THE INTERACTIONS BETWEEN MATURITY TYPE AND PRE-FEEDLOT PLANE OF NUTRITION ON THE GROWTH AND PERFORMANCE OF STEERS

by

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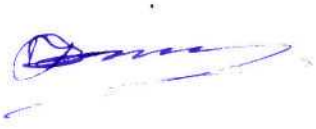
I hereby declare that the research reported in this study is my own work. Where use was made of the work of others it has been duly acknowledged in the text.



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"Some men are born great, some men achieve greatness, some men have greatness thrust upon them" (Shakespeare, Twelfth Night)

CONTENTS

ABSTRACT

The growth of tissues (bone, muscle and fat), along their natural growth curve, is controlled by a complex array of interactions. Growth gradients exist between the tissues and the body as a whole, bone being earlier maturing than muscle, and muscle being earlier maturing than fat. Growth waves within each tissue express its rate of deposition within in each area of the body. Differences between maturity types with respect to tissue growth, is that the earlier maturing animal is further along its normal growth curve. Comparison between maturity types must therefore be performed at an equal physiological age.

Restriction of nutrients to the growing animal results in an alteration of the body composition. The most affected tissue being the tissue with the highest growth impetus at the time. Severe restriction (loss of liveweight), can result in the tissues following a reverse order of their deposition. Re-alimentation results in tissue growth at a rate superior to animals of an equal chronological age. Those tissues that were most restricted in their growth, are the ones that show the most compensation.

A trial was carried out, to examine, the question of animals of differing pre-feedlot planes of nutrition, and maturity type, performances within the feedlot at an equal physiological age. Four treatments were used comprising earlier maturing fat and thin animals and later maturing fat and thin animals. Earlier maturing comprised Hereford and Sussex breeds, while later maturing comprised Charolias and Simmentaler breeds. The pre-feedlot planes of nutrition were imposed for 103 days, with the fat animals gaining at 0.40 kg per day and the thin animals at 0.10 kg per day. Both treatment groups lost condition but started the feedlot period significantly different with respect to liveweight and condition.

Physiological age was to be determined, by the weight of the animals body protein, as a proportion of the maturity types, mature body protein weight. The urea dilution technique was used to determine the body composition of the treatments at any one point of time. Due to complications with the application of the technique, the body

compositions of the animals were not determined with any degree of certainty. Thus it was impossible to compare the feedlot performances of the treatments at equal physiological ages, or compare their changes in tissue weights over time.

Differences in tissue deposition rates were measured. Compensating animals had significantly higher growth rates in terms of height. This equates to a higher rate of bone tissue growth. As height measurements were not taken during the pre-feedlot period this could not be attributed to compensatory growth. Ultrasonic measurements of eye-muscle diameters, showed that the compensating animals and the later maturing fat animals to be growing at a non-significantly different rate. This could, possibly, be due to the animals depositing at the maximum allowable rate. Subcutaneous fat deposition was measured as change in condition score. Compensating animals deposited fat at a significantly faster rate, with no significant differences between maturity types being apparent.

All animals were slaughtered at a set condition. This resulted in the early maturing fat animals, spending a significantly shorter period of time in the feedlot. Analysis of subcutaneous fat depths found no significant differences between treatments, indicating that the animals had been slaughtered at equal condition scores. Fat distribution over the measured sites however showed that there were significant differences between the compensating animals. The earlier maturing being to fat and the later maturing being to thin. Thus the two groups could have spent a shorter or longer period of time in the feedlot respectively.

Liveweight changes over time were as predicted. The late maturing treatments had significantly higher growth rates than their respective earlier maturing treatments. The compensating treatments also had significantly higher growth rates than respective non-compensating treatments. Only the early maturing thin animals managed to make up the deficit sixty kilograms.

Feed and net energy available for growth (NEg) intakes, were complicated, with anomalies being found in the data distribution. Animals feed and NEg intakes increased

linearly, before peaking at the same point of time irrespective of maturity type, and then followed a linear trend of significantly different slope. The quadratic model failed to follow the data trend accurately enough so the broken stick model was used. Compensating animals ate significantly more food and NEg per kilogram of metabolic weight ($W^{0.75}$). There utilisation of the food and NEg was however significantly more efficient than the non-compensating animals. No significant differences were found between maturity types within pre-feedlot treatments.

Further investigation is required into the anomalies surrounding the analysis of the feed and NEg intake data. A biological justification must be found for the use of the broken stick model. The change in the linear trend after peak feed intake appears to be due to a restriction. This restriction should be determined as it affects animals irrespective of nutritional requirements.

CHAPTER ONE

LITERATURE REVIEW

1.1 INTRODUCTION

With 85 per cent of it's total area utilisable as grazing, the Republic of South Africa is preeminently a pastoral country (Van Marle, 1974). In economic terms this makes beef production an important contributor to the total agricultural product and a critical part of the agricultural resource base of the Republic of South Africa (Van Marle, 1974; Paterson, 1981). A shortfall between the supply and demand of beef is indicated by the need to import beef. This shortfall has been reduced by the increase in intensification primarily with the growth in use of feedlots. The use in feedlots has increased significantly since the 1950's, to become an important contributor to the South African beef industry (Paterson, 1981).

The past 150 years has been most noticeable for the marked change in the preferred beef cattle type and body composition. These changes in beef cattle type have been necessary to keep up with the utilization of the various component parts concluded Hedrick (1972). The result of this is the presence of animals that vary in mature size and physical form. "The ideal beef animal is one that is capable of efficient conversion of feed grains and roughage's into the maximum amount of consumer acceptable meat that is physiologically feasible," (Hedrick, 1972). It is, however unrealistic to expect there to be a change by beef producers to beef animals of all the same size and form. The beef producer of today is therefore faced with economically producing cattle that yield a high per cent of high priced, tender, flavoursome, juicy retail cuts that have a minimal amount of waste in the form of fat trim (Guenther *et al.*, 1965; Hedrick, 1972; Prior *et al.*, 1977).

Wheeler *et al.* (1989) advised that long term fat removal should be by selection and correct management of the types of cattle that produce a product with the desired palatability. In North American breeds the selection for a decrease in fat and increased growth rate has resulted in an increase in mature body size, (Notter *et al.*, 1979). This is in contrast to the tradition of using early maturing animals. These were heavily

fattened to produce a high degree of marbling which was presumed to be more acceptable. The optimum size of beef producing animals is therefore debatable.

Having established the existence of a selection of breeds with varying propensities for fat deposition at equal chronological ages, the management of ones breed choice therefore becomes the most important factor. Smith *et al.* (1976) identified post-weaning growth and feed efficiency of beef steers as the most important components of the net efficiency of the beef production systems. In an attempt to attain slaughter finish at desirable carcass weights, a growth phase for cattle is usually imposed between weaning and finishing in a feedlot. This growth is normally on natural forage with a consequent low cost of gain. Natural forage has cycles of nutritional quality imposed by the seasons. This creates a natural period of nutritional deprivation for the animals. The effect of the nutritional deprivation upon the economically important tissues within the body, and subsequent ability to recover when the animal is placed on an improved nutritional plane, is of great importance to the livestock producer. Sainz *et al.* (1995) reported that animals entering the finishing phase attract unit price premiums if perceived by buyers to have undergone a period of nutritional stress. This is due to an animal's ability to exhibit compensatory growth when placed on full feed, which relates to an improved feed conversion efficiency.

Williams *et al.* (1995), attributed differences in composition, retail products, and quality of beef carcasses to the interaction of genotype, weight, sex and nutritional environment. Prior *et al.* (1977) suggested that more research on the influence of nutrition, mature size, and rate of growth on carcass composition and palatability would lead to an improvement in total production efficiency.

1.2 LITERATURE REVIEW

The aim of this literature review is to examine some of the general descriptions of growth. These descriptions will then be used in an attempt to unravel some of the complexities surrounding growth in specific areas such as unrestricted, restricted, compensatory growth and finally breed and maturity type differences.

1.2.1 GROWTH

Due to the complex nature of the topic under review a large number of definitions have been used by researchers to describe growth. A definition proposed by Reeds and Fiorotto (1990), describes growth as a collection of time related phenomena (e.g. liveweight), which occur between conception and maturity. The liveweight changes over time follow a sigmoid curve, (Figure 1), the curve being composed of a prepubertal, self-accelerating phase and the post-pubertal, self-inhibiting phase, (Owens *et al.*, 1993). Growth can then be expressed in terms of liveweight, carcass weight or tissue (muscle, fat or bone) gain per unit time, (Berg, 1968). Relative growth then involves the changing relationships among and within the tissues relative to age, weight or stage of physiological development.

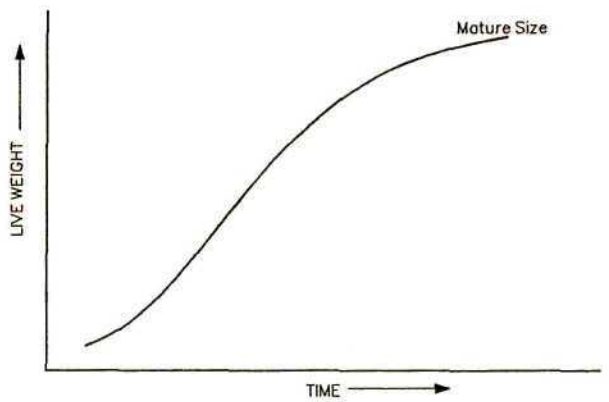

Figure 1. Growth unrestricted to maturity

Hammond (1960), in an attempt to standardize the definitions, described growth in the following way. As an animal grows, it will undergo an increase in weight up to a mature limit (this he called growth), and there will be changes in body conformation and shape (which he termed development). Reeds and Fiorotto (1990), further divided growth into three, the dimensional, the compositional and the developmental or functional. Ryan (1989), cited by Hogg (1991), described normal growth to be that where the increase in weight and size of the animal is at a potential maximum under the limiting restrictions of the animals environment. The initial growth in the animal is due to cell multiplication. As the increase in size, and hence growth, speeds up the continuing growth is due to the increase in size of these cells to their potential maximum. As with Hammond's definition, the accompanying process will be that of development. The description of an animals growth over time is best achieved with the use of the sigmoid growth curve (Figure 1).

1.2.1.1 **Growth Gradients**

On examination of carcasses by dissection, Hammond (1960) came to the conclusion that the proportional changes in the body are due to the different parts of the body growing at different rates. These rates of growth can be divided into growth gradients or growth waves. The primary growth wave starts at the head, spreading back to the lumbar region. The secondary waves start at the extremities of the limbs, passing upwards towards a junction in the loin region (Hammond, 1960).

Hammond (1960) extended this concept of growth waves to differences that exist in the growth patterns (growth gradients) of the different tissues. The growth gradients are initially made up of early maturing tissues, being the maintenance organs (nerve tissue, brain, alimentary tract). This is followed by later maturing tissues: bone; then muscle and finally, the latest maturing tissue, fat. This order of maturity provides for the tissue of greater importance at birth to be more mature than a tissue of less importance at birth. This work by Hammond illustrates the order of importance of each tissue at a set time within the body. Pálsson (1955), agreeing with the work performed by Hammond, showed that within the maintenance organs, the thoracic organs (eg. the heart), are

earlier maturing than the digestive tract. This division of an individual tissue(s) into maturity groups is an extension of the concept of a tissue's importance within the body. With the knowledge that there is an order to growth, it is possible to predict or model areas of growth impetus and their nutritional requirements.

1.2.1.2 Growth Model

"The Theory of Partition of Nutrients According to Metabolic Rate", proposed by Hammond, which described the supply of nutrients in order of demand by the organs, was modified by Berg and Butterfield (1976). They proposed an alternative model which incorporates the following points :

1) Priority will be allocated to different organs for the availability of nutrients for both maintenance and growth. Only under extremely low levels of nutrient supply, will the most vital organs have retardation or impairment of function.
2) If positive growth is maintained, this will ensure constant relative rates of growth between muscle and bone. The relative rates may however be altered by the protein : energy ratio of the diet.
3) The proportions of muscle to bone will be altered during body weight loss, this relative rate of depletion being affected by intake of energy as well as of protein.
4) The growth of fat relative to muscle and bone is dependent on the amount of energy in the diet. The higher the energy intake, the higher the growth rate of fat.
5) Fat, muscle and bone will be depleted during the loss of body weight.

This model, as proposed by Berg and Butterfield (1976), makes use of Hammond's growth gradients to allow for the partitioning of nutrients to the respective tissues, when the animal is in a positive or negative energy balance.

1.2.1.3 Mathematical Descriptions of Growth

The description of nutrient requirements of growth in a theoretical model (Berg and Butterfield, 1976) answers one of the questions of growth, ie which tissue has the highest

priority for growth and thus nutrient supply at any point in time? However, it is necessary to have a method of comparing the growth of a species, breed, tissue or organ against another. The ability to determine these comparative growth rates using mathematical descriptions of growth provides a useful tool to the researcher.

