protein Content.

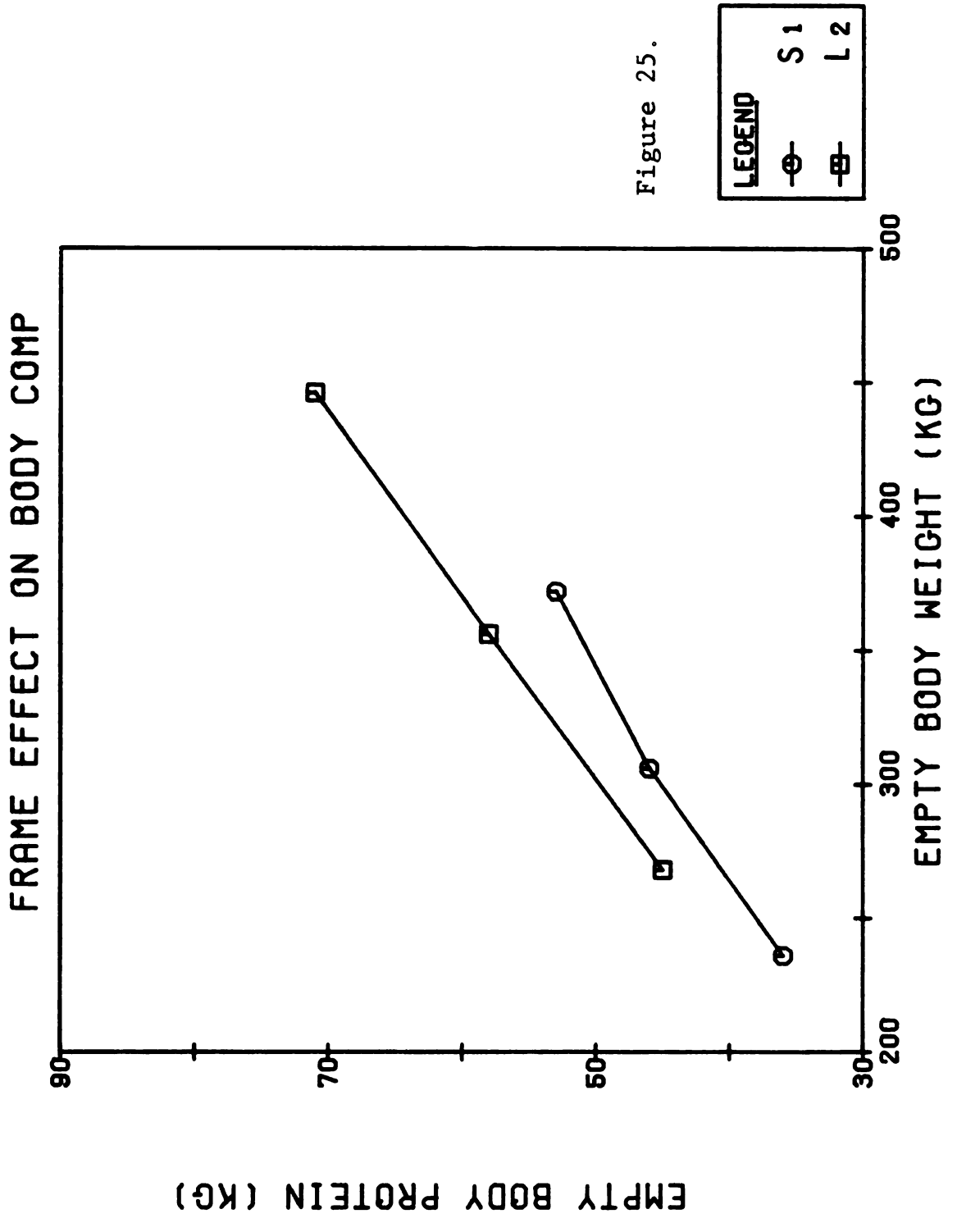
FRAME EFFECT ON BODY COMP
LEGEND
S 1
L 2
EMPTY BODY PROTEIN (KG)
90
70
50
30
200
300
400
500
EMPTY BODY WEIGHT (KG)

Figure 25.

Figure 26. Frame Effect on Empty Body Fat Content.

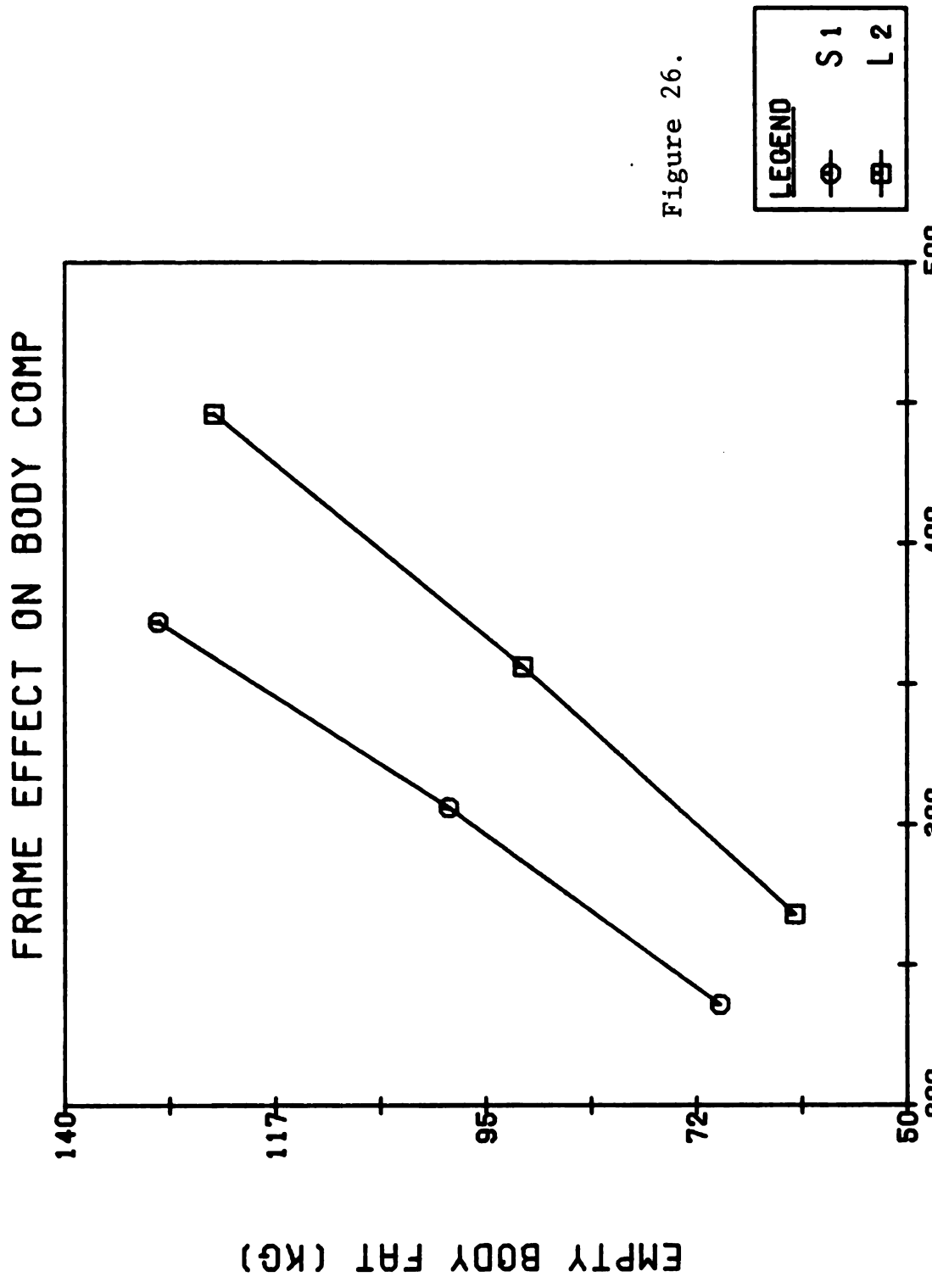
FRAME EFFECT ON BODY COMP
EMPTY BODY FAT (KG)
140
117
95
72
50
200
300
400
500
EMPTY BODY WEIGHT (KG)
LEGEND
S 1
L 2

Figure 26.

Table 16. Frame Size Effect on Carcass Characteristics (Trials 2 and 3)

	Treatments		
	Small	Large	SE
Item			
Adj. Fat Thickness, cm	1.43[c]	.72[d]	.074
Ribeye Area, cm^2	65.14[a]	84.88[b]	4.510
Quality Grade	8.50	7.80	.479
Yield Grade	3.26[c]	2.41[d]	.132
Empty Body Protein, %			
Initial	15.38[a]	16.76[b]	.271
Midterm	14.80[c]	16.32[d]	.175
Final	14.24[c]	15.89[d]	.177
Empty Body Fat, %			
Initial	30.11[a]	23.86[b]	1.233
Midterm	32.75[c]	25.87[d]	.795
Final	35.61[a]	30.39[b]	1.234

[ab]Means within rows with different superscripts differ ($P < .05$).

[cd]Means within rows with different superscripts differ ($P < .01$).

treatments and as would be expected from body composition data, the large frame cattle had a higher percentage of empty body weight as empty body protein and a lower percentage empty body fat than small frame cattle.

Composition of Gain

The effect of frame size on daily empty body weight and empty body tissue gains are summarized in Table 17. Large frame cattle had greater empty body weight gains during the growing phase and over the total trial. Although not statistically significant, daily empty body weight gain tended to be greater for large frame cattle during the finishing phase. This would coincide with the similar differences in average daily gains during the same periods. Daily empty body water gains and daily empty body proteins gains were greater for large frame cattle during both feeding phases and over the entire trial. There was however, no difference between daily empty body fat gain during the experimental trial.

Table 18 presents data summarizing the frame effect on empty tissue gains as percentages of the empty body weight gains. Cattle did not show a difference that was statistically significant for either empty body protein or empty body fat gains as percentages of empty body weight gains during the growing phase. There was a tendency for large framed cattle to have a higher percentage of empty

Table 17. Frame Size Effect on Daily Empty Body Gains (Trials 2 and 3)

	Treatments		
	Small	Large	SE
Item			
Empty Body Weight Gains, kg/day			
Growing	1.00[a]	1.29[b]	.060
Finishing	.78	.90	.043
Total	.87[a]	1.05[b]	.041
Empty Body Water Gains, g/day			
Growing	434.68[a]	626.09[b]	.039
Finishing	297.51[a]	435.53[b]	.026
Total	353.90[c]	503.52[d]	.023
Empty Body Protein Gains, g/day			
Growing	131.14[a]	188.89[b]	.012
Finishing	89.76[a]	131.40[b]	.007
Total	106.77[c]	151.91[d]	.007
Empty Body Fat Gains, g/day			
Growing	404.42	429.15	.050
Finishing	368.62	301.85	.035
Total	390.96	362.26	.031

[ab]Means within rows with different superscripts differ ($P < .05$).

[cd]Means within rows with different superscripts differ ($P < .01$).

Table 18. Frame Size Effect on Empty Body Tissue Gains As Percentages of Empty Body Weight Gains (Trials 2 and 3)

	Treatments		
	Small	Large	SE
Item			
Empty Body Protein Gains as % of Empty Body Weight Gains			
Growing	13.18	14.82	.738
Finishing	11.32[a]	14.92[b]	.787
Total	12.12[a]	14.87[b]	.540
Empty Body Fat Gains as % as Empty Body Weight Gains			
Growing	40.34	32.69	3.374
Finishing	48.55[a]	32.20[b]	3.575
Total	45.06[a]	32.59[b]	2.463

[ab]Means within rows with different superscripts differ ($P < .05$).

body weight gain as protein gain and lower fat gain. During the finishing phase and over the total trial, large frame cattle had a greater portion of the empty body gain in the form of protein gain and a lower percentage as fat gain.

Total dry matter consumed per kilogram of empty body weight was slightly higher in large type cattle. However, this slight increase in efficiency (Ferrell et al., 1978) by small cattle must be balanced against an 18% decrease in average daily gains observed in small frame cattle. Because large frame cattle were gaining empty body protein relative to empty body fat at a faster rate than small frame cattle, the energy requirements to maximize growth (protein deposition) in large frame cattle would be expected to be greater than for small frame cattle. Rattray et al. (1974) reported that protein synthesis requires 45.6 kcal of ME per gram of protein synthesized while fat synthesis requires only 10.2 kcal of ME per gram of fat synthesis which agrees with conclusions by Jesse et al. (1976b) that the fattening process is more efficient calorically than that of the growth process or protein deposition. A point still to remember is that fat has very little water associated with it, whereas muscle is approximately 80% water. This would mean that the

efficiency of muscle deposition, on a wet basis, would be more efficient that fat as a wet tissue. Hence we can therefore interject that large frame cattle at similar empty body weight have more efficient tissue deposition compared to the small frame cattle. Bergen (1974) reached the same conclusion in his work.

A possible explanation of the difference in responses of empty body fattening to the high plane of nutrition by the two types of cattle would be that they were in different stages of physiological maturity or development. Assuming relative chemical composition is a valid estimate of physiological maturity, the large frame cattle had not reached the same degree of physiological maturity as the small frame cattle. However, if degree of marbling or quality grades are used an in indicator of physiological maturity, then the two types were more similar.

3-Methylhistidine as an Index for Muscle Protein Breakdown in Cattle

The use of 3-methylhistidine as an index of muscle protein breakdown has been used in rats (Haverburg et al., 1975; Nishizawa et al., 1977; Ornstedt et al., 1978; Nishizawa et al., 1978; Ogata et al., 1978; Ward and Buttery, 1979; and Dunn et al., 1980) and in humans (Haverberg, 1975T; Bilmazes et al., 1978; Ballard et al., 1979; and Ward and Cooksley, 1979) for several years. This method has much potential as a means of quantitating protein breakdown in both man and animals. Limited work has been done to determine if this method can be used to quantitate muscle protein breakdown in farm animals. In view of the value of a non-destructive measure of muscle protein degradation in cattle, several studies were conducted to attempt validation of the method for use in cattle.

Reutilization of 3-Methylhistidine

The average recoveries of injected ^{14}C-radioactivity in urine from two yearling heifers are shown in Table 19. Urine was collected for 120 hours after injection to determine the percentage of the radioactive dose being

Table 19. Urinary Excretion of Radioactivity in Beef Cattle Following Intravenous Administration of ^{14}C-3-Methylhistidine[a]

Time After Injection (Hours)	CPM	Percent of Dose Excreted
4	6,612,365	30.2
8	2,667,103	12.2
12	1,296,233	5.9
16	768,285	3.5
22	849,413	3.9
28	1,118,693	5.1
34	840,198	3.8
40	523,445	2.4
52	1,100,978	5.0
64	1,104,375	5.0
80	1,206,000	5.5
96	816,400	3.7
108	496,125	2.3
120	261,300	1.2
		89.7

[a]Two crossbred Charolais heifers were used for recovery experiment.

excreted in that time period. Figure 27 graphically illustrates the clearance of ^{14}C-radioactivity by the heifers. A very high rate of ^{14}C elimination (1.65×10^6 CPM/hour) occurs within the first 4 hours after injection. The rate rapidly declines to a point of less than 0.22 CPM/hour at 120 hours after injection. Figure 28 depicts the increase in the percentage recovered of the original dose over time, the most rapid rate of excretion occurs within the first 4 hours. It was not surprising that a major portion of the total dose (30.2%) was excreted during that same 4 hour time period. At 8 hours after injection nearly half (42.4%) of the total dose had been recovered in the urine. The rate of elimination dropped steadily following the 8 hour collection time until the final collection at 120 hours, where 89.7% of the original dose had been recovered.

The total amount of ^{14}C-radioactivity recovered is very similar to the 80-90% recovery figures reported by Harris and Milne (1981) in cattle. The rapid recoveries of radioactivity in cattle urine suggest that the urinary excretion of 3-methylhistidine is quantitative and thus satisfactory for the use as an index of muscle protein breakdown in cattle. These values compare with recoveries of 100% of the dose in 3 days from rats (Young et al., 1972), 95% in 2 days from adult humans (Long et al., 1975),

Figure 27. Elimination of ^{14}C 3-Methylhistidine.

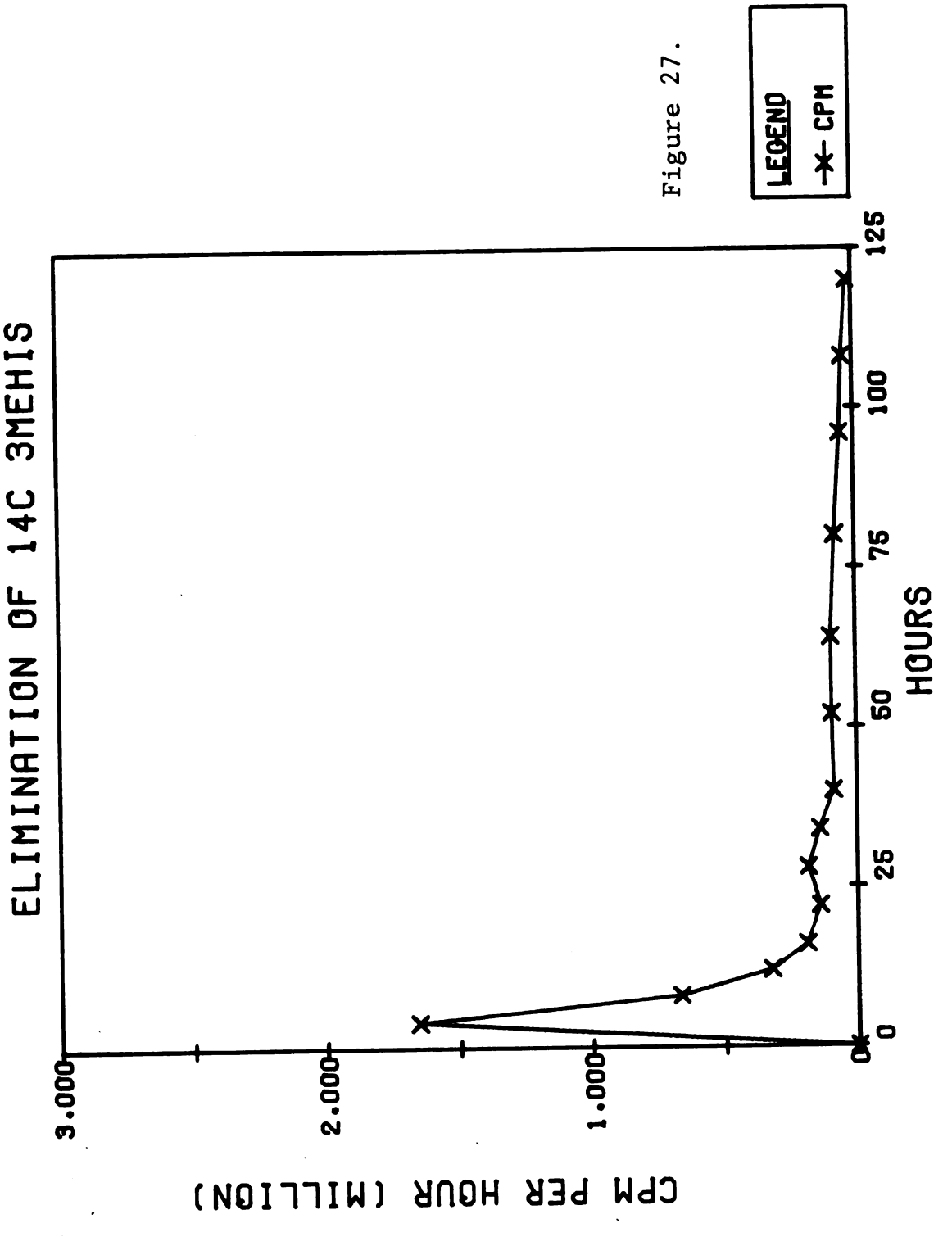
ELIMINATION OF 14C 3MEHIS
CPM PER HOUR (MILLION)
3.000
2.000
1.000
0.000
0
25
50
75
100
125
HOURS
LEGEND
CPM

Figure 27.

Figure 27. Elimination of ^{14}C 3-Methylhistidine.

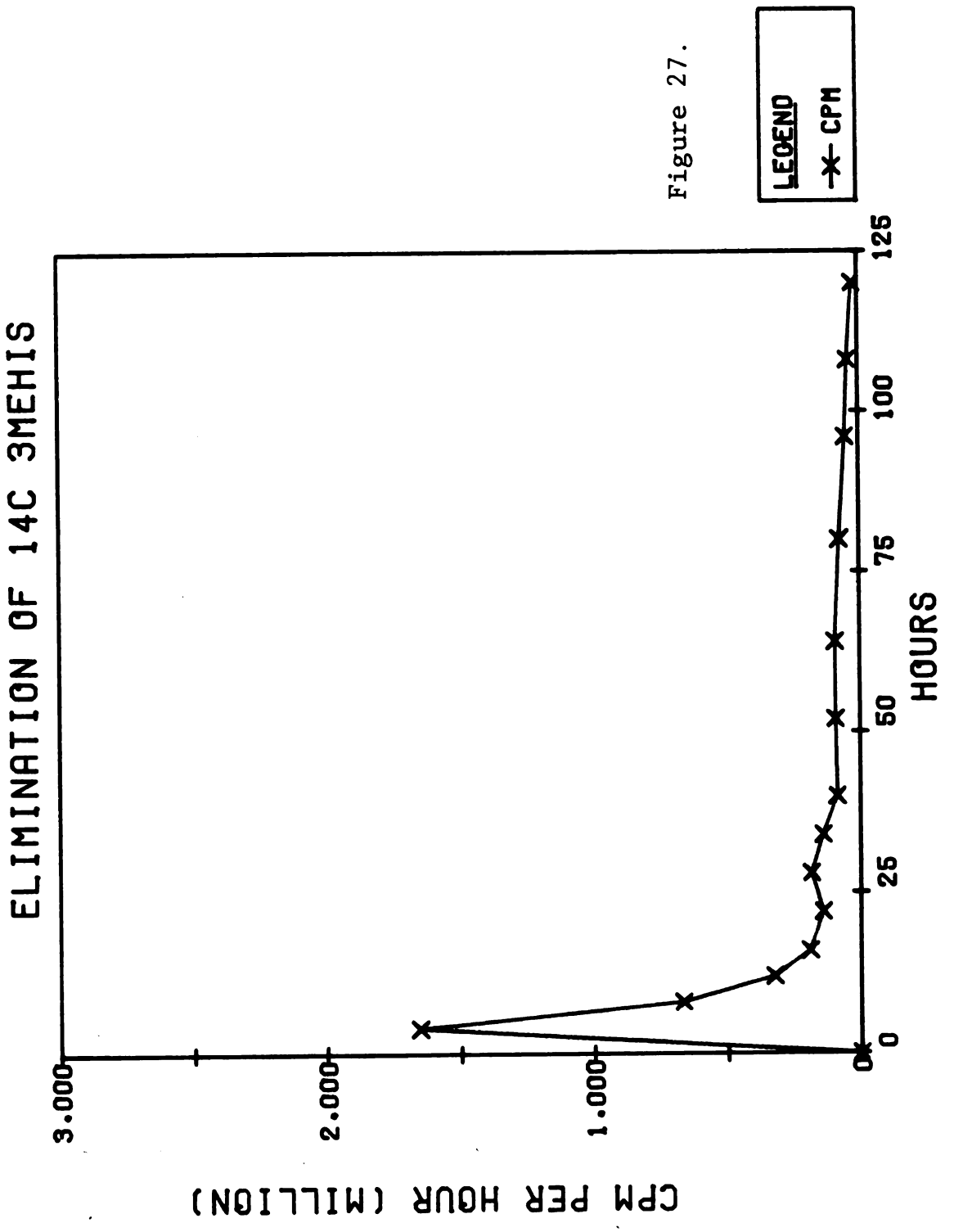
ELIMINATION OF 14C 3MEHIS
CPM PER HOUR (MILLION)
3.000
2.000
1.000
0
0
25
50
75
100
125
HOURS
LEGEND
CPM

Figure 27.

Figure 28. Recovery of ^{14}C 3-Methylhistidine.