1.2.1.3.1 Allometric Relationships

Huxley (1932) proposed that there is a simple and significant relationship between the magnitude of the following two variables: the increase in relative size of organs; and the absolute size of the body that bears them. The following equation, from Huxley (1932), was proposed to describe this relationship:

$$\log y = \log b + k \log x$$

where **y** = the magnitude (weight) of the organ,
x = the magnitude (weight) of the body,
b = the value of **y** when **x** = 1.0,
k = the growth coefficient of the organ or the part.

"This implies that, for the range over which the formula holds, the ratio of the relative growth rate of the organ to the relative growth rate of the body remains constant, the ratio itself being denoted by the value of *k*. The relative growth rate is defined as the rate of growth per unit weight, i.e. the actual absolute growth rate at any instant, divided by the actual size at that instant" (Huxley, 1932).

This equation from Huxley has provided researchers with the means of determining relative growth rates. For example in comparisons performed by Berg and Butterfield (1976), the ratio of percentage post-natal growth of the organ, or part thereof, to the whole body is represented by the growth coefficient. This allows the expression of relative maturity to be expressed. Berg and Butterfield (1976) found the growth coefficient for bone in beef cattle to be low (<1.0), indicating early maturity and a low impetus for growth, where impetus refers to the force or energy with which the tissue

grows. Muscle was found to be intermediate (> 1.0), and fat high (1.5 to 2.0), indicating late maturity and a high impetus for growth.

1.2.1.3.2 Measures of Maturity

With the ability to determine comparative growth rates, a further requirement is the need to be able to compare animals from a point of equality. A commonly chosen point is that when the animal is mature in terms of growth. Maturity was defined by Butterfield (1988) as the state of anatomical equilibrium achieved when an animal has ceased to grow. The following two sections provides examples of methods to determine comparative growth rates on a level of maturity basis.

1.2.1.3.2.1 Genetic size-scaling

Taylor (1980) discussed the striking similarities that exist in the growth processes of different mammalian genotypes. Two genetic size-scaling rules were presented by Taylor (1980) as a simple but general procedure for introducing information on genotype differences into equations, experimental designs and quantitative calculations.

1) Treat all age and time variables for the *i*th genotype as directly proportional to $A_i^{0.27}$, where A_i is the mature body weight of the *i*th genotype;
2) At every age standardized as in 1), treat all cumulated inputs and outputs for the *i*th genotype as directly proportional to A_i

These genetic size scaling rules imply that, in comparison to small animals, larger ones consume and produce more in proportion to their mature body weight, but take longer to do so in proportion to the 0.27th power of their mature body weight. The time component is particularly important, because it balances the large animals' advantage of maintaining themselves more efficiently or growing more rapidly, against the longer time that they would take to mature, attain puberty, or reach an optimal slaughter weight.

The time component of the scaling rule for time variables describes the concept of *metabolic age*. The corresponding concept for cumulated growth variables is *degree of maturity*. The combination of these two concepts allows for genetic comparisons to be made at the same metabolic age or at the same degree of maturity and are therefore independent of adult size (Taylor, 1985).

1.2.1.3.2.2 Maturity coefficient

Butterfield (1988) introduced a *maturity coefficient* for the comparative description of growth. The maturity coefficient was developed to aid the description of the effect that mature size has on the growth patterns of body components. Once the composition of mature animals has been determined, analyses of progress to maturity can be described in terms of units of mature weights of both the whole animal and its component anatomical structures (Butterfield, 1988). The maturity coefficient can be calculated using the following equation :

$$y = qx + (1-q)x^2$$

where **y** = the weight of the organ divided by its own mature weight (I/I_m).
x = the weight of the total (animal or tissue) divided by its own mature weight (T/T_m).
q = the relationship between **y** and **x**

" A 'q' value greater than 1.0 means a lesser rate of growth, i.e., "low impetus" relative to that of the whole animal and therefore a declining proportion of the whole. A 'q' value less than 1.0 means a greater rate of growth, i.e., "high impetus" relative to that of the whole animal and therefore an increasing proportion of the whole. A 'q' value not different to 1.0 means that the structure and the whole are growing at the same relative rate, i.e., "average impetus" and that therefore the proportion of the part to the whole remains unchanged" (Butterfield, 1988).

1.2.1.4 Normal Patterns of Growth

Normal patterns of growth will be described within the context where no environmental factors are affecting the animals ability to meet its genetic capacity for growth. This follows Paterson's (1981), description of growth as, that amount of genetic potential allowed to be expressed over a given time within the bounds of the existing environment. The theoretical models and mathematical descriptions of growth already covered will provide a basis for the explanation of growth anomalies found in the following sections.

The description of patterns of growth will centre on the carcass being the end product for producers. Anything that affects growth within the carcass is therefore of paramount importance to producers in their quest to provide the consumers with their desired product, as profitably as possible. The composition of the carcass can be divided largely into bone, muscle and fat. It has been determined that the quantitative requirements in a steer carcass are best met when the proportion of muscle is at a maximum, bone is at a minimum and fat is at an optimum which is determined by local consumer preferences.

Figure 2, shows the relation between the increase in liveweight and that of carcass weight and its constituents. It can be seen that the increase in carcass weight is strongly correlated with the increase in liveweight. The following sections will concentrate on the normal growth patterns of the carcass constituents.

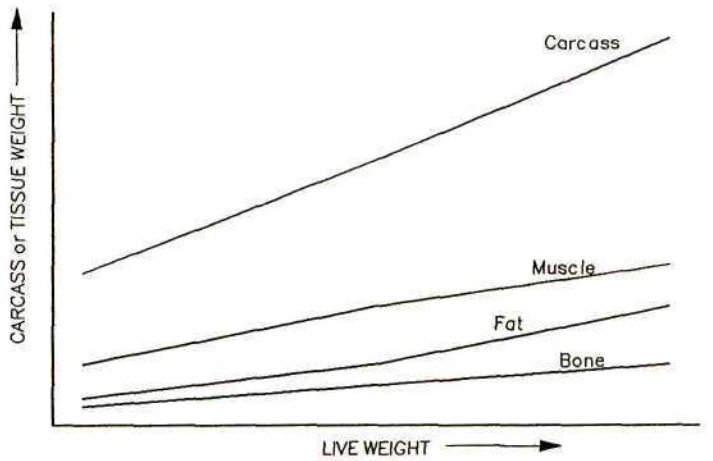

Figure 2. Growth of carcass and carcass tissues relative to liveweight (after Huxley, 1932)

1.2.1.4.1 Bone

At birth the skeletal structure needs to be at a high level of maturity for it to be effective in its function. Bone is one of the hard tissues making up a vertebrate animal's skeleton. It is therefore an essential tissue at birth. From Hammond's breakdown of tissue maturity gradients, it has been found that bone is at a higher level of maturity than the other tissues (muscle and fat). Due to its relative maturity at birth post-natal growth of bone is at a steady, but slow rate. The rate of growth was illustrated by Butterfield (1988) when he allocated bone a maturity coefficient 'q' of 1.4 using rams as an example. Bone may be increasing over time in relation to its original weight, but it is however becoming becoming a decreasing proportion of the total carcass (Figure 2).

Pálsson (1955) described the growth of bone in terms of external body measurements, the following three growth groupings were obtained. The least growth in the post natal stage is shown by the skull, leg length and height at the withers, the most growth by width of the hind quarters, with the length and the depth of the body being intermediate.

Pálsson (1955) mapped the following growth trends, matching the growth gradients suggested by Hammond. In the axial skeleton, the cranium is the earliest developing part, and from it the primary waves of increasing growth intensity pass backwards to the lumbar region and downwards to the nose and lower jaw. In each limb, secondary growth waves pass with age from the early developing metacarpals and metatarsals down to the distal bones and up towards the lumbar region, the pelvis and the scapular being later developing than the femur and the humerus. The ribs are found to be the latest maturing bones of the body. The bones of the fore-limb, as a whole, are somewhat later maturing than those of the hind limb.

The growth in length and thickness of the long bones of the limbs follow the same trend as growth in weight. The bones further along the secondary growth wave, increase relatively more both in length and thickness in postnatal life than the bones found nearer the start of the growth wave. Growth rate in length attains its maximum at an earlier age than growth rate in thickness (Pálsson, 1955).

1.2.1.4.2 Muscle

The musculature is made up of contractile fibrous bands or bundles that produce movement in an animal's body. Growth in the muscles that occurs in the post-natal period is largely due to increase in muscle fibre size (Hammond, 1960).

Figure 2 illustrates that muscle has a faster growth rate than bone during post-natal growth. The growth rate of muscle tissue leads to an increase in the proportion of muscle in the total carcass, until the period of increased fat deposition where muscle reduces its proportion in the carcass (Berg and Butterfield, 1976). Butterfield (1988) classified the overall growth of muscle tissue with a 'q' value of 1.3.

At birth a calf's muscles are utilised as a mechanism to augment its limited energy stores to survive. That is for the use of walking and suckling. The immediate needs of a new born calf are different from those of a mature bull. A transition must take place, in which there is differential muscle development (Berg and Butterfield, 1976). Division of

total muscle growth into four time periods was proposed by Berg and Butterfield (1976). The periods being the ante-natal, the immediate post-natal, the pre-pubertal and finally the maturing phase.

The ante-natal period of growth is thought to be controlled by a genetic template. This template ensures that at birth the animal can meet the challenges of the environment to its best capabilities as determined by its evolutionary development. Muscle growth is probably stimulated by passive tension from skeletal elongation (Berg and Butterfield, 1976).

The immediate post-natal phase involves the doubling to quadrupling of the birth weight of the musculature with the extent of development being largely influenced by the function of the muscle's. This development is similar to animals within the species. Differences that do occur are largely due to differences in function e.g. growth in abdominal wall muscles can be influenced by the physical nature of the diet (Berg and Butterfield, 1976). The pre-pubertal and adolescent phase is categorised by a large increase in muscle size with little change in relative weights associated with little change in function (Berg and Butterfield, 1976).

Musculature alters dramatically in the male during the maturing phase. This is to produce an animal with a dual responsibility of survival and of fighting to reproduce. The combination of androgens and the functional requirements to mate are thought to control this musculature development. Steers having been castrated have an inadequate supply of sex hormones and therefore only achieve a small proportion of the musculature development that a bull achieves during this phase (Berg and Butterfield, 1976). Only the post-natal and pre-pubertal / adolescent periods will be considered in this review.

The development of muscle in the different body regions is also governed by growth gradients similar to those in the skeleton. A wave of increasing growth intensity passes with age from the head and neck backwards and from the lower parts of the limbs (fore and hind limbs) upwards to the loin region (Pálsson, 1955). Berg and Butterfield (1976) showed that there was a tendency for small muscles to grow at a proportionately slower

rate than large muscles. This, they concluded, was due to the larger amount of connective tissue found in small muscles which has a lower potential for growth than muscle fibres.

Bone on a comparative basis with the other tissues is a low value product. Hence, the real value of a carcass can be measured by the ratio of muscle to bone (Butterfield, 1966). From the differing growth rates between bone and muscle Berg and Butterfield (1966) have been able to quantify that the ratio of muscle to bone increases at an estimated 0.03 per ten kilogram increase in carcass weight.

1.2.1.4.3 Fat

Fat is defined in this section to be the adipose tissues of the animal. The major biological roles of this tissue being as an energy store and as an insulator.

The proportion of fat in the carcass increases at a constant level, until an accelerated period of deposition (Figure 2). The accelerated period of deposition is termed the *fattening phase*. During this fattening phase the large increase in the deposition of fat tissue is at a rate greater than that compared to the other tissues in the carcass (Murray *et al.*, 1974; Berg and Butterfield, 1976; Baker *et al.*, 1991).

Pálsson (1955) explained the development of the fat deposition sites in the different body regions as also governed by growth gradients similar to those in the skeleton. A wave of increasing growth intensity passes with age from the head and neck backwards and from the lower parts of the limbs (fore and hind limbs) upwards to the loin region (Pálsson, 1955). As in the previous two tissues a definite order to fat deposition has been established. In Figure 3, Johnson *et al.* (1972) have graphically represented the changes in relative contribution to total side fat by the five fat deposition sites namely, intermuscular, subcutaneous, intramuscular, kidney and channel.