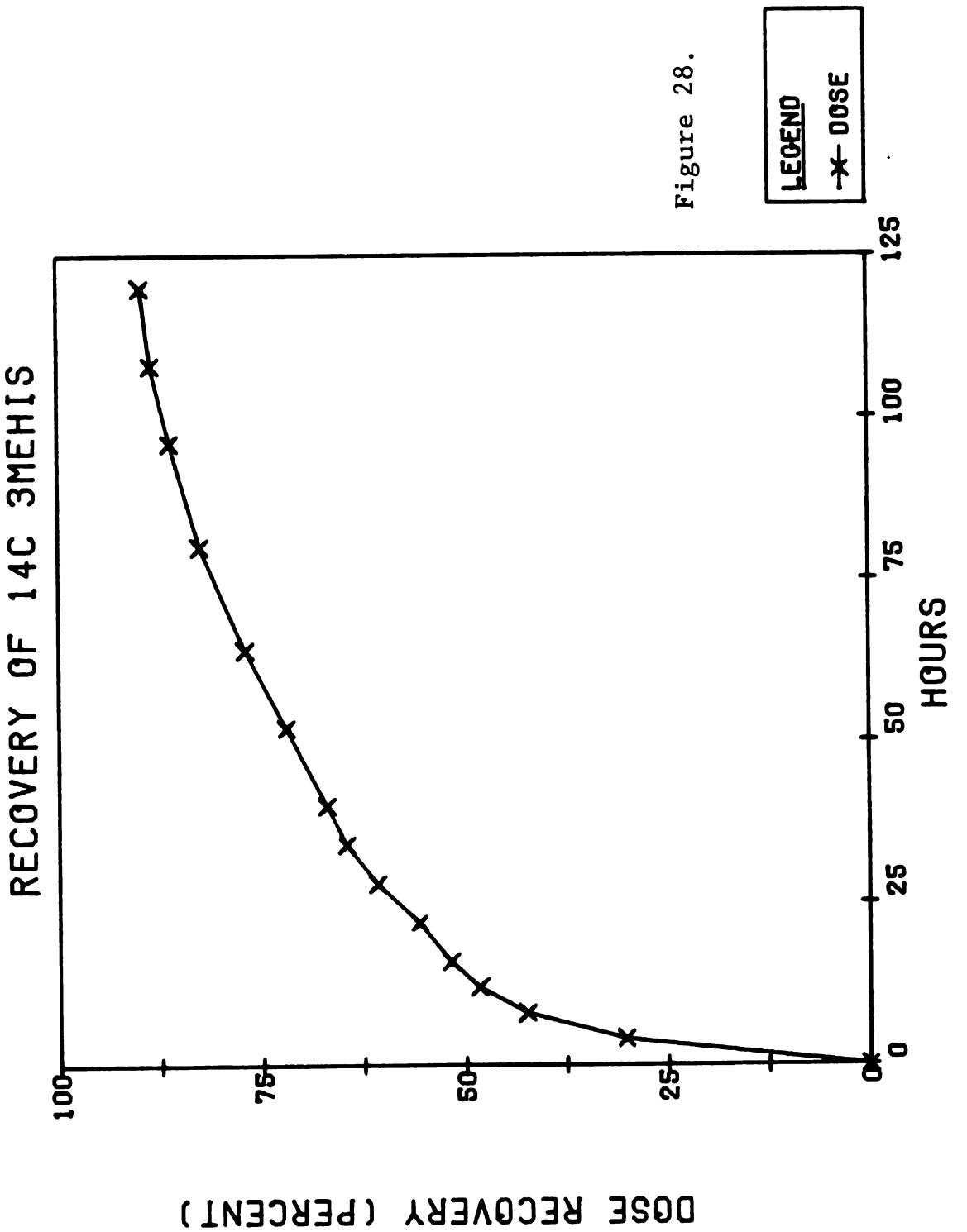
RECOVERY OF 14C 3MEHIS
LEGEND
DOSE
DOSE RECOVERY (PERCENT)
HOURS
0
25
50
75
100
125

Figure 28.

and 90-97% in 7 days from adult rabbits (Harris et al., 1977). In contrast, sheep (Harris and Milne, 1980a) and pigs (Harris and Milne, 1981) only slowly excreted labelled 3-methylhistidine.

Excretion of 3-Methylhistidine in Cattle-Frame Size Effects

A study was conducted with young cattle of two different frame sizes in order to explore the effect of frame size on 3-methylhistidine excretion. The study was also intended to determine the degradation rate and the synthesis rate of the two different frame size cattle.

Table 20 summarizes the effect of frame size on daily excretion of 3-methylhistidine, creatinine and total nitrogen. During periods 2, 3 and 4, large frame cattle excreted more 3-methylhistidine per day than small frame cattle. This would indicate that large frame cattle are breaking down more protein per day than small frame cattle. Total daily creatinine excretion followed the same pattern as the excretion of 3-methylhistidine. Large frame cattle excreted more creatinine than small frame cattle during periods 2, 3, and 4, corresponding with increased muscle mass for large frame cattle.

Table 20. Urinary Excretion of 3-Methylhistidine, Creatinine and Total Nitrogen in Cattle Over Time

	Treatments		
	Small	Large	SE
Item			
No. of Animals	4	4	
3-MeHis Excretion, mmoles/day			
Period 1	1.69	2.43	.386
Period 2	1.80[a]	3.07[b]	.228
Period 3	2.34[a]	2.91[b]	.110
Period 4	1.95[c]	2.78[d]	.109
Creatinine Excretion mg/day			
Period 1	6351.72	6732.51	1004.049
Period 2	7016.77[a]	10512.59[b]	563.442
Period 3	7999.24[a]	10928.02[b]	525.748
Period 4	8705.33[a]	11982.11[b]	576.069
Total Nitrogen Excretion, g/day			
Period 1	53.81	61.10	7.745
Period 2	52.83	69.17	7.646
Period 3	90.12	87.70	5.943
Period 4	76.79	80.91	6.271

[ab]Means within rows with different superscripts differ ($P < .05$).

[cd]Means within rows with different superscripts differ ($P < .01$).

Total nitrogen excretion was not different between the two frame sizes of cattle. This could be interpreted that large frame cattle were retaining more nitrogen, since their intake was greater than for small frame cattle. However, Harpster (1978) reported no difference in nitrogen retention between large and small frame cattle.

Figure 29 graphically shows the pattern of change in excretion exhibited by the two frame size groups. Urinary 3-methylhistidine excretion remained relatively steady for both treatments with the large frame cattle excreting in the range of 2.5 to 3.0 mmoles per day while the small frame cattle excreted between 1.6 and 2.4 mmoles per day, consistent with figures reported by Nishizawa et al., (1979) and Harris and Milne (1981). Thus, from these results it can be concluded that the excretion of 3-methylhistidine did not differ with time but did differ between cattle of different frame size.

Urinary creatinine excretion was measured throughout the study and the patterns of excretion are shown in Figure 30. There was a steady increase in creatinine excretion over time with both large and small frame cattle following a similar pattern. The differences between the two frame sizes in creatinine excretion were statistically significant, ranging from 6.732 to 11.982 mg/day for large

Figure 29. Daily 3-Methylhistidine Excretion.

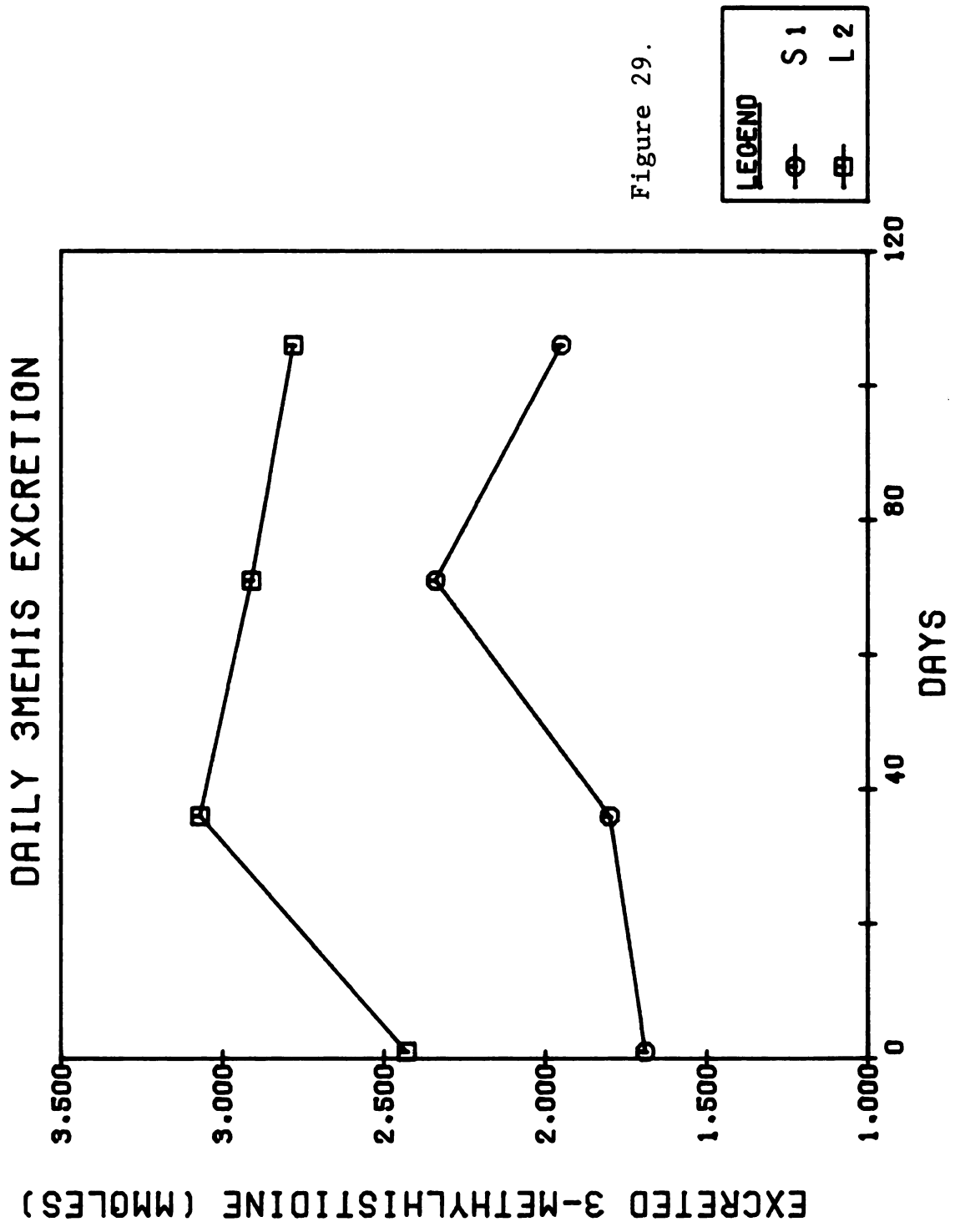
DAILY 3MEHIS EXCRETION
EXCRETED 3-METHYLHISTIDINE (MMOLES)
3.500
3.000
2.500
2.000
1.500
1.000
0
40
80
120
DAYS
LEGEND
S 1
L 2

Figure 29.

Figure 30. Daily Creatinine Excretion.

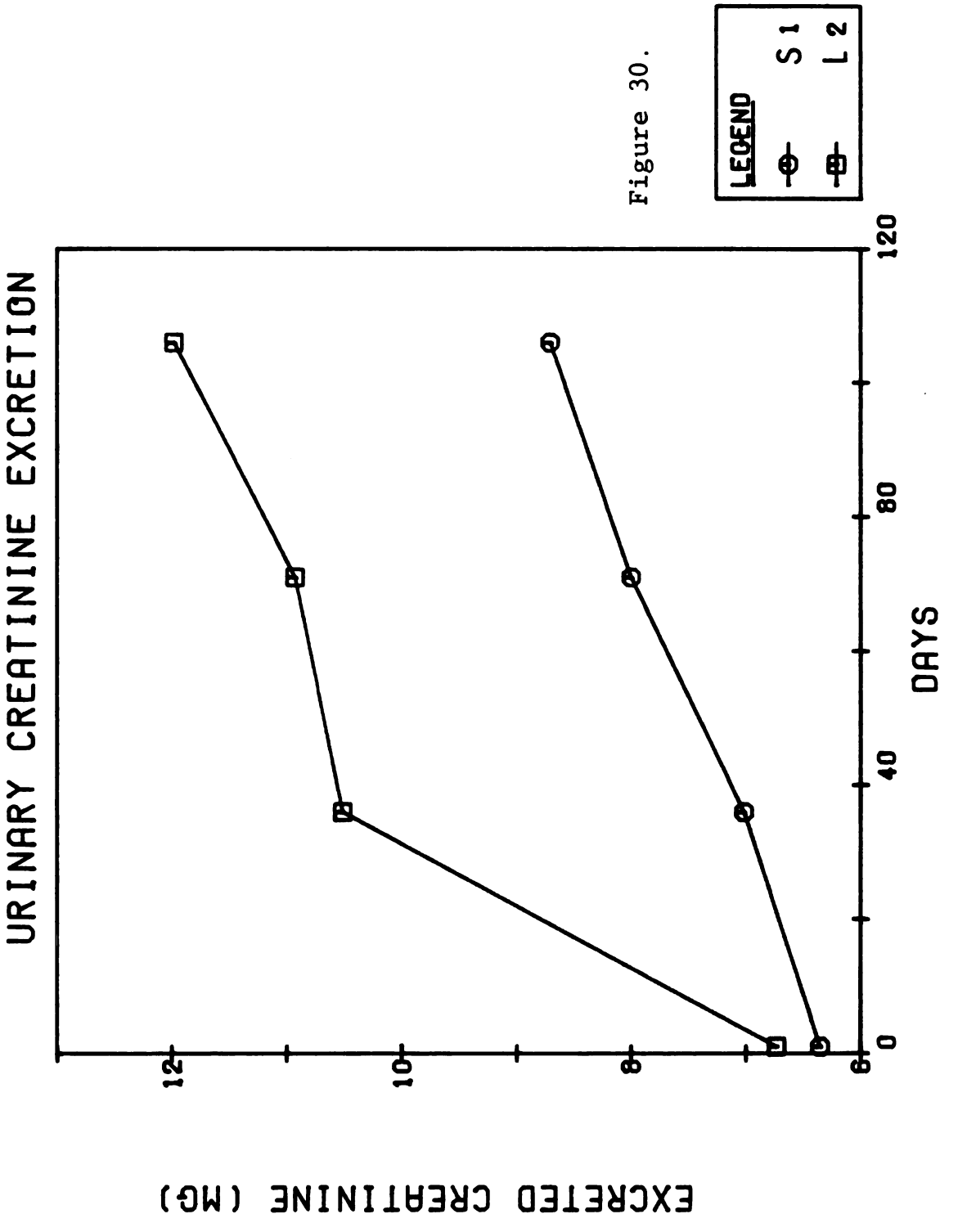
URINARY CREATININE EXCRETION
EXCRETED CREATININE (MG)
DAYS
LEGEND
S 1
L 2
0
40
80
120
6
8
10
12

Figure 30.

frame cattle and 6.351 and 8.705 mg/day for small frame cattle. The increase in creatinine excretion was expected since the muscle mass in the growing beef animal was increasing.

Table 21 shows the urinary outputs, body weights and the daily 3-methylhistidine output per unit of body weight for the large and small frame cattle during the study. Urinary output for both frame size cattle were not different, generally, reflecting water consumption during collection periods. Body weights were different between frame treatments for all periods except period 4 which shows near significant differences.

Daily output of 3-methylhistidine per unit of body was not different between treatments and did show similar patterns of change. Figure 31 diagrams the changes showing decline in 3-methylhistidine per unit of body weight. This could indicate that the muscle being broken down represented as a percent of the total empty body mass was declining with time.

Table 22 and Figure 32 show the change in urinary creatinine excretion per unit of body weight. There was a statistical difference due to frame size during period 2, but the differences during periods 3 and 4 were not significant although creatinine excretion remained somewhat higher than in small frame cattle. This would

Table 21. Relationship Between 3-Methylhistidine Excretion and Body Weight in Growing Cattle

	Treatments		
	Small	Large	SE
Item			
No. of Animals	4	4	
Average Daily Urine Output, liters			
Period 1	4.488	8.232	.868
Period 2	5.231	6.000	.346
Period 3	5.608	6.331	.442
Period 4	5.089	4.981	.519
Average Body Weight, kg			
Period 1	216.75[a]	298.50[b]	15.010
Period 2	254.25[a]	338.50[b]	15.819
Period 3	292.75[a]	377.00[b]	16.830
Period 4	332.00	416.00	17.951
Daily 3-MeHis Output/ Body Weight, mole/kg			
Period 1	7.238	8.125	1.208
Period 2	7.245	9.063	.704
Period 3	8.068	7.720	.158
Period 4	5.935	6.695	.227

[ab]Means within rows with different superscripts differ ($P < .05$).

Figure 31. 3-Methylhistidine Excretion Per Unit of Body Weight.

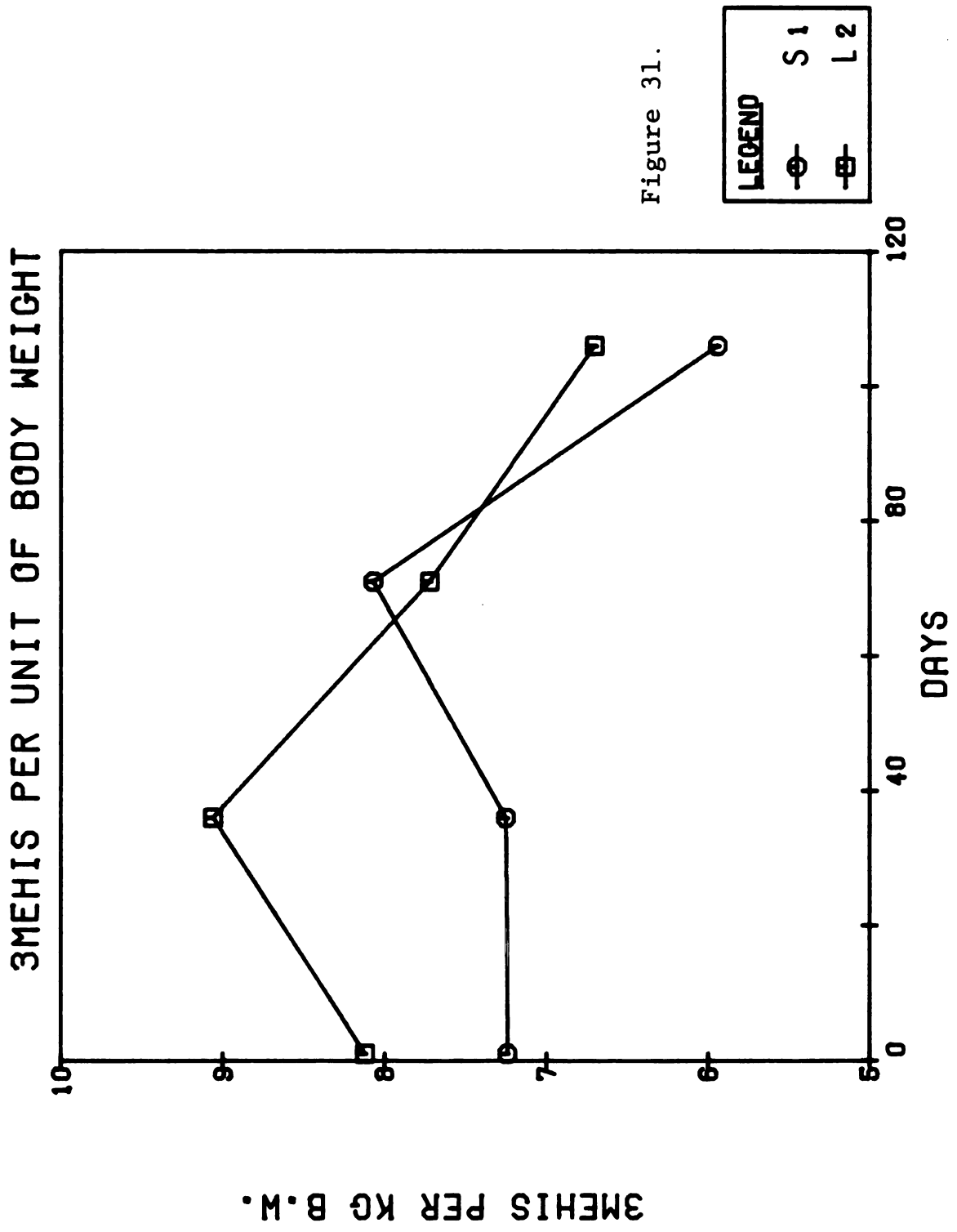
3MEHIS PER UNIT OF BODY WEIGHT
10
9
8
7
6
5
0
40
80
120
DAYS
LEGEND
S 1
L 2
3MEHIS PER KG B.W.

Figure 31.

Table 22. Urinary Creatinine Excretion Per Unit of Body Weight in Cattle

	Treatments		
	Small	Large	SE
Item			
No. of Animals	4	4	
Daily Creatinine Excretion Per Unit of Body Weight, mg/kg			
Period 1	19.55	17.08	3.422
Period 2	27.23[a]	31.05[b]	.697
Period 3	27.39	29.40	.721
Period 4	25.31	29.04	1.023

[ab]Means within rows with different superscripts differ ($P < .05$).

Figure 32. Creatinine Excretion Per Unit of Body Weight.

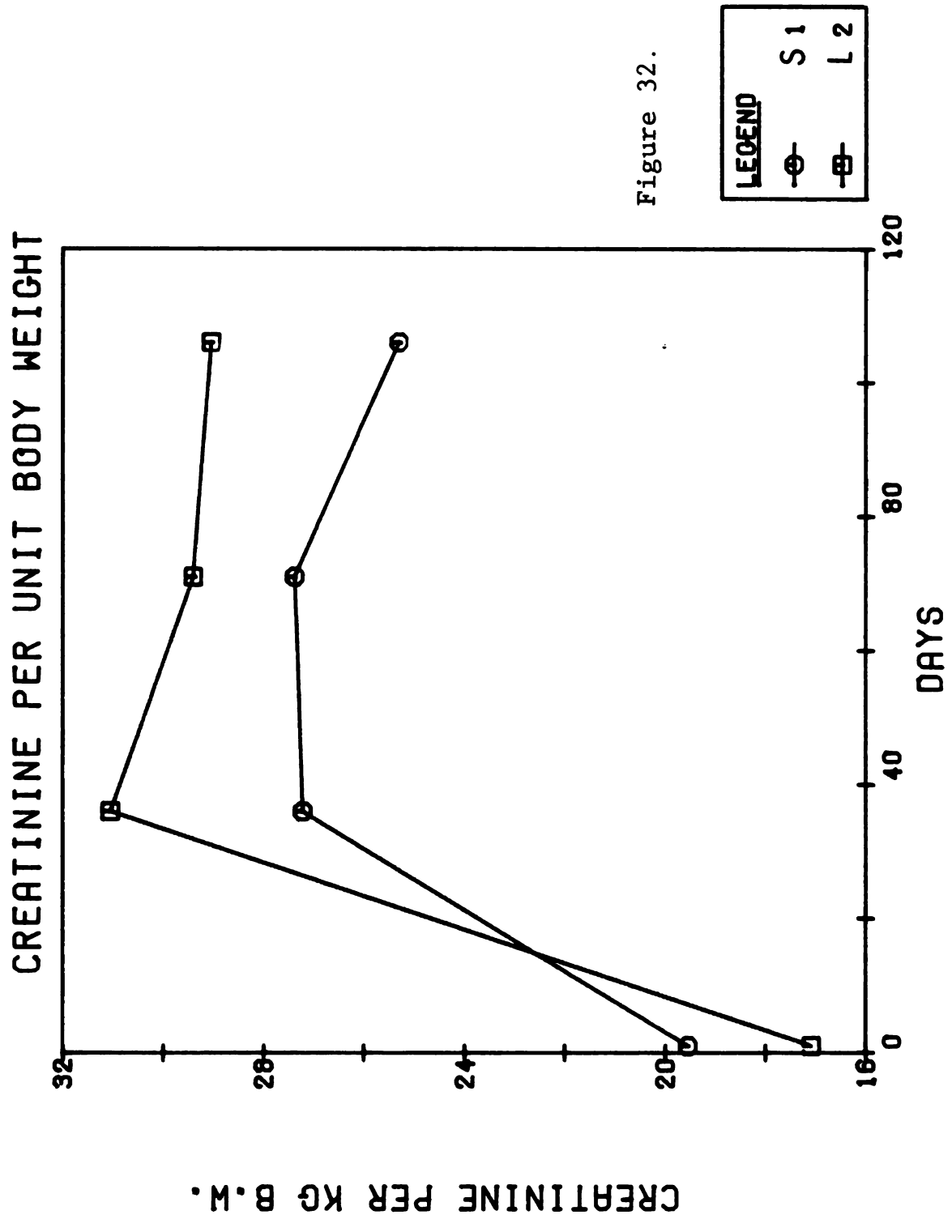
CREATININE PER UNIT BODY WEIGHT
CREATININE PER KG B.W.
DAYS
LEGEND
S 1
L 2
32
28
24
20
16
0
40
80
120

Figure 32.

coincide with the difference in body composition between the large frame and the small frame cattle, indicating more lean mass per unit of body weight. The levels of creatinine excreted per unit of body weight may decline slightly after period 1 coinciding with changes in the composition of the body and decline in the muscle mass relative to the empty body weight. Of course, once the animal has matured and body composition changes less, the creatinine excretion will remain rather constant.