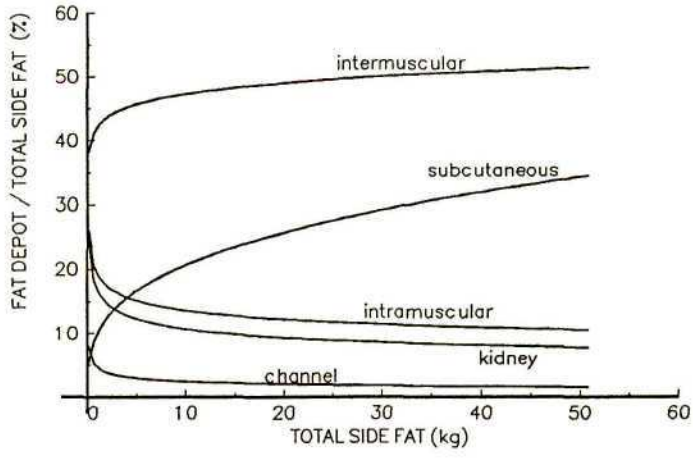

Figure 3. Relative contribution of the five fat depots to total side fat weight with increase in total side fat weight (after Johnson *et al*, 1972)

The kidney and channel sites were found to be at their greatest proportion in relation to total side fat at the lightest carcass weights. As carcass weight increased, their contribution became minimal. This led to the conclusion that in the early stages of fattening these two sites are preferentially deposited within (Hammond, 1960; Johnson *et al*, 1972). Intramuscular fat was found by Johnson *et al.* (1972) to make its greatest contribution in the lightest sides decreasing to a minimum in the heaviest sides. This is in contradiction to Hammond (1960) who found that the intramuscular fat site was the last to be deposited into. Figure 4, shows that there is continual deposition in all three of these sites. The rate of deposition is however lower than that of the total side fat. Consequently these three sites of deposition make up a decreasing contribution to the total side fat. Fat deposition in the intramuscular site is commonly known as marbling. Measurement of this site is normally by subjective visual assessment or chemical analysis. Johnson *et al.* (1972) used the chemical analysis and Hammond (1960) seems to have used the visual scoring method. The differences in their results could be due to Hammond only being able to determine intramuscular fat deposition once enough had been deposited for it to be seen.

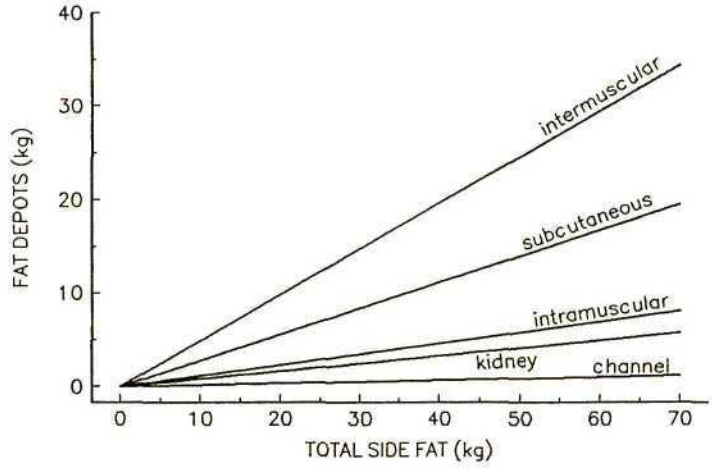

Figure 4. Changes in the weights of five fat depots relative to total side fat weight (after Johnson *et al.*, 1972)

Subcutaneous fat initially made the least contribution to total side fat before increasing rapidly in the final two weight classes (Figure 3). Intermuscular fat was the greatest contributor at all four side weight groups. In Figure 4, the rate of fat deposition for the subcutaneous and intermuscular sites is much higher than in the other fat sites, which explains why they make up an increasing proportion of the total side fat.

The chemical composition of the whole empty body of an animal represents a final state resulting from the influence of heredity and environment. In the usual nutritional or physiological experiment, attempts are generally made to control genetic influences by the use of large numbers of animals, random allotment, replication and other devices. Changes in the composition of the body (reflecting storage or loss of chemical components) become valuable criteria of response to the environmental treatments being imposed (Reid *et al.*, 1955). The growth patterns of the tissues described are closely matched by the major chemical components namely, protein, water, ash and fat. For example muscle contains all of these components. Therefore it is necessary to consider growth with respect to the chemical components of the body due to variation that may occur during weight loss and realimentation (Berg and Butterfield, 1976). Moulton 1923 cited by Reid *et al.*, (1955) introduced the concept of "chemical maturity", which he

defined as the age at which the concentration of water, protein and mineral matter in the fat-free cell becomes practically constant.

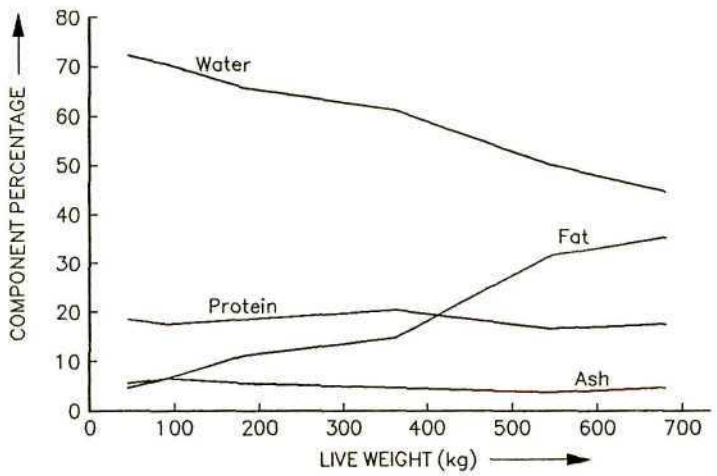

Figure 5. Chemical composition of steers as per cent of empty body weight (after Haecker 1920 cited by Berg and Butterfield, 1976)

1.2.1.4.4 Water

Water is the major component of the body and increases steadily in weight as an animal grows (Haecker, 1920 cited by Berg and Butterfield, 1976; Rule *et al.*, 1986). Water content can vary from approximately 80 to 40 per cent of empty body weight, Figure 5 (Berg and Butterfield, 1976). This is due to the decrease in water concentration as animals grow (Haecker, 1920 cited by Berg and Butterfield, 1976; Reid *et al.*, 1955; Seebeck and Tulloh, 1969) which was shown to be from 71.84 per cent at 45 kg in weight to 43.48 per cent at 680 kg in weight. The cause of this reduction in water proportion is due to the decreased weight of water deposited in the body per kilogram increase in weight (Haecker, 1920 cited by Berg and Butterfield, 1976). Figure 6, illustrates the proportion of total water that occurs in each tissue as the animal increases in weight. Water deposition occurs in those tissues still actively growing i.e. muscle tissue. In bone tissue the proportion of water found decreases due to the relatively slow growth found in the bone tissue. The change in distribution of water as total carcass side water increased was slight (Seebeck and Tulloh, 1969).

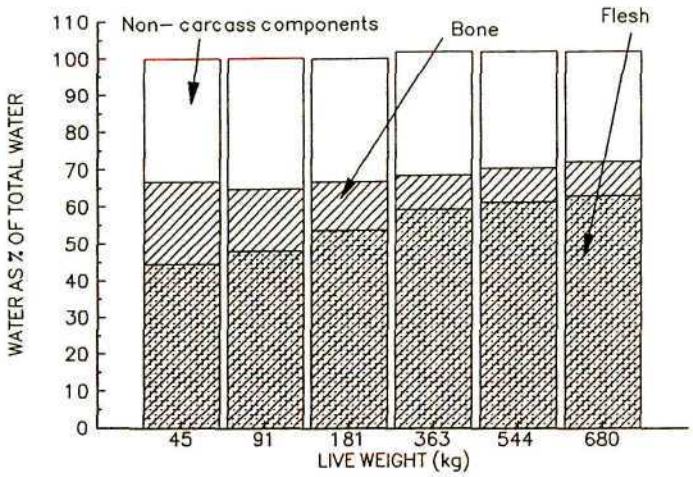

Figure 6. Distribution of water in the empty body of cattle (after Haecker, 1920 cited by Berg and Butterfield, 1976)

1.2.1.4.5 Protein

The rate of protein deposition during post-natal growth, is primarily dependent on the degree to which the dietary protein supply allows the animal to attain its goal to achieve a rate of protein deposition as close as possible to its genetic maximum (Reeds and Fiorotto, 1990). The concentration of protein increases in the fat-free body until reaching a constant value for that species (Reid *et al.*, 1955; Berg and Butterfield, 1976). This supports a definition for the mature state, "the mature state is one in which the relationship between the cell protein and cell water has reached a stable level" (Reeds and Fiorotto, 1990).

Protein shows a fairly steady increase relative to liveweight as can be seen in Figure 5. Protein is however being deposited at a lower rate, which results in protein becoming a decreasing component as a proportion of the carcass. Support for this was also found by Seebeck and Tulloh (1969), who showed the proportion of protein in the jointed side weight of the carcass (the left side of the carcass divided into 13 commercial butcher's cuts) decreased as the jointed side weight of the carcass increased.

Protein deposition follows Hammond's growth gradients. During developmental growth the relative growth rate of protein was higher for the loin and lower for the silverside than for total carcass side protein. However the overall changes in distribution of protein as total carcass side protein increased were slight (Seebeck and Tulloh, 1969).

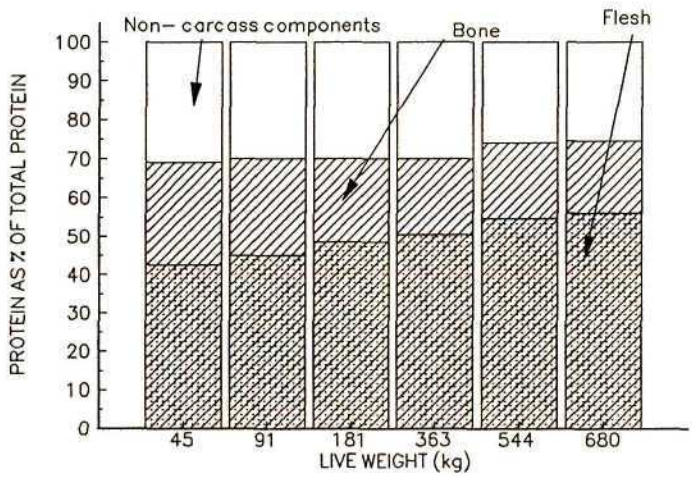

Figure 7. Distribution of protein in the empty body of cattle (after Haecker, 1920 cited by Berg and Butterfield, 1976)

From Figure 7, protein deposition occurred at a higher proportion in the flesh, lower proportion in non-carcass components and the lowest proportion in the bone. As the weight of the animal increased the flesh acquired an increasing protein proportion, the bone a decreasing proportion and the non-carcass components remained constant. The rate of protein deposition again matches those tissues which are at a greater impetus of growth.

1.2.1.4.6 Chemical Fat

The deposition of fat is a process of accumulation of energy with little accompanying deposition of water. The level of fat deposition is therefore a measure of the degree to which the level of energy intake exceeds the amount of energy utilised by maintenance and deposition of protein (Reeds and Fiorotto, 1990). Butterfield (1966) put another proviso upon this statement being that, the amount of fat in a carcass is due to a balance between the maturity type of the animal and the level of nutritional intake. This provides

for the differences between animals with regards to the existence of maturity types with respect to weight and composition (see section 1.2.1.7).

Koch *et al.*, (1979) reported that fat deposition was a linear function over time at least until early adulthood, with deviation from linearity after the first few months of age being very slight. However, Reeds and Fiorotto (1990) reported that the amount of chemical fat increases at an accelerating rate, slowing some what in the heavier animals. Figure 5 illustrates a combination of these reports. Fat deposition appears to follow a linear function over time in early post-natal growth before following an accelerating rate of deposition.

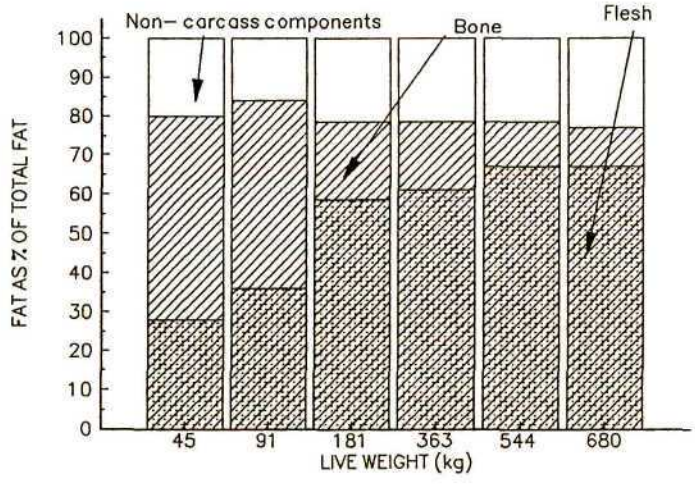

Figure 8. Distribution of fat in the empty body of cattle (after Haecker, 1920 cited by Berg and Butterfield, 1976)

As liveweight increases (Figure 8), the proportion of total fat found in the flesh increases substantially. The proportion of total fat found in the bone decreases and that in the non-carcass components remains constant. The explanation for this is that during post-natal growth most of the fat is being deposited in the flesh.

As the jointed side weight of the carcass increases, the chemical fat proportion increases. Changes in the distribution of chemical fat probably reflects changes in the distribution of subcutaneous and intermuscular fat that also occurs during growth (Seebeck and Tulloh, 1969). The major effect of fattening upon the concentration of water, protein and

mineral matter in the whole empty body is that of dilution, thus reducing their proportional contribution to the empty body (Reid *et al.*, 1955).