The ratio of 3-methylhistidine to creatinine excretion is an indication of the rate of muscle degradation relative to muscle mass. Table 23 and Figure 33 show the changing of the ratios in growing cattle. There were no differences between treatment groups in excretion of 3-methylhistidine per unit of creatinine. There was however, a trend shown to decrease the excretion of3-methylhistidine per unit of of creatinine as the animals age increased. We can conclude from these ratios that degradation of muscle protein per unit of muscle mass for large frame cattle and small frame cattle does not differ.

An estimate of myofibrillar protein degradation rates can be made from the output of 3-methylhistidine in cattle. An example calculation is shown in Table 24 using a steer weighing 300 kg with 50 kg of empty body protein. Berg and Butterfield (1976) report that approximately 55% of the empty body protein is associated with skeletal muscle.

Table 23. Urinary 3-Methylhistidine to Creatinine ratios in Growing Cattle

	Treatments		
	Small	Large	SE
Item			
No. of Animals	4	4	
3-MeHis/Creatinine Ratio			
Period 1	.305	.290	.191
Period 2	.268	.289	.022
Period 3	.297	.267	.011
Period 4	.227	.235	.011

Figure 33. 3-MeHis to Creatinine Ratio.

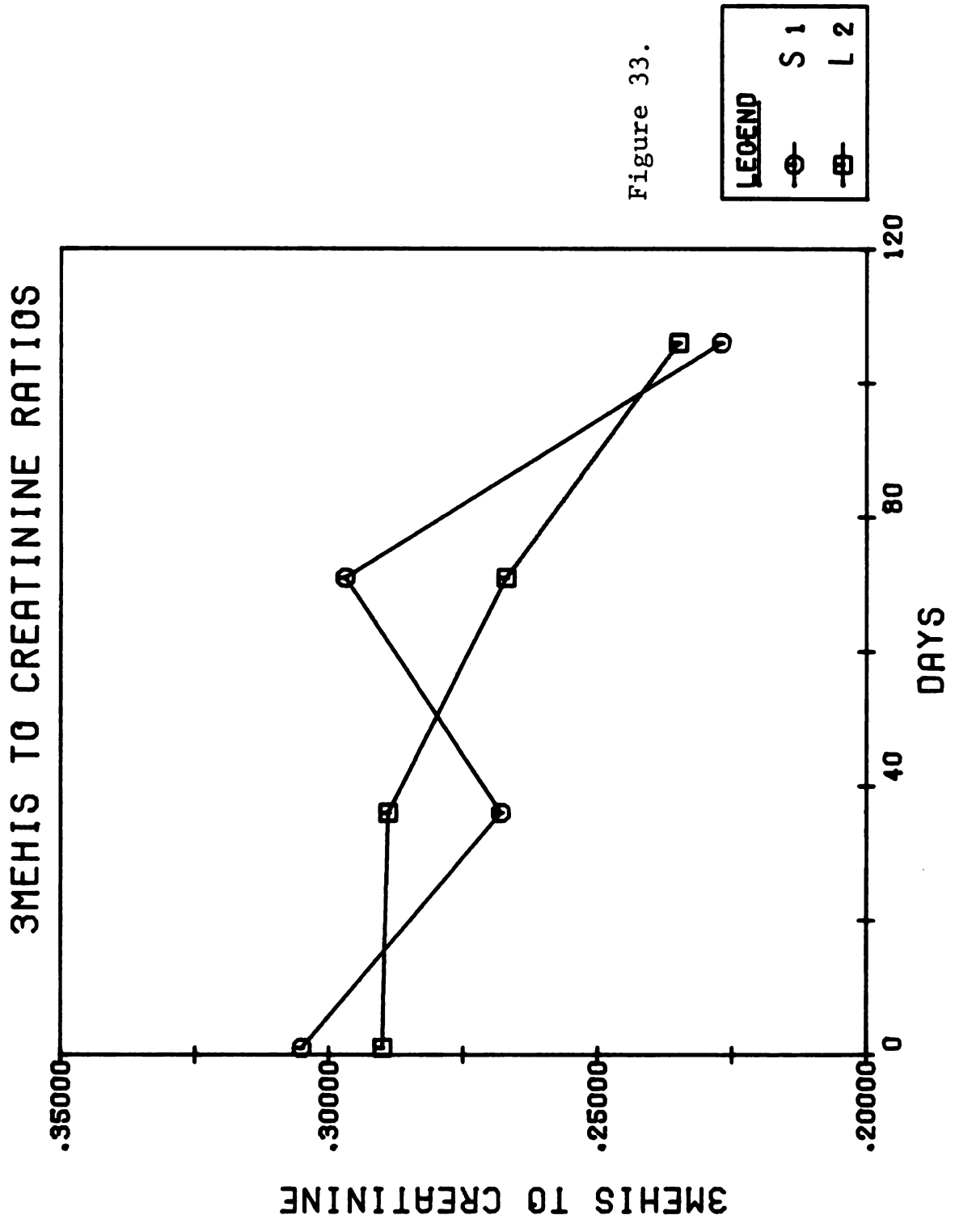
3MEHIS TO CREATININE RATIOS
3MEHIS TO CREATININE
DAYS
.35000
.30000
.25000
.20000
0
40
80
120
LEGEND
S 1
L 2

Figure 33.

Table 24. Example Calculation of Muscle Protein Turnover From Urinary 3-Methylhistidine Excretion in Cattle

Average Body Weight =	300 kg
Empty Body Protein =	50 kg
Percent of Empty Body Protein Associated with Skeletal Muscle[a] =	55 %
Amount of Empty Body Protein Associated with Skeletal Muscle =	27.5 kg
Quantity of 3-MeHis in Mixed Skeletal Muscle Proteins[b] =	590 mg/kg
Total 3-MeHis in Mixed Skeletal Muscle Proteins =	16.23 g .096 moles
Average Daily 3-MeHis Excretion =	340.0 mg 2.01 m moles
Fraction of Total 3-MeHis Pool Excreted Per Day (Daily Excr./Total Pool) =	.021
Half-life of Muscle Protein = $\frac{0.693}{0.021}$ =	$t_{\frac{1}{2}}$=33 days

[a]Berg and Butterfield, 1976.

[b]Nishizawa et al., 1979.

Nishizawa et al. (1979), determined the concentration of 3-methylhistidine in mixed skeletal muscle protein to be 590 mg/kg of protein. The total pool of bound 3-methylhistidine in skeletal muscle protein was then calculated. Knowing the daily 3-methylhistidine excreted, one can calculate the fraction of total pool excreted per day or the fractional breakdown rate. The half-life of the protein can then be calculated to help define the turnover rate of the protein.

Using the method just explained to calculate muscle protein turnover, comparisons between cattle of different frame size are shown in Table 25. The values showed near significance in favor of large frame cattle over small frame cattle when comparing amount of empty body protein and skeletal muscle protein along with skeletal muscle 3-methylhistidine pool. These values all showed gains in amount from period 1 through period 4. The fraction of the total 3-methylhistidine skeletal muscle pool excreted daily was not different between treatments or periods and when the half-lives of the proteins were calculated no differences between treatments were observed. The length of time that the cattle were on feed and the age of the cattle could be a partial reason as to why we did not see large change in the half-lives of the muscle protein. The muscle did appear to begin to decrease in its rate of

Table 25. Muscle Protein Degradation in Cattle Using 3-Methylhistidine as an Index

	Treatments		
	Small	Large	SE
Item			
No. of Animals	4	4	
Empty Body Protein, kg			
Period 1	29.37	40.80	2.605
Period 2	33.96	46.80	2.794
Period 3	38.43	52.64	3.016
Period 4	42.90	58.48	3.266
Skeletal Muscle Protein, kg			
Period 1	16.15	22.44	1.433
Period 2	18.68	25.74	1.536
Period 3	21.14	28.95	1.658
Period 4	23.59	32.16	1.796
3-MeHis Pool in Skeletal Muscle, moles			
Period 1	.0563	.0782	.005
Period 2	.0653	.0870	.005
Period 3	.0750	.0976	.006
Period 4	.0826	.1122	.006
Fraction of Total 3-MeHis Pool Excreted Daily, day^{-1}			
Period 1	.0271	.0310	.004
Period 2	.0299	.0397	.004
Period 3	.0330	.0288	.000
Period 4	.0247	.0249	.000
Half-life of Muscle Protein, days			
Period 1	29.84	23.97	3.574
Period 2	24.68	21.64	2.132
Period 3	21.45	24.07	.894
Period 4	28.74	28.29	1.603

turnover toward the end of the study and perhaps the use of older cattle or a longer study would help to determine if or when the beef animal begins to decline in rate of muscle protein turnover.

Table 26 shows calculated accretion, degradation and synthesis rates of protein in cattle. The accretion rates were calculated by using the D_2O dilution method to determine body composition then converted from empty body protein gains to muscle gains using the conversion factor reported by Berg and Butterfield (1976). Degradation rates, in g/day, were calculated using the half-life values and the quantity of muscle protein in the animal. An assumption was made for this calculation, that the relationship between muscle half-life and muscle quantity is relatively constant. Protein synthesis rates were calculated by summing both accretion rates and degradation rates.

Figure 34 graphically shows the difference between rates of protein degradation of large frame and small frame cattle. There is a statistically significent difference in protein degradation during periods 2 through 4, with large frame cattle showing higher rates than small frame cattle. A trend did seem to develop toward the end of the study for degradation rates to decline indicating that the animal is beginning to decelerate in its growth curve.

Table 26. Calculation of Muscle Protein Synthesis by Difference Using Rates of Accretion and Degradation

	Treatments		
	Small	Large	SE
Item			
Rate of Max. Potential Muscle Protein Accretion, g/day			
Period 1	72.13	103.89	----
Period 2	72.17	94.36	5.525
Period 3	70.21	91.77	5.364
Period 4	69.66	91.73	5.369
Rate of Muscle Protein Degradation, g/day			
Period 1	348.61[a]	501.37[b]	79.968
Period 2	371.64[a]	634.59[b]	47.305
Period 3	483.17[a]	602.23[b]	22.863
Period 4	403.23[c]	575.42[b]	22.346
Rate of Muscle Protein Synthesis, g/day			
Period 1	420.74	605.26	----
Period 2	443.81[a]	728.95[b]	54.129
Period 3	553.38[a]	694.00[b]	30.898
Period 4	472.89[c]	667.15[d]	26.792

[ab]Means within rows with different superscripts differ ($P < .05$).

[cd]Means within rows with different superscripts differ ($P < .01$).

Figure 34. Muscle Protein Degradation.

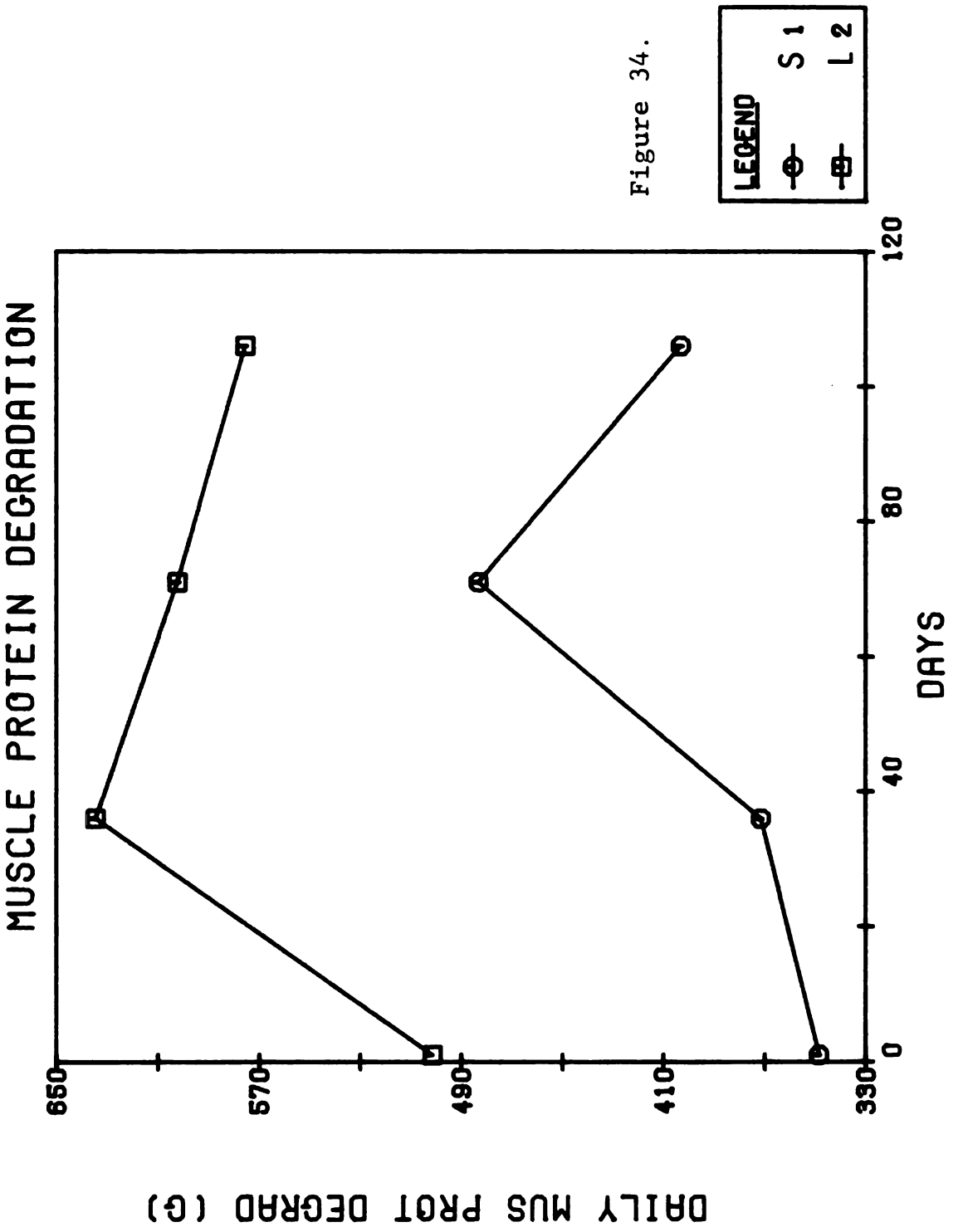
MUSCLE PROTEIN DEGRADATION
DAILY MUS PROT DEGRAD (G)
650
570
490
410
330
0
40
80
120
DAYS
LEGEND
S 1
L 2

Figure 34.

Figure 35 depicts the rates of synthesis of the large frame and small frame cattle. The large frame cattle did have greater rates of muscle protein synthesis than small frame cattle from period 2 on through the en of the study. A similar pattern to declining degradation rates are shown for synthesis rates also and may play a role in decreased rates of protein accretion with age.

Fractional degradation, synthesis and growth rates are shown in Table 27. The fractional rates between frame sizes did not differ significantly during the study. Calculation of the FBR was determined by dividing the daily amount of 3-methylhistidine excreted in the urine by the 3-methylhistidine pool in the muscle. The FSR of the muscle protein pool was determined according to formulas suggested by Funabiki et al. (1976). FSR equaled the synthesis rate (S) divided by the amount of 3-methylhistidine in the muscle pool. S was calculated from the following formula:

$$S = \frac{K_d(P - P_o e^{-K_d t})}{1 - e^{-K_d t}}$$

P and P_o are the sizes of the 3-methylhistidine pool in skeletal muscle at time t and t_o, respectively and K_d is the breakdown rate or FBR. Results show that FBR for both large and small frame cattle were approximately .03 day^{-1} which was higher than FBR reported by Harris and Milne (1981) of .015 day^{-1} in mature cattle and .012 day^{-1} shown

Figure 35. Muscle Protein Synthesis.

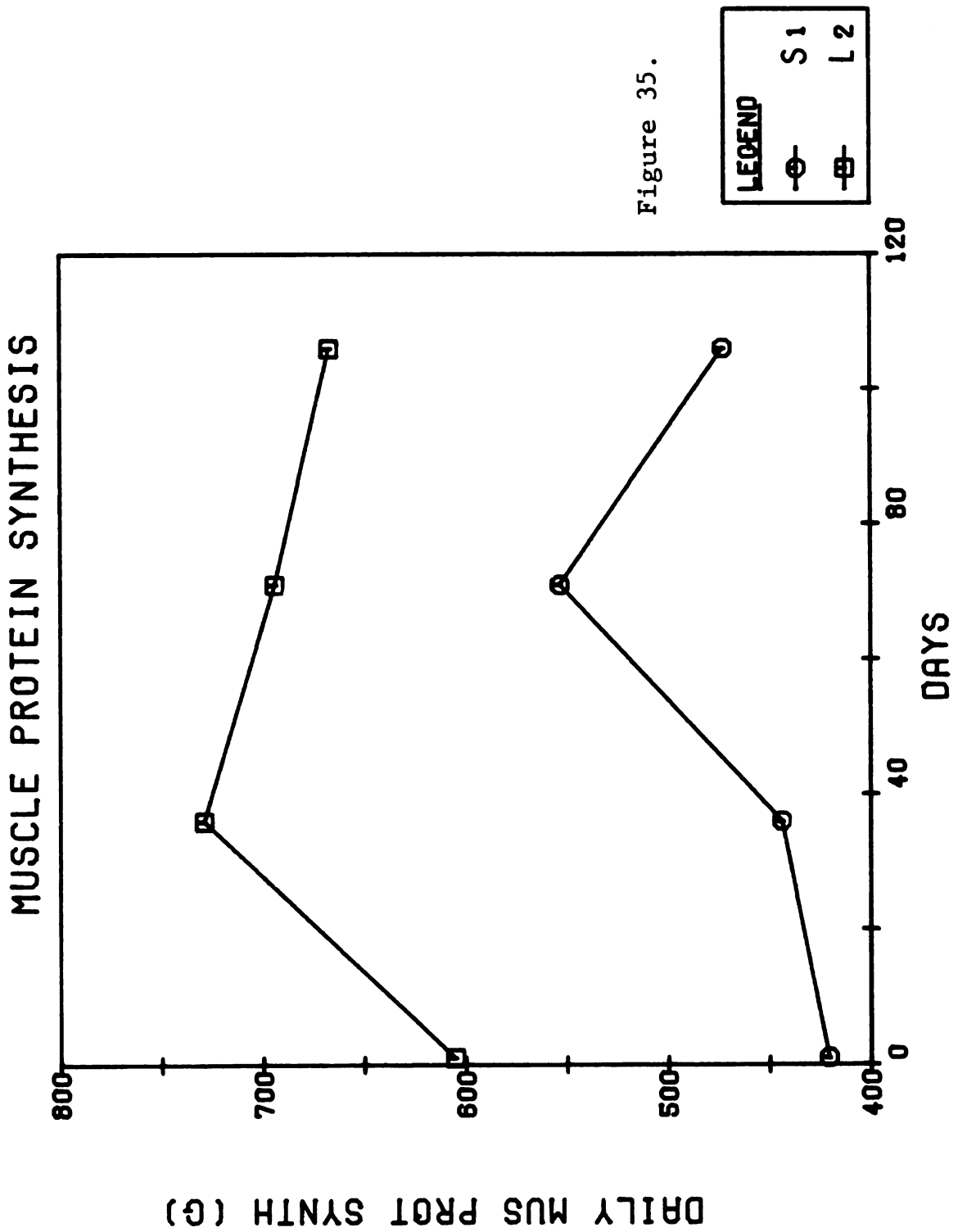
MUSCLE PROTEIN SYNTHESIS
DAILY MUS PROT SYNTH (G)
800
700
600
500
400
0
40
80
120
DAYS
LEGEND
S 1
L 2

Figure 35.

Table 27. Fractional Breakdown, Synthesis and Growth Rates for Muscle Protein

	Treatments	
	Small	Large
Item		
Fractional Breakdown Rate (FBR), day^{-1}		
Period 1	.0271	.0310
Period 2	.0299	.0397
Period 3	.0330	.0288
Period 4	.0247	.0249
Fractional Synthesis Rate (FSR), day^{-1}		
Period 2	.0321	.0410
Period 3	.0350	.0306
Period 4	.0264	.0272
Fractional Growth Rate (FGR), day^{-1}		
Period 2	.0022	.0013
Period 3	.0020	.0018
Period 4	.0017	.0023

by Nishizawa et al., (197) in growing cattle. The FSR was approximately .032 day^{-1}, similar to data reported by Lobley et al. (1980) where they showed FSR of around .02 day^{-1} in growing cattle using ^{3}H-tyrosine infusion.

When comparisons were made between accretion rates calculated from the fractional growth rates and measured accretion rates on the same animals, discrepancies occurred. The measured accretion rates were greater than the calculated rates. The differences in the two accretion rates are large enough to warrant some discussion.

Discrepancies between the two accretion rates may be due to the methods of calculation. The direct measurement may have some experimental errors however, the values are in line with data reported by Byers (1979) which would tend to validify those results. The calculated accretion rates using the FGR are based on measures of 3-methylhistidine excretion and the FBR. The calculation of the FBR was accomplished using several assumptions: (1) 55% of empty body protein is associated with skeletal muscle; (2) the concentration of 3-methylhistidine in skeletal muscle is 590 mg/kg protein; and (3) nearly 100% of the 3-methylhistidine excreted is contributed by the muscle 3-methylhistidine pool. If errors have occurred in these assumptions, the FBR would also be in error.

Because of the large quantity of muscle protein being dealt with in cattle only minute changes in the FGR would alter daily accretion dramatically. For example, if the calculated FGR, approximately .002 day^{-1}, were increased to .004 day^{-1}, the accretion rate would double from 55 g/day to 110 g/day. Any small error in the estimation of FGR is magnified when calculating accretion rates.

The refinement of the technique along with additional research in areas where facts rather than assumptions are needed, may produce a very useful research tool in the future to estimate protein turnover in cattle.

CONCLUSIONS

1. Steers on a high energy diet during the finishing phase had greater daily empty body weight gains over the entire trial.

2. Steers on a high energy diet during the finishing phase had greater daily empty body protein and fat gains over the entire trial.

3. Steers on a high energy diet throughout the trial had a greater amount of empty body fat upon termination, than steers receiving low energy during finishing phase.

4. A maximum rate of protein gain per day was not seen in these later maturing cattle, even at daily empty body weight gains of 1.2 kg/day.

5. Large frame cattle gained at a faster rate and consumed more dry matter per day, but showed no feed efficiency differences from small frame cattle.

6. Large frame cattle gained more protein per day than small frame cattle with no difference in daily fat gain.

7. At any given empty body weight during growth, large frame cattle contain more protein and less fat.

8. 3-Methylhistidine is quantitatively excreted in cattle.

9. Large frame cattle excreted a greater total amount of 3-methylhistidine and creatinine per day than small frame cattle.