1.2.1.4.7 Ash

Ash is defined as the residue or inorganic constituents of a sample after a known weight of sample is ignited at 500°C until all carbon has been removed. The concentration of ash increases in the fat-free body until reaching a constant value for that species. Ash shows a fairly steady increase relative to liveweight (Reid *et al.*, 1955; Berg and Butterfield, 1976). The proportional increase of ash as compared to the increase in the jointed carcass side weight was found to be non-significantly different (Seebeck and Tulloh, 1969). When compared to total carcass side, with ash increasing, the distribution of ash changed in relation to bone weight and to reflect the early maturity of the leg bones and the later maturity of the axial skeleton (Seebeck and Tulloh, 1969).

The importance of proportional tissue growth is the increase in the ratio of muscle to bone due to the higher post-natal growth rate of muscle. This effect is diluted by the potentially high deposition of fat. The requirements of these tissues is closely matched by their chemical constituents. The chemical constituents are potentially vulnerable to nutritional variation leading to tissue changes.

1.2.1.5 Restricted Patterns of Growth

Seasonal fluctuations in the quantity and/or quality of natural forages can result in an animal's growth rate falling behind its genetic potential growth rate or even becoming negative. This condition is defined as *restricted growth* (Hogg, 1991).

In Figure 9, three scenarios on the possible effects of nutritional deprivation are illustrated. The first scenario is one in which the limiting nutrient causes a reduction in the growth rate, with the growth rate still remaining positive. This was defined as *mild restriction* by Wilson and Osbourn (1960). The second scenario is one in which the

limiting nutrient has resulted in a complete lack of growth, as seen by the maintenance of the liveweight. Wilson and Osbourn (1960) defined this scenario as *restriction*. As with Hammond's (1960) definition, growth in terms of increase in weight of the animal may be stationary but growth in terms of developmental changes within the animal will continue. The third scenario is the extreme condition suggested by Hogg (1991), in which, due to nutritional deprivation, there is a reduction in liveweight. Wilson and Osbourn (1960) defined this as *severe restriction*. Again, this does not exclude the possibility of developmental changes continuing.

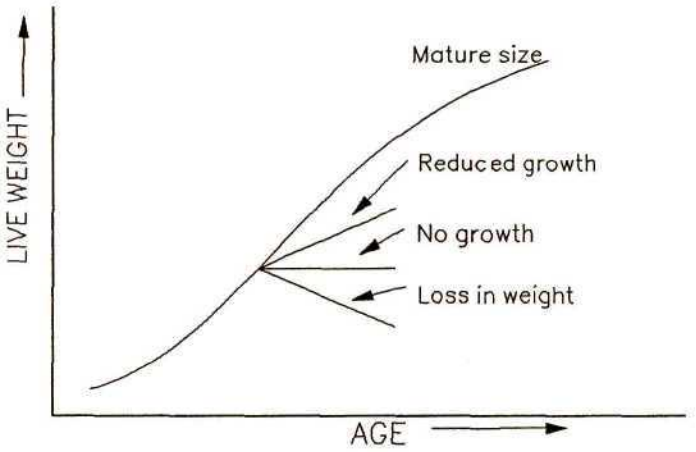

Figure 9. Normal growth showing three growth restriction possibilities induced by nutritional deprivation

Where possible the limiting nutrient will be identified in bold type within brackets i.e energy (**E**), protein (**P**), if this is not known and a restrictive feed intake was practised (**RI**) will identify this situation.

1.2.1.5.1 Bone

As described in 1.2.1.4.1, bone is an early maturing and priority tissue to receive nutrients. Thus *mild restriction* is expected to have a limited effect on bone growth. As the restriction becomes more limiting (*restriction* to *severe restriction*), there should be an increasing effect on bone growth, as expressed in Berg and Butterfield's model.

The work by Waters (1908 and 1909) cited by Pálsson (1955) provided the first proof of the priority claim that bone has on available nutrients. Bone growth was found to continue (at least in length), at the expense of previously stored up reserves (fat). A large number of researchers have since supported Waters findings. Animals that have been raised on differing planes of nutrition are found to have the same weight of bone when slaughtered at a set age or set liveweight ((**RI**) Butterfield *et al.*, 1971; (**RI**) Sully and Morgan, 1982). However, as predicted in Berg and Butterfield's model there is an increasing effect on bone growth as nutrient restriction increases ((**P**) Joubert, 1954; (**E,P**) Carroll *et al.*, 1963; (**E,P**) Guenther *et al.*, 1965; (**RI**) Henrickson *et al.*, 1965; (**E,P,RI**) Seebeck and Tulloh, 1968; (**E,RI**) Dockerty *et al.*, 1973; (**E,P,RI**) Seebeck, 1973; (**RI**) Murray *et al.*, 1974).

Seebeck and Tulloh (1968) (**E,P,RI**) reported that bone weights remained relatively constant, with the rate of change varying significantly with the size of the animal before weight loss (Figure 10). In their trial, two groups of steers were raised on similar planes of nutrition; one group being slaughtered at set weights. The other group was then forced to lose weight until having reached the same liveweight as that reached by the other group which had been slaughtered previously. At lighter weights, a negative change in bone weight was found when the animals were forced to lose weight. This trend was reversed as the weights of the slaughter group increased, with the bone showing a positive change in bone weight (becoming an increasing proportion of the carcass). Reasonably fat steers, when made to lose weight, have only a small actual weight loss from bone (Berg and Butterfield, 1976).

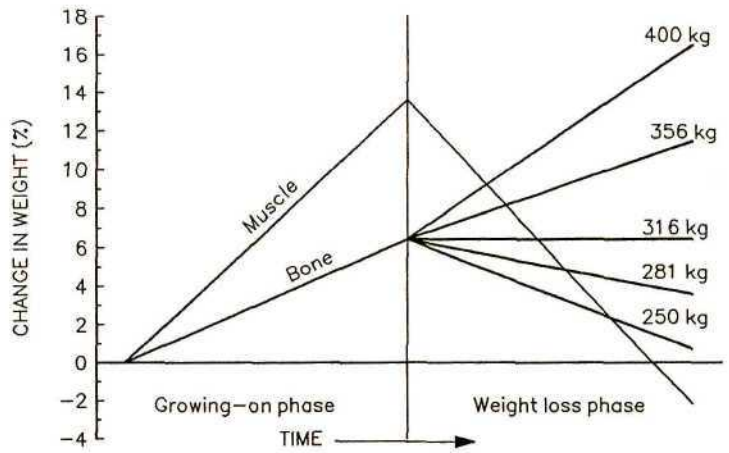

Figure 10. Percentage changes in weights of muscle and bone during the growing-on and weight loss phases, relative to their weight at the beginning of the growing-on phase (after Seebeck and Tulloh, 1968)

Results of the trial, shown in Figure 10, map the maturity expectations in that bone will be more susceptible to nutrient restrictions at lighter weights, being an early maturing tissue. As the animal matures and increases in weight any decrease in weight will affect the other tissues (muscle and fat), which are now the main areas of growth.

As covered in 1.2.1.1, Hammond described tissue maturity differences, as well as demonstrating the growth waves within the body. Bone tissue as a whole is found to be earlier maturing than the other tissues. The growth waves within the tissue provide for some bones being earlier maturing than others. Thus, having established that there is limited overall effect on bone growth by nutritional restriction, investigation of the effect within growth gradients is necessary. As reported by Waters (1908 and 1909 cited by Pálsson 1955), conformational alterations were found even though bone growth continued.

As with the three tissues covered (bone, muscle and fat) those areas of latest maturity are the ones most affected by nutritional restriction. From 1.2.1.4.1, the areas of late maturity are the lumbar region and pelvis. Growth in length of bones is found to be

unaffected, with steers continuing their growth in height at the withers, as well as in their length of head. Restricted growth in the late maturing areas will result in reduced width measurements at the hips and chest. The final animal is one that has become relatively large, but narrow and thin. When Joubert (1954) (**P**), compared animals reared on differing levels of nutrition, his results were similar to those of Waters (1908 and 1909). He found the animals on the lower plane of nutrition retained their juvenile form, with a body which was leggy, narrow and shallow especially in the hindquarters, and with a long large head. Joubert concluded that within bone development there are maturity gradients, in that the growth in the width of the animal must be of a later maturing growth phase than height, as this is where the nutritional deprivation affected the bone growth the most. A change in component distribution was illustrated in a report by Seebeck (1973) (**E,P,RI**) where he found minor changes in bone distribution in the leg bones as compared to the axial skeleton. Thus the areas of late maturing bone growth, as established during normal growth patterns, are most affected by nutritional restrictions.

An analysis of the literature concerning the effect of nutritional deprivation on bone growth it can be seen that the results closely match the predictions made in the Berg and Butterfield model. That is, the loss attributed to bone weight is lower than that to any of the other tissues due to bone having a higher priority on the nutrient supply. Only severe nutritional deprivation resulted in there being any actual losses in bone weight. At any of the other nutritional levels the effect on bone growth was to either slow down the growth rate or to have an effect purely on a conformational basis within the maturity gradients of bone growth. Bone growth allowed to continue during an overall check in growth, results in a lengthening of the growth and fattening phase. This leads to an increased bone percentage at a set slaughter weight, (Hammond, 1960; (**RI**) Murray *et al.*, 1974). This they concluded was due to bone growth being related to age and carcass weight. Therefore, growth of bone, being an earlier maturing tissue, and not at a high impetus of growth due to its a high degree of maturity at birth, is relatively unaffected by nutritional deprivation.

1.2.1.5.2 Muscle

With differing severities of nutritional restriction, the response by muscle tissue will also differ. The response by muscle is further confounded by the type of limiting nutrient (protein or energy), as well as the measurement time (at a set weight or chronological age).

The general conclusion reached by researchers where the animals were slaughtered at a set weight, as compared to the controls, and not at a set chronological age, is that when animals are exposed to a low plane of nutrition, the lean proportion of the carcass increases (Wellington *et al.*, 1954; **(RI)** Henrickson *et al.*, 1965; **(E)** Waldman *et al.*, 1971; **(E)** Hironaka *et al.*, 1979; **(RI)** Sully and Morgan, 1982; Berge, 1991).

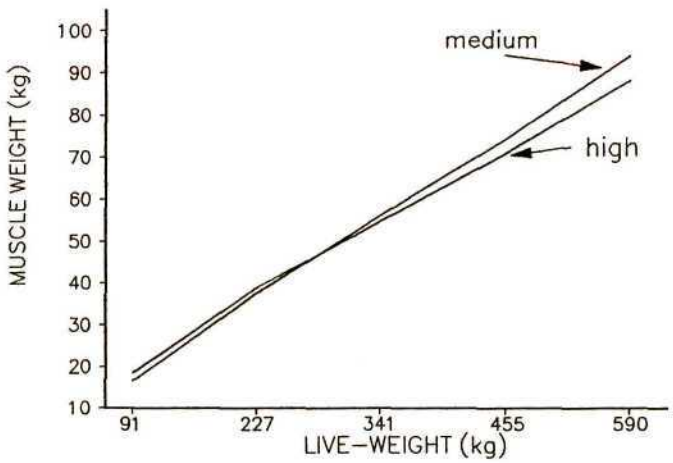

Figure 11. The effect of differing nutrient levels on muscle tissue growth, relative to liveweight (after Waldman *et al.*, 1971)

Figure 11, illustrates this general conclusion. Waldman *et al.* (1971) compared treatments involving differing nutritional planes indicated that at slaughter weights of 91, 227 and 341 kg's there was no significant difference in lean percentage in the carcass. However, at the slaughter weights of 455 and 590 kg's, there was more lean tissue in those animals on a lower nutritional plane.

These trials have, however, compared the lean contents of the carcasses at a set slaughter weight. This allowed the restricted animals to reach the slaughter weight over a longer period of time, which meant that the restricted animals could reach the same or greater levels of lean tissue at a slower deposition rate.

The trends shown in muscle growth rate over liveweight are apparent in Figure 2. The reduction of the contribution to the liveweight made by muscle tissue as it reaches its maximum deposition rate which then remains constant, allows for an increased level of deposition of fat tissue. Waldman *et al.* (1971) (**E**) in Figure 11, showed that on a high plane of nutrition, the proportion of the liveweight made up by muscle is reduced at higher liveweights due to the dilution effect of fat deposition. The composition of growth in a restricted animal is one of a greater proportion of bone and muscle and a lower proportion of fat than for an animal exhibiting normal growth. Growth along these proportions allows for a continuation of deposition of muscle tissue as the animal attempts to maintain the muscle : bone ratio with a normal bone growth rate and a slower muscle growth rate. Therefore, at a set liveweight the animal that was restricted in growth will have significantly more lean in the carcass.

Berg and Butterfield (1976) in their model on the distribution of nutrients for growth allowed for a constant relative rate of growth between muscle and bone as long as there is positive growth. From the literature it can be concluded that animals grown on a lower plane of nutrition, but with a positive growth maintenance throughout, have a higher level of lean tissue as compared to controls. This supports the model.

The comparison in lean tissue deposition rates of steers at set chronological ages under differing nutritional levels, shows that under a lower plane of nutrition the lean deposition rate is reduced when measured against that achieved by animals with unrestricted growth. This results in the animals on the lower plane of nutrition having a lighter muscle weight at a set chronological age ((**E,P**) Guenther *et al.*, 1965; (**RI**) Henrickson *et al.*, 1965; Berg and Butterfield, 1976). The lean weight of cuts obtained from eight month old calves increased progressively as nutrition increased ((**RI**) Stuedemann *et al.*, 1968). Carroll *et al.* (1963) (**E,P**), Berg and Butterfield (1968) and

Dockerty *et al.* (1973) (**E,RI**) found that the nutritional limitations placed on their animals resulted in no lean growth or a reduction in muscle growth as compared to the controls.

Wellington *et al.* (1954) contradict this finding, with age producing no consistent influence on percentage weight of muscles in the carcass. The treatments imposed on these animals must have therefore not affected muscle growth but must have severely limited fat deposition.

The imposition of severe restriction shows that muscle tissue deposition can cease and even reverse. Animals forced to lose liveweight had a significantly lower muscle content as compared to animals grown at a normal growth rate and of the same weight ((**E,P,RI**) Seebeck and Tulloh, 1968). This trial is illustrated in Figure 10. This loss of muscle weight was at a rate greater than the loss of weight from the carcass as a whole. Seebeck (1973) (**E,P,RI**) expounded on these findings by showing that live- weight loss resulted in an approximate reversal of the growth path of development of muscle. It was also shown that liveweight loss affects the muscle-weight distribution. The relative proportion of abdominal muscle weight having the most marked affect by falling in comparison to that of control animals. The loss of muscle weight from the more valuable loin and silverside was larger than the relatively smaller loss from the less valuable fore shin and chuck ((**E,P,RI**) Seebeck and Tulloh, 1968). Murray *et al.* (1974) (**RI**) stated there were no differences in the total weight of expensive muscles and the proportion of shin muscle to total muscle weight will be smaller in younger cattle (those on a higher plane of nutrition) than older cattle (those on lower plane of nutrition). Lalande and Fahmy (1975) measured the area of the *longissimus thoracis* and concluded that, the faster the growth of an animal, the larger the area of the respective muscle. The growth gradients that exist within the body and tissues show that the affected tissue, at the time of restriction, will be those at a higher level of impetus. Berg and Butterfield (1976) summarised this muscle weight loss, by saying that, those muscles which are essential for survival are least affected, and those muscles less essential for survival more affected.

Using measurements of growth patterns of muscle Lohse *et al.* (1973) attempted to differentiate between normal and recovering muscle in wethers. This demonstrated that those muscles of a low impetus showed no significant differences between groups of wethers on differing nutritional levels whereas those muscles of a high impetus were highly significantly different ((**RI**) Lohse *et al.*, 1973).

Table 1. Composition of weight change in muscle and fat during uninterrupted growth (C1-C2), semi-starvation (C1-S) and recovery (S-R) of Poll Hereford steers (after Berg and Butterfield, 1976).

TREATMENT	MUSCLE kg	FAT kg	Ratio of muscle gain or loss to fat gain or loss
Uninterrupted growth (C1-C2)	+ 16.8	+ 9.8	1.71
Semi-starvation (C1-S)	- 11.3	- 9.6	1.18
Recovery (S-R)	+ 18.2	+ 6.5	2.80

When the results Table 1, were examined by Berg and Butterfield (1976), the effect of semi-starvation was found to reduce the muscle weight and fat weight by a similar amount. However, the percentage lost as a proportion of their original weight was 21% for muscle and 70% for fat.

That animals strive to maintain a muscle : bone ratio can further be seen in Table 2. The restricted diet had no significant effect on the overall muscle : bone ratio at any of the slaughter weights.

Table 2. Muscle : Bone ratio for calves reared on three levels of fresh and reconstituted milk and slaughtered at seven weights (after Butterfield *et al.*, 1971)

GROUP	PREDETERMINED LIVEWEIGHT FOR SLAUGHTER (kg)							
	44	48	54	61	68	75	82	MEAN
HIGH	2.18	2.34	2.15	2.51	2.43	2.56	3.09	2.46
MODERATE	2.07	2.05	2.40	1.96	2.68	2.38	2.51	2.28
LOW-MODERATE	1.99	1.86	2.17	2.00	2.23	2.43	2.69	2.33
MEAN	2.08	2.08	2.24	2.16	2.44	2.46	2.76	-

Fumagalli *et al.* (1989) (**E**) found that there was a reduction in the muscle to bone ratio in their restricted animals, indicating a large reduction in the growth rate of the muscle tissue.

As with the model proposed by Berg and Butterfield (1976) the loss of liveweight results in there being an alteration in the proportions of muscle to bone. The body proportions are therefore altered to a higher proportion of bone, and a lower proportion of muscle and fat.

1.2.1.5.3 Fat

The rate of fat tissue deposition is more variable and vulnerable to nutritional fluctuations due to it being the last maturing tissue in the body (Lawrence and Pearce, 1964). The effect of a restricted diet on fat metabolism is generally a reduction in the level of fat deposition in the body ((**RI**) Verbeek, 1961; Lalande and Fahmy, 1975; (**RI**) Sully and Morgan, 1982; Berge, 1991; (**E,RI**) Sainz *et al.*, 1995).

The examination of animals raised on different planes of nutrition (Figure 12) shows that, even with a lower plane of nutrition, there is some deposition of fat. This deposition

seems to be related to liveweight, as on reaching 227 kg, regardless of plane of nutrition, there was a marked increase in the rate of fat deposition ((E) Waldman *et al.*, 1971).

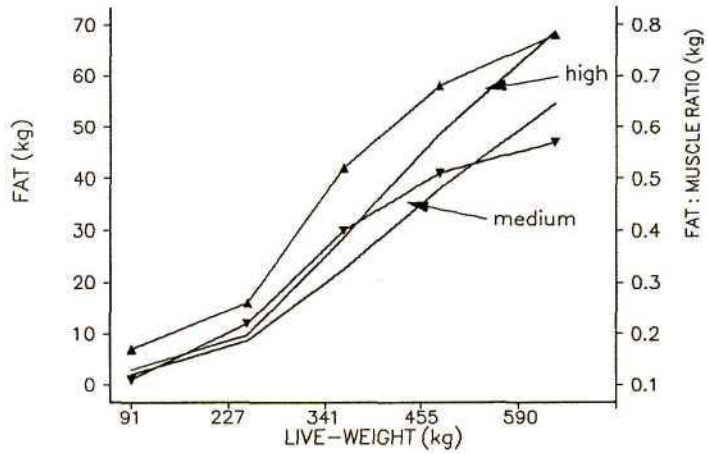

Figure 12. The effect of differing nutrient levels on fat tissue growth and the fat to muscle ratio (▲ - high nutrient level; ▼ - medium nutrient level), relative to liveweight (after Waldman *et al.*, 1971)

The deposition of fat, as predicted in the Berg and Butterfield model and as demonstrated by the literature, is largely dependant on the level of energy in the diet. Those animals on a higher plane of nutrition, with no significant differences in the bone and muscle weights, have significantly higher levels of fat as compared to animals raised on a lower plane of nutrition (Berge, 1991). Even at lower planes of nutrition, fat deposition does occur (Figure 12). Waldman *et al.* (1971) (E), found that on a lower plane of nutrition there was still a deposition of fat tissue, even though the energy used in this deposition, could have been utilised to increase the deposition of muscle (Figure 11). This indicates that there exists a ratio between muscle and bone which the animal strives to achieve once the threshold level of fat deposition has been reached.

When the restriction has been so severe as to cause weight loss, the response has generally been a marked decrease in the level of fatness within the body. The model proposed by Berg and Butterfield (1976) allows for no single loss of a tissue during weight loss but rather a combination of tissues, with a hierarchial structure as to the relative amounts of loss within each tissue. The loss of fat tissue is found to be a much

larger proportion of the existing deposited tissue than any of the other types of tissue losses. In Table 1, the growth rate of the fat under normal conditions is below that of the muscle. However, during weight loss, there is an almost equal weight loss between the two tissues, except that the loss of fat tissue accounts for 70% of the total fat tissue in the animal, whereas the muscle tissue accounts for 21% (Berg and Butterfield, 1976). The proportional loss of protein will however be found to increase as the amount of fat decreases. This was also found by Carroll *et al.* (1963) (**E,P**), whereby the animals on restriction lost 20% of their fat tissue, as compared to no change in the continually grown animals.

A comparison of animals of equal age, but different nutritional history, showed that the animals raised on a high plane of nutrition had a high level of fatness. However, when the restricted animals were compared to younger animals of equal weight, it was found that the younger animals also had a higher level of fatness ((**E,P**) Guenther *et al.*, 1965; (**RI**) Henrickson *et al.*, 1965; (**RI**) Baker *et al.*, 1985).

As proposed by Hammond (1960) there is variation in the level of fat tissue removal, due to there being different growth gradients in types of fat tissue, as well as in the area of the body where the fat is deposited. Sully and Morgan (1982) (**RI**) reported that deposition of fat due to a higher plane of nutrition was greatest in the subcutaneous fat deposits, with the loin region being very pronounced with its variation between nutrition levels. Seebeck (1973) (**E,P,RI**) established that the distribution of subcutaneous fat was unaffected by weight loss whereas intermuscular fat was. The intermuscular fat in the early maturing areas of the carcass (the fore-quarter) increased in weight whereas the intermuscular fat in the later maturing areas (the hind-quarter) decreased in weight. Restricted animals were found to have a reduced level of subcutaneous fat ((**E,RI**) Dockerty *et al.*, 1973; (**RI**) Murray *et al.*, 1974; Hogg, 1991). The level of fat deposited intermuscularly, or defined as marbling by most researchers, was found to be influenced by level of nutrition. Restricted animals had significantly lower levels of marbling ((**RI**) Henrickson *et al.*, 1965; (**RI**) Stuedemann *et al.*, 1968; (**E,RI**) Dockerty *et al.*, 1973).

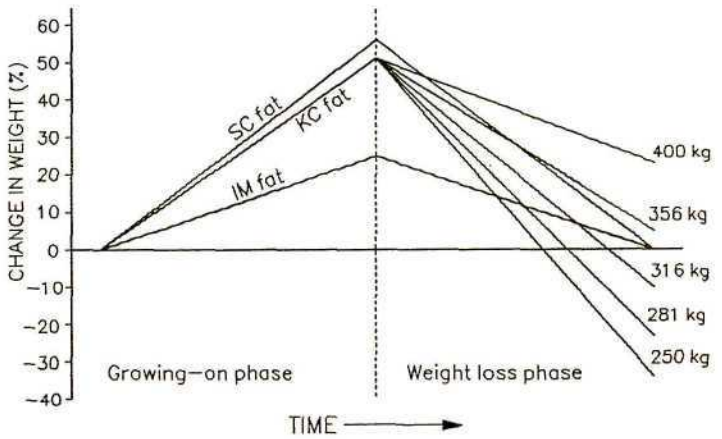

Figure 13. Percentage changes in weights of subcutaneous fat (SC), intermuscular fat (IM) and kidney and channel fat (KC) during the growing-on and weight loss phases, relative to their weight at the beginning of the growing-on phase (after Seebeck and Tulloh, 1968)

The examination of individual fat deposition sites and their individual responses to weight loss by Seebeck and Tulloh (1968) (**E,P,RI**) is shown in Figure 13. During the growing-on phase, the subcutaneous, kidney and channel fat were deposited at a greater rate than the intermuscular fat. More subcutaneous fat was deposited in the rib and loin areas while decreasing in the fore shin, topside and hind shin regions and remaining the same in other areas. Intermuscular fat followed a similar trend as that of the subcutaneous fat except that it was also decreased in the clod, blade, rump, thick flank and silverside.

During the weight loss period, no significant differences were found between the two groups in the amount of subcutaneous fat. However, more subcutaneous fat was lost from the ribs and loin, and less from the silverside. There was also no significant difference between the two groups in the amount of intermuscular fat. It was noted that there was less intermuscular fat in the loin and more in the brisket, blade and hind-shin in the larger animals. Seebeck and Tulloh (1968) (**E,P,RI**) reported that there was a differential effect of weight-loss on the weight of kidney and channel fat as compared to the

carcasses of animals that had not experienced weight loss. When the weight loss occurred at a high body weight, there was a higher proportion of kidney and channel fat in the carcass.

Due to the fat tissue being considered overall to be the latest maturing tissue, it shows the largest amount of differential tissue depletion. This is most widely demonstrated in the reversal of the growth curves during weight loss. The loss of fat tissue from specific fat depots in preference to others, shows that these sites are those that are of a higher impetus of growth at that time. The most graphic demonstration of this is illustrate in Figure 13 where Seebeck and Tulloh (1968) (**E,P,RI**) demonstrated the significantly greater loss of fat from the kidney and channel fat. These were being deposited at a greater rate than the non-significantly affected intermuscular fat. This is supported by the observations of there being a greater level of fat deposition in those fat depots that were more significantly affected during weight loss than when the animals experience compensatory growth.