10. When compared on a per unit of body weight basis, there was no difference in excretion of 3-methylhistidine or creatinine per day.

11. A decrease in the 3-MeHis-creatinine ratio was shown with increasing age, with no frame differences indicated.

12. FBR, FSR, AND FGR were not affected by frame size differences.

APPENDIX

Table A.1 RATION INGREDIENTS

Ingredient	International Reference No.
Alfalfa Hay	1-00-071
Corn cob pellets	---
Corn silage	3-08-153
Corn	4-02-931
Soybean meal	5-04-604
Ground limestone	6-02-632
Defluorinated phosphate	6-01-780
Sugarcane, molasses	4-04-696
Trace mineral salt	---
Vitamin A premix[a]	---
Vitamin D premix[b]	---

[a] 30,000 IU vitamin A per gram.
[b] 3,000 IU vitamin D per gram.

Table A.2 Individual Performance Data. (Trial 1)

Pen No.	Steer No.	Treat-ment	Treat-ment No.	Days on Feed	Initial Wt,kg	Final Wt,kg	Total ADG kg	Growing Pd.final wt,kg	Growing Pd. days	Growing Pd. ADG,kg/day	Finishing Pd. Days	Finishing Pd. ADG,kg/day
34	550	HH	1	230	226.5	480.3	1.10	307.8	70	1.16	160	1.08
34	577	HH	1	230	239.7	520.3	1.22	344.1	70	1.49	160	1.10
34	530	HH	1	230	281.4	581.1	1.30	383.2	70	1.45	160	1.24
34	569	HH	1	230	244.7	521.6	1.20	337.3	70	1.32	160	1.15
34	544	HH	1	230	233.8	497.1	1.14	319.6	70	1.22	160	1.11
34	508	HH	1	230	290.1	553.9	1.15	392.7	70	1.47	160	1.01
33	527	HL	2	281	278.8	544.3	0.94	351.9	70	1.04	211	0.91
33	551	HL	2	281	271.9	528.9	0.91	376.8	70	1.50	211	0.72
33	543	HL	2	281	255.1	515.7	0.93	326.9	70	1.03	211	0.99
33	575	HL	2	281	236.5	508.0	0.97	322.8	70	1.23	211	0.88
33	532	HL	2	281	262.9	507.6	0.87	338.2	70	1.08	211	0.80
33	518	HL	2	281	239.7	482.6	0.86	308.3	70	0.98	211	0.90
35	581	LL	3	300	241.1	520.3	0.93	326.4	102	0.79	198	0.98
35	507	LL	3	300	254.7	527.1	0.91	343.2	102	0.82	198	0.93
35	545	LL	3	300	263.3	546.2	0.94	374.6	102	1.09	198	0.87
35	580	LL	3	300	246.5	539.4	0.98	343.2	102	0.95	198	0.99
35	537	LL	3	300	282.8	574.8	0.97	390.9	102	1.06	198	0.93
35	590	LL	3	300	209.3	456.7	0.83	271.9	102	0.61	198	0.93
36	524	LH	4	242	264.7	499.4	0.97	342.8	103	0.76	139	1.13
36	573	LH	4	242	246.1	550.7	1.03	374.1	103	1.24	139	1.27
36	565	LH	4	242	210.2	443.1	0.96	294.6	103	0.82	139	1.07
36	554	LH	4	242	255.6	527.1	1.12	359.1	103	1.00	139	1.21
36	576	LH	4	242	240.6	524.3	1.17	347.3	103	1.03	139	1.27
36	523	LH	4	242	296.5	544.3	1.03	401.3	103	1.02	139	1.03

Table A.3 Individual Carcass Data (Trial 1).

Pen No.	Steer No.	Treatment No.	Hot carcass weight kg	Dressing %	Adj. Fat, in^2	Ribeye Area, in	KPH Fat %	Yield Grade	Marbling[a]	Quality[b] Grade
34	550	1	307.5	66.5	0.635	101.93	2.5	1.2	7	7
34	577	1	338.4	66.4	1.143	93.55	3.5	2.7	9	9
34	530	1	362.4	65.0	1.397	92.90	3.0	3.0	12	11
34	569	1	325.7	64.7	0.889	87.74	3.0	2.9	11	10
34	544	1	318.0	66.5	1.143	90.32	3.5	2.6	9	9
34	508	1	356.5	67.2	1.270	100.00	3.5	2.6	12	10
33	527	2	336.6	65.1	0.381	104.51	2.5	1.2	10	10
33	551	2	332.4	65.5	0.381	113.55	4.0	1.2	9	9
33	543	2	312.5	64.5	0.381	90.32	2.0	1.6	9	9
33	575	2	308.9	64.5	0.254	99.35	1.5	0.9	6	6
33	532	2	312.1	64.5	0.254	100.00	1.5	0.9	7	7
33	518	2	296.6	64.1	0.508	87.74	3.0	1.9	8	9
35	581	3	306.6	60.2	0.635	76.13	3.0	2.5	7	7
35	507	3	319.3	61.8	0.381	100.00	3.0	1.5	9	9
35	545	3	352.4	65.2	0.635	114.19	2.5	1.2	9	9
35	580	3	337.0	63.8	0.762	100.00	2.5	2.0	7	7
35	537	3	361.1	64.1	0.762	98.71	2.0	1.8	9	9
35	590	3	280.3	62.7	0.381	89.03	1.0	1.1	3	5
36	524	4	323.9	66.7	0.508	102.58	2.5	1.9	6	6
36	573	4	360.2	67.3	1.143	120.00	3.5	1.7	7	7
36	565	4	266.3	63.1	0.508	79.35	3.0	2.0	9	9
36	554	4	343.4	67.0	0.635	94.84	2.5	2.8	7	7
36	576	4	335.2	66.0	0.635	105.80	3.5	1.7	7	7
36	523	4	355.2	66.6	0.762	92.90	2.5	2.3	12	10

[a]Marbling score: Slight o = 8; Slight + = 9; Small - = 10.
[b]Quality score: Average good = 8; High good = 9; Low choice = 10.

Table A.4 Individual D_2O Empty Body Composition, Initial (Trial 1).

Pen No.	Steer No.	Treatment No.	EB Weight, kg	EB Water, kg	EB Protein, kg	EB Mineral, kg	EB Fat, kg
34	550	1	199.42	122.38	36.92	8.43	31.69
34	577	1	213.61	130.25	39.30	8.97	35.09
34	530	1	242.53	145.01	43.75	9.99	43.78
34	569	1	220.33	140.94	42.52	9.71	27.15
34	544	1	213.98	132.46	39.96	9.13	32.43
34	508	1	247.02	148.93	44.93	10.26	42.89
33	527	2	259.59	160.38	48.39	11.05	39.77
33	551	2	237.94	145.62	43.93	10.03	38.36
33	543	2	232.58	146.15	44.09	10.07	32.26
33	575	2	206.96	135.94	41.01	9.37	20.64
33	532	2	243.33	157.93	47.65	10.88	26.88
33	518	2	214.63	129.71	39.13	8.93	36.85
35	581	3	223.71	135.88	40.99	9.36	37.48
35	507	3	236.47	145.72	43.96	10.04	36.74
35	545	3	220.88	142.18	42.90	9.79	26.00
35	580	3	224.77	135.14	40.77	9.31	39.55
35	537	3	242.86	145.47	43.89	10.02	43.48
35	590	3	187.43	122.07	36.83	8.41	20.13
36	524	4	230.63	137.88	41.60	9.50	41.65
36	573	4	218.40	139.26	42.02	9.60	27.53
36	565	4	187.83	116.89	35.27	8.05	27.62
36	554	4	220.61	134.85	40.68	9.29	35.79
36	576	4	215.20	149.44	45.09	10.30	48.23
36	523	4	252.86	162.21	48.94	11.18	30.54

Table A.5 Individual D_2O Empty Body Composition, Period 1 (Trial 1).

Pen No.	Steer No.	Treatment No.	EB Weight, kg	EB Water, kg	EB Protein, kg	EB Mineral, kg	EB Fat, kg
34	550	1	243.62	149.69	45.16	10.31	38.45
34	577	1	259.39	154.75	46.69	10.66	47.28
34	530	1	269.93	120.53	36.36	8.30	104.74
34	569	1	279.12	165.63	49.97	11.41	52.11
34	544	1	260.45	152.55	46.02	10.51	51.37
34	508	1	308.42	181.09	54.63	12.48	60.22
33	527	2	291.05	179.44	54.14	12.36	45.11
33	551	2	292.24	172.89	52.16	11.91	55.29
33	543	2	258.37	158.85	47.92	10.95	40.65
33	575	2	252.52	159.36	48.08	10.98	34.11
33	532	2	269.51	162.23	48.94	11.18	47.16
33	518	2	256.05	151.51	45.71	10.44	48.39
35	581	3	240.30	144.71	43.66	9.97	41.96
35	507	3	260.84	150.79	45.49	10.39	54.17
35	545	3	275.23	163.38	49.29	11.26	51.30
35	580	3	249.07	148.49	44.80	10.23	45.56
35	537	3	292.99	176.52	53.26	12.16	51.05
35	590	3	219.50	139.23	42.01	9.59	28.67
36	524	4	249.61	144.75	43.67	9.97	51.22
36	573	4	265.33	161.77	48.81	11.15	43.61
36	565	4	229.95	142.15	42.89	9.79	35.12
36	554	4	265.18	159.07	47.99	10.96	47.15
36	576	4	259.58	155.98	47.06	10.75	45.79
36	523	4	304.81	173.30	52.28	11.94	67.29

Table A.6 Individual D_2O Empty Body Composition, Period 2 (Trial 1).

Pen No.	Steer No.	Treatment No.	EB Weight, kg	EB Water, kg	EB Protein, kg	EB Mineral, kg	EB Fat, kg
34	550	1	274.70	165.34	49.88	11.39	48.09
34	577	1	311.10	175.20	52.86	12.07	70.98
34	530	1	345.95	198.13	59.78	13.65	74.39
34	569	1	299.86	175.49	52.95	12.09	59.34
34	544	1	284.60	164.91	49.75	11.36	58.58
34	508	1	363.42	209.34	63.16	14.42	76.50
33	527	2	325.24	192.35	58.03	13.25	61.61
33	551	2	340.08	195.03	58.84	13.44	72.78
33	543	2	302.75	177.40	53.52	12.22	59.61
33	575	2	298.99	174.40	52.62	12.02	59.97
33	532	2	311.81	188.46	56.86	12.99	53.50
33	518	2	291.54	164.27	49.56	11.32	66.39
35	581	3	295.27	182.40	55.03	12.57	45.28
35	507	3	311.59	189.73	57.24	13.07	51.55
35	545	3	321.63	186.52	56.27	12.85	65.99
35	580	3	310.80	184.23	55.58	12.69	58.29
35	537	3	350.21	212.37	64.07	14.63	59.13
35	590	3	255.57	158.81	47.91	10.94	37.91
36	524	4	312.19	185.47	55.96	12.78	57.99
36	573	4	334.42	197.62	59.62	13.62	63.57
36	565	4	272.89	157.33	47.46	10.84	57.25
36	554	4	324.75	196.40	59.26	13.53	55.56
36	576	4	313.20	185.47	55.96	12.78	59.01
36	523	4	357.49	197.63	59.63	13.62	86.62

Table A.7 Individual D_2O Empty Body Composition, Period 3 (Trial 1).