The existence of growth gradients throughout the body proposed by Hammond (1960), is supported by the selective loss of fat in the body. The conclusions drawn by Seebeck and Tulloh (1968) (**E,P,RI**) show that the greater loss of fat from the rib area is an example of growth gradients as proposed by Hammond (1960) who proposed this area to be late maturing.

Depending on the age and nutrition restraint (animals at a young age and not severely inhibited), no effect on the level of fatness has been found (Hammond, 1960; Wilson and Osbourn, 1960; (**RI**) Verbeek, 1961; (**E,P**) Carroll *et al.*, 1963; (**RI**) Henrickson *et al.*, 1965; Berg and Butterfield, 1968; (**E,P,RI**) Seebeck and Tulloh, 1968, 1969; (**RI**) Butterfield *et al.*, 1971; (**E,RI**) Dockerty *et al.*, 1973; (**E**) Fumagalli *et al.*, 1989). From the review on normal growth the main emphasis on fat deposition is at an older chronological age compared to the other tissues. Therefore, if calves are restricted, the effect on fat tissues is limited due to fat not being a major deposition tissue at the time.

1.2.1.5.4 Water

Water was shown to decrease as a proportion of the carcass as an animal grows due to the actively growing tissues containing a lower proportion of water. This means that restriction of growth is not likely to affect water to the same extent as it does actively growing tissues.

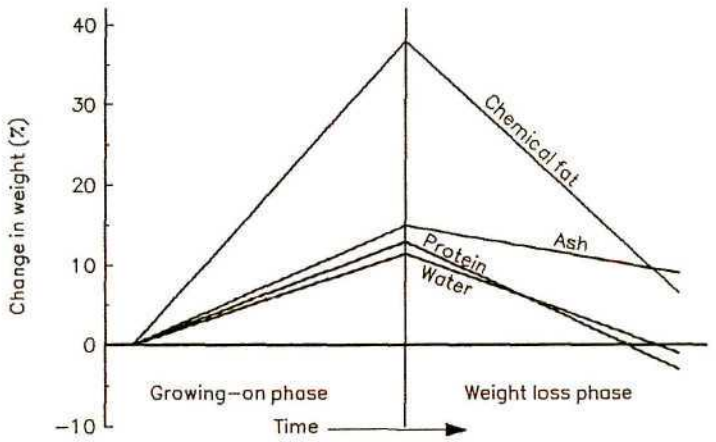

Figure 14. Percentage changes in weights of chemical components during the growing-on phase and weight loss phases, relative to their weights at the beginning of the growing-on phase (after Seebeck and Tulloh, 1968).

Significantly more water in the carcass is the result of a lower plane of nutrition ((**RI**) Verbeek, 1961; Tudor *et al.*, 1980). Dockerty *et al.* (1973) (**E,RI**) found this to occur only in the lower weights. The increase in moisture in restricted animals is due to the reduction in fat or the increasing proportion of muscle in the carcass. A comparison by O'Donovan (1984), between pasture fed cattle and intensively fed cattle, illustrates this point in that the slower growing pasture fed animals with a higher protein content and a lower fat content, had a higher moisture content.

No significant differences between treatments were found by ((**RI**) Patterson and Steen, 1995), which may be due to the restriction not being as severe as that represented in Figure 14.

The trial on which Figure 14 is based involved, "Two groups of animals were used, viz. group A which grew continuously and group B which grew like group A and were then subjected to a period of weight loss before slaughter. Corresponding animals in both groups were killed at the same body weight," ((**E,P,RI**) Seebeck and Tulloh, 1969).

When compared, the differences between groups in mean weight of water were not significant (Figure 14). This indicates that during the weight loss phase, the developmental change in the water was a near reversal of that during the growing-on phase. The rate of change of water was the same at all weights. Distributional changes of water within the carcass did however occur, generally in the same direction as changes in distribution of protein and also reflected the changes in muscle weight distribution. The weight of water was significantly lower in the loin and silverside and significantly higher in the fore shin in group B than in group A ((**E,P,RI**) Seebeck and Tulloh, 1969).

1.2.1.5.5 Protein

A protein limiting diet results in the rate of protein accretion and hence the overall protein level in the carcass being reduced. This increases with the increase in restriction. There is an increase in protein retention with increasing energy intake until protein supply becomes limiting (Ørskov, 1982). It has been established that even though energy is available to achieve equal protein deposition between animals on different planes of nutrition, those animals on a lower plane of nutrition choose to reduce protein deposition in order to deposit some fat ((**E**) Koch *et al.*, 1979).

When compared at an equal chronological age, the protein content of the carcass of an animal on a restricted plane of nutrition is significantly lower (Tudor *et al.*, 1980; (**RI**) Patterson and Steen, 1995). This can be attributed to a lower protein accretion rate at lower nutritional levels. However, Patterson and Steen (1995) (**RI**), on comparing restricted animals against animals of equal weight found there to be no significant differences. The animals used by Patterson and Steen were calves indicating that slightly older animals might have had higher protein accretion rates. The lack of extra fat deposition by the animals on the lower plane of nutrition indicates that the limiting

nutrient was energy and the animals could not consume any more feed to satisfy their nutritional requirements.

A number of researchers have however found the opposite. Carcasses of animals from a lower plane of nutrition have a higher protein content when compared at the same age ((**E,RI**) Dockerty *et al.*, 1973; O'Donovan, 1984; (**E**) Baker *et al.*, 1985). Animals kept on a lower plane of nutrition will not deposit the same amount of fat, which has a dilution effect on the protein concentration. Proof that the restricted animals were still depositing protein at a lower rate was provided by Baker *et al.* (1985) (**E**). When animals of different ages but on an equal weight basis were compared, those on a lower plane of nutrition had a lower level of protein in the carcass.

Differential mobilization of tissues during undernutrition resulted in the ratio of fat to protein mobilized being 1.7 : 1.0. The greatest losses from this high protein mobilization occurred in the metabolically active tissues such as the liver and the alimentary tract. These tissue losses are thought to enable the animal to reduce its maintenance requirement and increase its chances of survival during undernutrition ((**RI**) Ryan *et al.*, 1993).

Comparison of groups after weight loss results in significantly less protein in group B than in group A as can be seen in Figure 14. The loss of protein from specific sites was irrespective of the weight at which weight loss was imposed. This loss of protein was significantly more than would be expected by a reversal of developmental growth during the weight loss phase. This is in agreement with the extra loss of muscle found in the joint dissection. It indicates that the loss of muscle weight was not merely a result of dehydration or mobilization of intramuscular fat ((**E,P,RI**) Seebeck and Tulloh, 1969).

Protein's distributional losses corresponded with that of muscle weight's distributional losses. Protein in three of the more expensive joints (loin, topside, silverside) was significantly lower and in three of the less expensive joints (fore shin, clod, hindshin) significantly higher in group B than in group A ((**E,P,RI**) Seebeck and Tulloh, 1969). The areas of greatest removal of protein were again those of high growth impetus.

1.2.1.5.6 Chemical Fat

Daily accretion of chemical fat is found to be reduced with lower planes of nutrition. This has been established in lower planes of nutrition from *mild restriction* to *restriction* ((**E,RI**) Dockerty *et al.*, 1973; Tudor *et al.*, 1980; O'Donovan, 1984; (**RI**) Patterson and Steen, 1995). Dockerty *et al.* (1973) (**E,RI**) stressed that this reduction of fat accretion between controls and those animals kept on maintenance, occurs only at lighter weights. As the animal grows the emphasis of growth turns towards fat accretion as can be seen in normal growth. Therefore a heavier animal's growth emphasis is fat accretion. It must be stressed however that as predicted by Berg and Butterfield's model, all nutritional groups will synthesise fat, reducing protein deposition to achieve this, as found by ((**E**) Koch *et al.*,1979).

As explained by Ørskov (1982) the first limiting nutrient is important when analysing changes in fat accretion. With a protein limiting diet the fat level in the carcass increased with the increase in restriction. This is due to an increase in food intake and hence energy intake in an attempt to satisfy protein requirements.

In Figure 14, the differences between groups in mean weight of chemical fat were not significant. Thus the developmental growth of these components during the growing-on phase was approximately reversed during the weight loss phase. Chemical fat was higher in group B than in group A, but the difference was not significant. Chemical fat loss from the loin during the weight loss phase was proportionately greater in the larger animals than in the smaller ones and for chemical fat in the brisket the opposite was obtained. These changes were presumably related to similar changes that occurred in intermuscular fat ((**E,P,RI**) Seebeck and Tulloh, 1969).

The differences between groups A and B in mean weights of chemical fat within the joints were significant for the fore shin, clod, silverside and hind shin. In each case there was more chemical fat in group B than in group A. The greater level of chemical fat in the silverside corresponded with the increase in subcutaneous fat of the joint dissection. The difference in that of the hind shin was due to increase in intermuscular fat. In the

fore shin and clod, greater levels of chemical fat were probably due to differences in intramuscular fat and fat in the bones or in the composition of the dissectible fat ((**E,P,RI**) Seebeck and Tulloh, 1969).

1.2.1.5.7 Ash

Ash was found to be significantly higher on a low plane of nutrition (Tudor *et al.*, 1980; O'Donovan, 1984), when the treatments were compared at an equal weight which allows the animals on the lower plane of nutrition more time to deposit ash. This was emphasised when the daily accretion of ash was found to be not significantly different over the different planes of nutrition ((**RI**) Patterson and Steen, 1995). Ash is closely related to bone growth, and as discussed previously, has a low impetus of growth and is therefore likely to be effected the least by nutritional restriction.

From Figure 14, ash losses are small during the weight loss phase. Weight loss showing no differential effect on weight of ash at the different killing weights. Significantly more ash in group B animals than in group A indicates that ash decreases at a lower rate than carcass weight loss. On weight loss ash was lost from those areas of highest (with respect to ash) growth impetus. In the fore shin and hind shin, weight of ash was significantly greater in group B than in group A, this probably reflecting the early development of the radius-ulna and the tibia ((**E,P,RI**) Seebeck and Tulloh, 1969).

1.2.1.6 Compensatory Patterns of Growth

The previous sections have covered normal patterns of growth and the effects that differing nutritional restrictions have on these growth patterns. G1 in Figure 15, describes the classic normal growth curve, to a mature liveweight L4 after time T3. At a liveweight of L1 a period of nutritional restriction has been imposed to maintain the liveweight at L1, for a period of time T1 to T2. After time T2 the restricted animals are provided with a non-limiting diet, allowing them to express their own growth pattern. The following paragraphs will explain possible growth patterns (G2...G6) which these previously restricted animals could now follow.

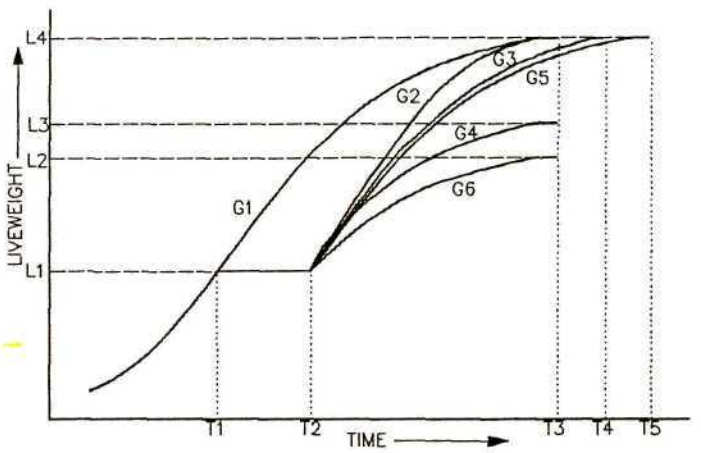

Figure 15. Growth curves (G2...G6), represent differing responses to an improved nutritional regime as compared to normal growth (G1). T1...T5 represent differing time points, and L1...L4 represent differing liveweight points.

G2 : with growth pattern G2, the liveweight gain L1 to L4 is achieved over a shorter period of time T2 to T3, as compared to T1 to T3 by G1. The two growth curves do however reach L4 at the same time T3. This growth rate is faster than that of normal growth. Complete compensation is achieved.

G3 : growth pattern G3 attains the mature liveweight L4 at time T4. Even though L4 has been reached it was not at the same time T3. The period of time T2 to T4 is shorter than that of T1 to T3, achieved by G1, therefore the growth rate of G3 was greater then that of G1. Partial compensation is achieved.

G4 : the growth pattern G4 is one in which the growth rate is faster than that achieved by G1, however growth ends at the same time T3 as in G1. The result is a lighter mature liveweight L3 for G4, however the difference in liveweight between G1 and G4 is less at T3 (L3 to L4) than it was at T2 (L1 to L2). Partial compensation is achieved.

G5 : this growth pattern is a replica of G1. The same mature liveweight is achieved after time T5. The difference in time to achieve this liveweight T3 to T5 is the same as the period of time when nutrition was limiting T1 to T2. No compensation.

G6 : this growth pattern follows that of G1 after the period T2, ending at time T3. The result is that the difference in liveweight at T2 (L1 to L2) is the same at T3 (L3 to L4). No compensation.

G2, G3 and G4 are all examples of compensatory growth. That is the growth rate of these animals is at a rate greater than that achieved by animals of an equal chronological age following a normal growth curve. Ragsdale (1934), cited by Wilson and Osbourn (1960), suggested that physiological aging continues at a slower rate during times of nutritional deprivation, due to a disturbance in the normal relationship between chronological age and physiological age by undernutrition. A compensating animal has the possibility of growing at a rate equal to that achieved at their physiological age rather than that achievable at a particular chronological age.

There has been very little published research concerning the theoretical prediction of growth following nutritional limitation. An attempt was made by Kyriazakis and Emmans (1992) to fill this void. The theory that they propose consists of the following three propositions :

1. Animal growth following nutritional deprivation is a factor of a scale of normal growth of the animals of equivalent liveweight.
2. On compensation, any abnormalities within composition will be corrected over time.
3. The conditions of rehabilitation will place constraints upon the rate of correction of the abnormalities of composition.

By the use of a description for normal growth, consideration of the limitation by its effects and the use of simple assumptions about energy and protein scales and requirements the above authors proposed their theory on growth following nutritional

limitation. The theory led to the conclusions that a comparison of the animal's state following nutritional deprivation with that of normal growth, as well as a careful consideration of the choice of and description of treatments during rehabilitation, is necessary.

1.2.1.6.1 Bone

One of two results is obtained when comparing re-alimented animals with unrestricted animals at an equal slaughter weight, where re-alimentation is the restoration of nutritional planes. First, the re-alimentated animals have a greater proportion of bone in the carcass ((**E,P**) Guenther *et al.*, 1965; (**RI**) Murray *et al.*, 1974; Morgan, 1979; Tudor *et al.*, 1980). Second, no differences in skeletal development or the bones proportion of the total carcass ((**E,P**) Carroll *et al.*, 1963; (**RI**) Henrickson *et al.*, 1965; (**RI**) Stuedemann *et al.*, 1968; (**RI**) Broadbent *et al.*, 1969; Morgan, 1972; (**RI**) Sully and Morgan, 1982; (**RI**) Yambayamba and Price, 1991). Examinations by Dockerty *et al.*, (1973) (**E,RI**), revealed that at lighter recovery weights compensating animals had significantly more bone, at intermediate weights equal amount of bone, and at final weights had more bone. At equal weights the animals exhibiting compensatory growth will be undergoing a redress of the tissue proportions of the carcass as compared to controls. Depending on the level and length of restriction, the animals will be exhibiting an increase in muscle and fat tissue growth. In trials in which the bone tissue maintains a higher proportion of the carcass the animals will still be undergoing growth to become equal to other animals at a physiological age basis.

Coleman and Evans (1986) (**RI**), found that trends for compensation in skeletal growth were apparent, though not significant. Small amounts of bone growth on re-alimentation were reported by Jones (1983). Morgan (1979), however was able to show that the late maturing bone growth areas (radii, ulnae and ribs) in compensating animals were longer.

Animals kept on a maintenance ration, and then subsequent full feeding, had skeletal characteristics of animals of a younger age than that expected from animals of such a chronological age. This suggests that following a period of undernutrition there may be

a shift in the order of priorities of tissue deposition so that muscle deposition takes priority over bone development ((**E,RI**) Dockerty *et al.*, 1973).

1.2.1.6.2 Muscle

The effects of re-alimentation upon the muscle tissue proportion within the carcass is to reduce any significant effects that did exist prior to re-alimentation to a status of non-significance when slaughtered at a constant liveweight ((**E,P**) Carroll *et al.*, 1963; Lawrence and Pearce, 1964; (**RI**) Henrickson *et al.*, 1965; (**RI**) Stuedemann *et al.*, 1968; (**RI**) Broadbent *et al.*, 1969; (**RI**) Morgan, 1972; Morgan, 1979). By examination of muscle proportion in increasing carcass weights Dockerty *et al.* (1973) (**E,RI**), revealed that at lighter recovery weights, compensating animals had significantly more muscle. This difference was however removed to one of equal muscle at final weights. When two groups of animals were compared after having recovered from differing lengths of restriction, the group with the shorter restriction had significantly more muscle than the group from the longer restriction period. Neither group did, however, differ significantly from the control ((**RI**) Yambayamba and Price, 1991).

Morgan (1979), overall muscle distribution differed significantly, with compensating animals containing a higher proportion of major muscles. This differed with Berg and Butterfield (1976), where re-alimented steers regained their normal muscle weight distribution upon recovery. The contradiction in these reports could be due to the length of time the animals had had to recover. A proposal by Lohse *et al.* (1973) (**RI**), to identify recovering muscles involves comparing selected muscle weights as a proportion of the total muscle weight. These proportions when compared to those proportions for unrestricted animals will reveal recovering muscles. The sections which cover normal and restricted growth reveal that these muscles are likely to be the high impetus muscles.

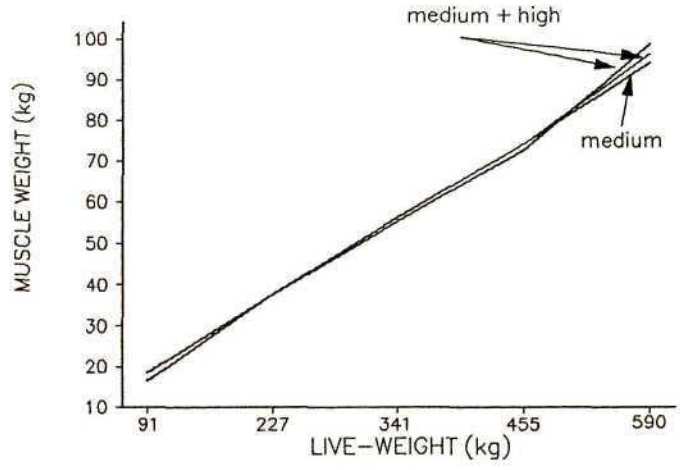

Figure 16. The result of having at least one period of muscle growth at a higher plane of nutrition as compared to a constant lower plane of nutrition in relation to liveweight (after Waldman *et al.*, 1971)

The graphical representation (Figure 16), derived from Waldaman *et al.* (1971) reveals the effect of applying a high plane of nutrition to a previously restricted group. The high plane of nutrition resulted in there being a significant increase in the amount of muscle as compared to that of the restricted animals. Berg and Butterfield (1968) suggested that the increase in muscle deposition occurs until the muscle to bone ratio is restored. In Table 1, the loss in muscle weight was nearly equal to that of fat. On re-alimentation the gain in muscle was greater than that of fat. The amount of muscle gained was greater than the amount lost. This matches Berg and Butterfield's proposal, as upon realimenation, in order to achieve the required muscle : bone ratio, the animal has to recover the muscle lost as well as that needed to compensate for the continued bone growth. The result is therefore one of a higher weight gain in muscle than that lost.

1.2.1.6.3 Fat

The re-alimentation of animals after a period of nutritional deprivation has the general effect of removing significant differences with regards to fat proportion of the carcass, between those animals that have undergone compensatory growth as compared to

unrestricted animals ((**E,P**) Carroll *et al.*, 1963; (**RI**) Henrickson *et al.*, 1965; (**RI**) Stuedemann *et al.*, 1968; (**RI**) Broadbent *et al.*, 1969; (**E,RI**) Fox *et al.*, 1972; (**RI**) Morgan, 1972; (**E**) Hironaka *et al.*, 1979; (**RI**) Sully and Morgan, 1982; Berge, 1991; (**RI**) Yambayamba and Price, 1991).

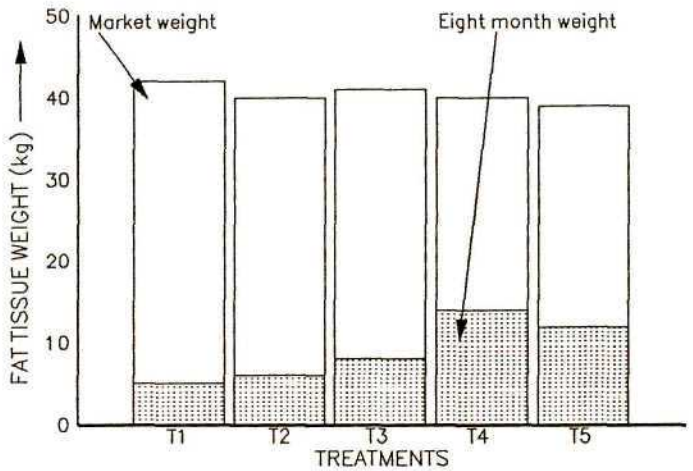

Figure 17. Effect of nutritional level imposed from birth to eight months of age and at a constant market weight after fattening in the feedlot, on the fat tissue of calves. Treatments; T1 = very restricted, T2 = restricted, T3 = normal, T4 = high, T5 = very high, (after Stuedemann *et al.*, 1968)

In Figure 17 it is shown that treatments four and five (high and very high), resulted in significantly ($P<.05$ level) higher levels of fatness as compared to the other three treatments, during the period of nutritional restriction (birth to eight months of age). Treatment three (normal) was also found to be significantly fatter at the 5% level as compared to the two restrictive treatments one and two. After a period of re-alimentation, the animals were slaughtered at a set weight, and no significant difference between treatments as to the amount of fat present in the animals was recorded ((**RI**) Stuedemann *et al.*, 1968).

Butterfield *et al.* (1971) and (**RI**)Carstens *et al.* (1991) (**RI**) showed that there was a lower level of fat deposited on re-alimentation with a high energy diet. An illustration of this is given in Table 1, where upon recovery the fat tissue did not reach the same weight as it had before restriction. However, it was noted by Berg and Butterfield (1976)

that this would have been achieved if a longer period of compensation had been allowed. Similar trends were found by other researchers for the early parts of re-alimentation. This early part of re-alimentation was followed by periods of fat growth at rates greater than controls ((**E,P**) Guenther *et al.*, 1965; (**E**) Waldman *et al.*, 1971; (**E,RI**) Fox *et al.*, 1972; (**E,RI**) Dockerty *et al.*, 1973). This was contradicted by Allden (1970) who stated that fat growth rate is much greater than any other tissues at high nutrient intakes in the period immediately after rehabilitation.

A trend of there being a greater fat deposition by animals of greater age was reported by Coleman and Evans (1986) (**RI**), Graham and Price (1982) and Jones (1983) in cull cows. In conclusion it can be stated that the fat tissue proportion increased in relation to the plane of nutrition and length of the compensation period (Berg and Butterfield, 1968; (**E,P**) Bruce *et al.*, 1991).

Within normal and restricted growth, gradient's of deposition were shown to exist between sites of fat deposition. The fat deposited during re-alimentation differed in its sites of deposition in that compensating animals were found to deposit more channel and kidney fat and lower levels of intramuscular fat compared to continuously grown animals ((**E,P**) Carroll *et al.*, 1963, Lawrence and Pearce, 1964). Morgan (1979), identified the subcutaneous fat site to be significantly greater in non-compensating animals. Stuedemann *et al.* (1968) (**RI**) and Sully and Morgan (1982) (**RI**), contradict this by finding that the treatments that they imposed, did not significantly affect the amount of subcutaneous fat as a proportion of carcass weight.

1.2.1.6.4 Water

Contradiction exists as to the effect of re-alimentation on water deposition. It has been found by some researchers that during re-alimentation the previously restricted animals deposited more water than the continuously grown animals of equal age ((**E**) Baker *et al.*, 1985; (**RI**) Carstens *et al.*, 1991). A large number of researchers have reported no significant differences between compensating and continuously grown animals ((**RI**) Henrickson *et al.*, 1965; Baker, Young and Laws, 1992; (**RI**) Ryan *et al.*, 1993; (**RI**)

Patterson and Steen, 1995). Hill (1967) (**RI**), looking specifically at the chemical composition of muscles from steers which experienced compensatory growth, found there to be no significant differences between treatments with respect to moisture. In contradiction to the other reports Tudor *et al.* (1980), found there to be significantly less water in the carcasses of re-alimented animals.

Table 3. The total weight (kg) and the proportion of each chemical component in the weight lost and gained by the restricted cattle during the period of restriction throughout re-alimentation until the end of the experiment (after Ryan *et al.* 1993)

	TOTAL LOSS / GAIN (kg)	% WATER	% PROTEIN	% FAT	% ASH
ON LOSS	10.4	65.7	10.9	18.5	4.9
ON GAIN	313	37.3	13.3	46.9	2.5

The water proportion of the weight gain (Table 3), is lower than that of the weight loss. Ryan *et al.* (1993) (**RI**) reported that the water proportion of the whole animal is non-significantly different from controls after weight recovery. An explanation for these results and contradictions is provided by Dockerty *et al.* (1973) (**E,RI**) and Wright and Russel (1991), during early recovery (lighter weights) compensating animals showed enhanced proportions in water deposition. Later in recovery (heavier weights) water deposition decreased. The final result is one in which no significant differences are found. The contradictions in literature appear to be largely due to the length of time during which the animals are allowed to compensate.