Pen No.	Steer No.	Treatment No.	EB Weight, kg	EB Water, kg	EB Protein, kg	EB Mineral, kg	EB Fat, kg
34	550	1	347.82	213.59	64.44	14.72	55.08
34	577	1	400.54	211.07	63.68	14.54	111.25
34	530	1	412.71	202.39	61.06	13.94	135.31
34	569	1	381.19	215.87	65.13	14.87	85.32
34	544	1	349.85	184.05	55.53	12.68	97.59
34	508	1	439.39	243.78	75.55	16.80	105.27
33	527	2	391.76	226.39	68.30	15.60	81.47
33	551	2	401.51	221.71	66.89	15.28	97.64
33	543	2	363.83	203.11	61.28	13.99	85.44
33	575	2	360.62	227.34	68.59	15.66	49.03
33	532	2	369.36	219.16	66.12	15.10	68.98
33	518	2	329.81	174.78	52.73	12.04	90.25
35	581	3	339.20	204.50	61.70	14.09	58.91
35	507	3	365.55	199.52	60.20	13.75	92.09
35	545	3	383.47	207.49	62.60	14.30	99.08
35	580	3	380.67	206.28	62.23	14.21	97.95
35	537	3	410.19	221.68	66.88	15.27	106.35
35	590	3	309.11	189.58	57.20	13.06	49.27
36	524	4	355.20	210.45	63.49	14.50	66.75
36	573	4	403.43	247.16	74.57	17.03	64.67
36	565	4	303.44	179.02	54.01	12.33	58.08
36	554	4	389.35	243.13	73.35	16.75	56.12
36	576	4	355.20	210.45	63.49	14.50	66.75
36	523	4	414.04	220.44	66.51	15.19	111.91

Table A.8 Individual D_2O Empty Body Composition, Period 4 (Trial 1).

Pen No.	Steer No.	Treatment No.	EB Weight, kg	EB Water, kg	EB Protein, kg	EB Mineral, kg	EB Fat, kg
34	550	1	411.53	248.57	74.99	17.13	70.84
34	577	1	425.01	220.46	66.51	15.19	122.84
34	530	1	467.36	230.34	69.49	15.87	151.66
34	569	1	426.83	232.14	70.04	15.99	108.67
34	544	1	421.33	231.86	69.95	15.98	103.55
34	508	1	439.72	235.58	77.48	15.64	111.02
33	527	2	436.14	243.87	73.58	16.80	101.89
33	551	2	434.40	237.67	71.71	16.38	108.64
33	543	2	404.35	235.81	71.14	16.25	81.15
33	575	2	407.84	235.61	71.08	16.23	69.92
33	532	2	402.63	236.53	71.36	16.30	78.43
33	518	2	367.95	206.29	62.24	14.21	85.20
35	581	3	414.99	228.45	68.92	15.74	101.89
35	507	3	407.39	230.64	69.58	15.89	91.28
35	545	3	429.59	234.47	70.74	16.16	108.22
35	580	3	421.86	224.03	67.59	15.44	114.81
35	537	3	474.41	257.64	77.73	17.75	121.29
35	590	3	331.33	197.70	59.65	13.62	60.36
36	524	4	427.79	251.89	76.00	17.36	82.55
36	573	4	470.96	253.04	76.34	17.43	124.14
36	565	4	365.98	205.80	62.09	14.18	83.91
36	554	4	448.24	252.06	76.05	17.37	102.77
36	576	4	426.72	232.03	70.00	15.99	108.70
36	523	4	461.13	240.02	72.41	16.54	132.16

Table A.9 Individual D_2O Empty Body Composition, Period 5 (Trial 1).

Pen No.	Steer No.	Treatment No.	EB Weight, kg	EB Water, kg	EB Protein, kg	EB Mineral, kg	EB Fat, kg
34	550	1	428.91	235.07	70.92	16.20	106.72
34	577	1	489.63	250.31	75.52	17.25	146.56
34	530	1	492.56	260.84	78.70	17.97	135.05
34	569	1	471.33	251.01	75.73	17.30	127.29
34	544	1	455.55	236.38	71.32	16.29	131.56
34	508	1	521.30	275.72	83.18	19.00	143.40
33	527	2	488.63	259.19	78.20	17.86	133.38
33	551	2	478.01	257.86	77.80	17.77	124.59
33	543	2	470.12	256.83	77.49	17.70	118.11
33	575	2	445.90	263.98	79.64	18.19	84.10
33	532	2	433.28	262.29	79.13	18.07	83.78
33	518	2	423.30	222.05	66.99	15.30	118.96
35	581	3	466.38	237.96	71.79	16.40	140.24
35	507	3	480.92	261.50	78.89	18.02	122.51
35	545	3	503.57	288.15	86.93	19.85	108.64
35	580	3	438.20	233.31	70.39	16.08	118.43
35	537	3	535.26	298.08	89.93	20.54	126.72
35	590	3	419.02	260.11	78.48	17.92	62.51
36	524	4	457.74	259.79	78.38	17.90	101.67
36	573	4	520.52	275.90	83.24	19.01	142.38
36	565	4	418.77	255.55	68.05	15.54	109.63
36	554	4	487.79	286.89	86.55	19.77	94.58
36	576	4	489.35	266.93	80.53	18.39	123.50
36	523	4	487.07	246.18	74.27	16.96	149.66

Table A.10 Individual Performance Data (Trial 2 and 3).

Steer No.	Treat-ment No.	Year No.	Initial wt,kg	Final wt,kg	Period 1 Final wt,kg	Period 1 Days	Period 1 Average Daily Dry matter Intake, kg	Period 2 Days	Period 2 Average Daily Dry matter Intake, kg	Total Average Daily Dry matter Intake, kg
198	1	1	350.63	519.36	433.18	57	17.44	83	21.53	19.87
50	1	1	270.34	434.54	337.93	57	12.72	111	18.08	16.26
154	1	1	282.59	365.14	327.04	57	10.76	83	11.57	11.23
122	1	1	323.86	428.64	383.29	43	17.90	83	18.77	18.48
156	1	1	314.34	430.91	385.55	57	16.14	83	16.90	16.59
200	1	1	297.56	444.52	374.21	57	14.72	83	19.43	17.51
63	2	1	312.53	542.04	430.91	57	19.71	111	23.98	22.53
60	2	1	330.22	531.16	415.04	57	18.60	111	24.59	22.56
65	2	1	323.41	540.23	405.97	57	17.63	111	22.48	20.84
64	2	1	340.19	563.82	439.98	57	20.03	111	23.43	22.28
61	2	1	298.46	49.69	371.95	57	15.11	111	19.32	17.90
62	2	1	324.32	531.16	410.50	57	16.39	111	24.95	22.05
198	1	2	188.24	400.52	299.37	84	14.49	84	15.60	15.04
187	1	2	220.45	410.95	315.25	84	14.57	84	16.02	15.30
160	1	2	126.55	285.31	217.72	84	11.01	84	10.67	10.84
141	1	2	271.25	469.92	376.48	84	18.27	84	19.15	18.71
139	2	2	285.76	456.31	376.48	84	16.60	84	17.69	17.14
154	2	2	289.85	453.59	385.55	84	17.42	84	14.89	16.16
109	2	2	276.24	497.14	403.70	84	20.60	84	18.80	19.70
101	2	2	299.82	485.34	396.89	84	17.26	84	26.05	21.66

Table A.11 Individual Carcass Data (Trial 2 and 3).

Steer No.	Treatment No.	Year No.	Adj. Fat CM	Ribeye Area CM^2	Yield Grade	Quality Grade[a]
198	1	1	.65	10.5	3.6	9
50	1	1	.35	11.5	2.7	9
154	1	1	.50	9.8	2.8	7
122	1	1	.50	11.5	2.8	7
156	1	1	.70	10.0	3.8	9
200	1	1	.55	10.6	3.1	6
63	2	1	.50	12.1	3.1	10
60	2	1	.40	14.0	2.4	10
65	2	1	.20	11.4	2.3	7
64	2	1	.25	12.3	2.8	7
61	2	1	.25	13.8	2.0	7
62	2	1	.35	12.2	2.4	10
196	1	2	.55	10.3	3.1	10
187	1	2	.60	9.1	3.7	10
160	1	2	.50	6.9	3.3	11
141	1	2	.75	10.8	3.7	7
139	2	2	.20	9.7	2.8	9
154	2	2	.40	10.0	3.3	7
109	2	2	.50	12.4	2.8	10
101	2	2	.30	12.0	2.2	9

[a]Quality score: Average good = 8; High good = 9; Low choice = 10.

Table A.12 Individual Empty Body Composition, Initial (Trial 2 and 3).

Steer No.	Treatment No.	Year No.	EB Weight, kg	EB Water, kg	EB Protein, kg	EB Mineral, kg	EB Fat, kg
198	1	1	350.94	159.93	48.25	11.02	93.54
50	1	1	270.58	141.61	42.73	9.76	57.72
154	1	1	282.84	129.58	39.09	8.93	83.76
122	1	1	324.16	142.70	43.05	9.83	88.93
156	1	1	314.62	150.08	45.28	10.34	87.73
200	1	1	297.82	154.29	46.55	10.63	62.76
63	2	1	312.81	178.06	53.72	12.27	36.12
60	2	1	330.51	143.46	43.28	9.88	77.29
65	2	1	323.70	184.27	55.59	12.70	39.96
64	2	1	340.50	199.85	60.29	13.77	35.47
61	2	1	298.73	150.87	45.52	10.40	63.88
62	2	1	324.61	163.74	49.40	11.28	63.39
196	1	2	204.75	97.89	29.53	6.74	49.91
187	1	2	220.64	89.43	26.98	6.16	69.72
160	1	2	126.67	53.50	16.14	3.69	41.17
141	1	2	271.49	135.88	41.00	9.36	62.29
139	2	2	286.02	135.60	40.91	9.34	76.66
154	2	2	290.11	127.69	38.52	8.80	88.26
109	2	2	276.49	127.26	38.40	8.77	67.24
101	2	2	300.09	133.88	40.39	9.22	74.88

Table A.13 Individual Empty Body Composition, MIDTERM (Trial 2 and 3).

Steer No.	Treat-ment No.	Year No.	EB Weight, kg	EB Water, kg	EB Protein, kg	EB Mineral, kg	EB Fat, kg
198	1	1	390.78	195.71	59.04	13.48	122.55
50	1	1	309.13	168.65	50.88	11.62	77.98
154	1	1	301.53	150.23	45.32	10.35	95.63
122	1	1	320.17	163.22	49.24	11.25	96.46
156	1	1	343.63	167.13	50.42	11.52	114.57
200	1	1	334.72	176.62	53.29	12.17	92.64
63	2	1	387.83	229.51	69.24	15.81	73.26
60	2	1	359.64	193.68	58.13	13.28	95.55
65	2	1	358.60	212.60	64.14	14.65	67.21
64	2	1	394.44	216.55	65.33	14.92	97.63
61	2	1	334.95	180.63	54.50	12.45	87.38
62	2	1	370.37	216.74	65.39	14.93	73.31
196	1	2	281.10	139.41	42.06	9.61	90.03
187	1	2	286.23	139.50	42.09	9.61	95.03
160	1	2	180.66	74.52	22.48	5.13	78.53
141	1	2	350.55	171.15	51.64	11.79	115.97
139	2	2	327.81	171.24	51.66	11.80	93.10
154	2	2	348.31	173.12	52.23	11.93	111.03
109	2	2	361.61	184.15	55.56	12.69	109.21
101	2	2	355.70	188.99	57.02	13.02	96.67

Table A.14 Individual Empty Body Composition, FINAL (Trial 2 and 3).

Steer No.	Treatment No.	Year No.	EB Weight, kg	EB Water, kg	EB Protein, kg	EB Mineral, kg	EB Fat, kg
198	1	1	474.77	220.53	66.53	15.19	172.52
50	1	1	398.70	195.54	59.00	13.47	130.69
154	1	1	327.23	160.57	48.45	11.06	107.15
122	1	1	383.42	189.24	57.09	13.04	124.04
156	1	1	387.17	175.24	52.87	12.07	146.99
200	1	1	395.90	185.10	55.85	12.75	142.20
63	2	1	476.84	240.71	72.62	16.59	146.93
60	2	1	494.53	257.89	77.81	17.77	141.06
65	2	1	473.73	248.97	75.11	17.15	132.50
64	2	1	505.11	267.70	80.77	18.44	138.20
61	2	1	431.64	214.30	64.65	14.77	137.92
62	2	1	463.64	248.34	74.92	17.11	123.26
196	1	2	365.59	182.42	55.04	12.57	115.57
187	1	2	359.03	168.73	50.91	11.63	127.77
160	1	2	255.51	100.18	30.22	6.90	118.21
141	1	2	419.90	207.94	62.74	14.33	134.89
139	2	2	394.66	206.27	62.23	14.21	111.95
154	2	2	407.20	215.04	64.88	14.82	112.46
109	2	2	425.74	233.74	70.52	16.10	105.38
101	2	2	434.84	238.71	72.02	16.45	107.67

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