1.2.1.6.5 Protein

A comparative reduction in protein deposition is the result of a restricted plane of nutrition. This however leads to the dual possibility of a reduced protein proportion (due to increased fat deposition) or an increased protein proportion (due to a lower fat

deposition) of the carcass at the end of the restrictive period. On a weight basis the restricted animals have a lower protein weight. A large number of researchers have recorded non-significant differences in protein weights at the end of the re-alimentation period ((**RI**) Henrickson *et al.*, 1965; Baker *et al.*, 1992; (**RI**) Ryan *et al.*, 1993; (**RI**) Patterson and Steen, 1995). In order to achieve the result of there being non-significant differences in protein weights, those animals that had been subjected to a nutritional level that had affected protein deposition by reducing or reversing it, must have achieved a higher protein deposition rate ((**E,P**) Carroll *et al.*, 1963; (**RI**) Carstens *et al.*, 1991). As shown in Table 1, protein deposition must account for previous deficiencies and make up developmental changes. In the trial performed by Tudor *et al.* (1980), the re-alimented animals had less protein due to their higher levels of fat deposition. The chemical composition of muscle's from steers which experienced compensatory growth showed no significant differences between treatments with respect to total protein ((**RI**) Hill, 1967).

Dockerty *et al.* (1973) (**E,RI**), found that trends were apparent for protein weight. At lighter recovery weights compensating animals had significantly more protein, and at final weights had equal protein. The reason for these changes is that during the early periods of recovery there is enhanced protein deposition with a decrease in this deposition later in the recovery period ((**E,P,RI**) Rompala *et al.*, 1985; Wright and Russel, 1991). Coleman and Evans (1986) (**RI**) found that the upper limit to increased protein deposition occurs at a liveweight of 200 kg's, whereas Fox *et al.* (1972) (**E,RI**) established increased protein deposition rates up to a liveweight of 364 kg's, and at higher weights there was no significant difference.

On comparison of animals of equal age, but of differing growth rates, Baker *et al.* (1985) (**E**) noted that the restricted animals deposited greater quantities of protein when fed for re-alimentation. When the re-alimentated animals were compared with animals younger, but of an equal weight which had not experienced any restriction, the protein deposition rate of the restricted, older animals was seen to be higher.

Protein deposition in animals which experienced compensatory growth was at a high level initially, but, reduced over time. This protein deposition rate was higher than that achieved by animals of equal chronological age, but heavier, and higher than animals of a younger chronological age, but lighter. This resulted in any protein proportional or weight differences being eliminated by the end of the recovery period.

1.2.1.6.6 Chemical Fat

Fat deposition during re-alimentation is less in previously restricted animals as compared to controls ((**E**) Baker *et al.*, 1985; (**RI**) Carstens *et al.*, 1991). The restricted animals do deposit a greater level of fat than continuously grown animals of equal weight but younger in age ((**E**) Baker *et al.*, 1985). The reduced chemical fat deposition can result in the final carcass containing less fat (Morgan, 1979), no significant differences ((**RI**) Henrickson *et al.*, 1965; Baker *et al.*, 1992; (**RI**) Ryan *et al.*, 1993; (**RI**) Patterson and Steen, 1995), or finally more chemical fat (Tudor *et al.*, 1980). The differences with these results are due to the length of time the animals are allowed to re-aliment. For example, the set slaughter weights may not have been high enough to allow the animal to regain it's muscle to bone ratio and the following increased chemical fat deposition. The chemical fat weight gain, as a proportion, during re-alimentation is at a higher level than that of the chemical fat weight loss during the restrictive period (Table 1).

Serial slaughtering revealed that at lighter recovery weights compensating animals had significantly less fat, and as weights increased to final slaughter weights, equal fat resulted ((**E,RI**) Dockerty *et al.*, 1973). The chemical fat deposition is lower in compensating animals at lighter weights increasing later in recovery to become non-significantly different at heavier weights ((**E,P,RI**) Rompala *et al.*, 1985; Wright and Russel, 1991). If a long enough recovery period is allowed for, the final result shows no significant difference.

Hill (1967) (**RI**), examination of chemical composition of muscles from steers which experienced compensatory growth found no significant differences between treatments with respect to intramuscular fat.

1.2.1.6.7 Ash

Ash deposition in steers exhibiting compensatory growth is significantly higher ((**RI**) Carstens *et al.*, 1991). At final carcass weights ash proportion is found to be non-significantly different ((**RI**) Henrickson *et al.*, 1965; Baker *et al.*, 1992; (**RI**) Ryan *et al.*, 1993; (**RI**) Patterson and Steen, 1995). As the ash proportion is higher in most trials at the end of the restrictive period and from Table 1, which shows ash making up a lower proportion of the weight gain not much change is expected in ash deposition during re-alimentation. Due to the high levels of chemical fat and it's dilution effect on the proportions of the other chemical constituents less ash was found in the re-alimented animals (Tudor *et al.*, 1980). Hill (1967) (**RI**), no significant differences between treatments with respect to ash on examination of chemical composition of muscles from steers which experienced compensatory growth.

The results revealed in the literature covered in this seminar support this theory proposed by Kyriazakis and Emmans (1992). The deposition of tissue, particularly protein and fat is of a rate higher than of a comparative chronological age and is closer to that of a similar physiological age. It has also been found that given an adequate period of time for compensation the restricted animals can recover to a point where no significant differences within composition occur.

1.2.1.7 Patterns of Growth as Affected by Maturity and/or Breed Type

Examination of factors that affect the carcass and it's constituents would not be complete without investigation into possible maturity and/or breed type differences. A fierce debate has raged over the existence of improved carcass traits (that is more lean with less bone and fat) between breeds . The differing points of comparison have led to difficulties in understanding. These have varied from a set age to a set weight and from a chronological age to an assumed physiological age. The following section will attempt to highlight similarities or differences that researchers have been able to gather on the afore mentioned topic.

There should be no important breed differences with respect to the physiological systems that contribute to growth. Breed's with different growth rates and mature size's are simply exhibiting differences in the rate of function of these physiological systems (Paterson, 1981). The logical assumption drawn from this by Paterson was that if these physiological systems are similar in all breeds of cattle then there will be similarity in their carcass structure as well, and any differences will mainly be due to their mature size.

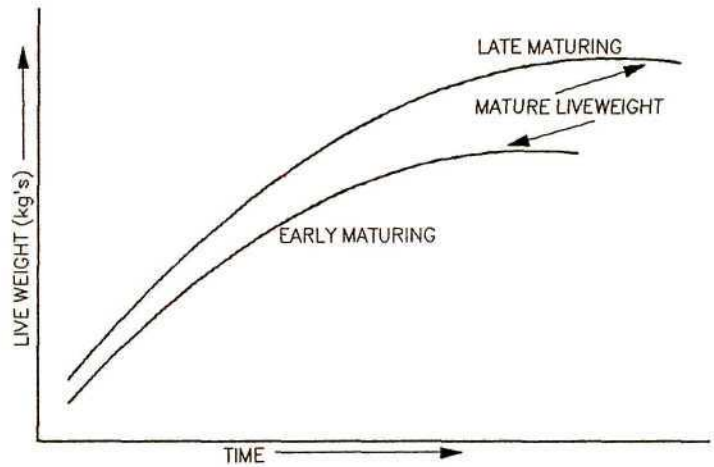

Figure 18. Differences in the growth rates between early maturing and late maturing animals

In Figure 18, the late maturing animal is shown to have a higher growth rate, a heavier mature mass and takes longer to achieve this mature mass (Smith, 1979). Shown in this description is that the maturity type in cattle is closely related to size, with larger animals being generally later maturing (Butterfield, 1966). An illustration of growth by differing maturity type's follows. The Friesian is considered late maturing as it is potentially a large animal. Its bone and muscle has been found to be capable of absorbing large amounts of concentrated nutrition over that portion of the life span during which it is growing to market weight. Despite *ad lib* food intake and minimum environmental stress only sufficient fat is deposited in the fat depots to ensure a desirable amount of finish by the time slaughter weight is reached. The Angus is an example of the early maturing type of animal. As an early maturing animal it is less capable of absorbing large amounts of concentrated nutrition while being fed to slaughter weights. Thus to avoid over fatness

it must gain weight more steadily. The early maturing animal has the advantage however that it can be rapidly fattened at almost any weight by an increase in nutritional intake (Butterfield, 1966).

A general conclusion that can be drawn from these growth patterns is that late maturing animals will have a heavier carcass than early maturing ones (Hedrick, 1972; Berg and Butterfield, 1976; Ferrell *et al.*, 1978; Coleman and Evans, 1986). The differences between breeds in size is due to differences in the size of the skeleton and in the number but not the size of muscle cells (Owens *et al.*, 1993). With carcass weight differences at any point in time, explanations on developmental changes between the maturity types are necessary. The differing rates of function of the physiological systems described by Paterson (1981), result in the developmental changes taking place in much shorter chronological time and are extended further in an early maturing animal than in a late maturing one (Pálsson, 1955).

Section 1.2.1.4 covers normal patterns of growth. Underlined in the section is the maturity gradients of the different tissues. That is, bone being earlier maturing than muscle and fat being later maturing than both these tissues. Gradients of maturity with through the body was also covered. These maturing patterns for each tissue and individual structure should be the same irrespective of animal size. This is essentially what occurs, very few structures and tissues differ in their maturation patterns because of size (Butterfield, 1988). Most results comparing maturity differences will simply indicate the animals position on the growth curve, with early maturing animals being physiologically more mature at a set chronological age during growth than a late maturing animal. Being more mature, early maturing animals have a higher proportion of muscle in the carcass at similar carcass weights (unless excessively fat) than late maturing dairy and beef breeds. Consequently a higher muscle to bone ratio also exists (Paterson, 1981). Both maturity types follow the general developmental trend of an increase in muscle to bone ratio as mass increases. At heavier masses that are approaching mature mass this rate of increase slows down (Paterson, 1981). Being physiologically more mature, the early maturing animal, will enter the fattening phase

at a lower weight and have a higher proportion of fat at similar weights (Hedrick, 1972; Berg and Butterfield, 1976).

The variation in growth of bone, muscle and fat between animals, particularly if determined on the basis of chronological age, as described above is less evident if determined on the basis of physiological maturity (Hedrick, 1972). With these maturity differences the following quote from Butterfield (1988) is therefore very relevant, "it is, therefore, likely that any comparisons made at equal age, equal weight, or equal anything, other than degree of maturity, will give comparisons which have little bearing on the genetic body composition of the animals being compared".

The literature covering the topic is summarised by Baker *et al.* (1992). In his findings significant differences in the relative growth coefficients between the breed types were detected only for empty body weight. The relative growth of carcass protein and carcass fat to carcass weight are found to be similar across breed types. Work by Butterfield (1988) on growth between breeds of sheep shows that the differences in structure and in composition of mature sheep which can be directly related to size appear to be few (Table 4). Examination at equal percentages of maturity shows there to be little if any differences in the proportions of carcass tissues relative to liveweight, in muscle weight distribution, or in fat partitioning.

Table 4, Progress to maturity of carcass tissues of large and small Merino rams relative to progress to maturity of shorn full liveweight (after Butterfield, 1988).

	PERCENTAGE MATURITY									MATURE WEIGHT (kg)	
										LARGE	SMALL
LIVE - WEIGHT	20	30	40	50	60	70	80	90	100	= 116.5	90.9
MUSCLE	24	35	46	56	66	75	84	92	100	= 25.9	20.7
BONE	26	39	50	60	70	79	86	94	100	= 6.4	4.9
FAT	5	11	18	27	38	51	65	82	100	= 26.7	18.8

Comparison between maturity types without adjusting to a common physiological age finds large late maturing breeds, are leaner at constant age or at weight end points than, are small breeds (Bond *et al.*, 1972; Smith, 1979; Lowman *et al*, 1994). This was quantified by Wheeler *et al.* (1989) who showed that carcasses from late maturing cattle were 40 to 50 per cent leaner than the carcasses from early maturing animals. The daily lean gain as predicted from the comparative growth curves was lowest in the early maturing breed and highest in the later maturing breeds (Korver *et al.*, 1987). A higher daily lean gain by the late maturing animals will explain their higher lean content at a constant age. Comparisons at a constant weight basis will emphasise the difference in the maturity of the carcasses, a carcass from an early maturing animal will contain more fat diluting it's lean content. This could explain how a late maturing animal's carcass can contain more lean at a set weight. In contradiction, the growth coefficient for muscle was found to be similar between breeds by Mukhoty and Berg (1971). However comparisons at a common muscle to bone ratio resulted in significant breed differences. As the coefficient for growth was the same, these differences appear not to be caused by differences in relative growth over the growth period represented, but to have already been established at an earlier stage of growth (Mukhoty and Berg, 1971).

The belief held by some people that a maturity type has a superior expensive muscle cut to another, leads to comparisons of individual muscle's and muscle distribution between maturity types. A muscle commonly measured is the eye muscle or *longissimus*. As with total lean content the weight and area of the eye muscle increased with maturity type (Bond *et al.*, 1972; Coleman and Evans, 1986). When the eye muscle area was expressed as a proportion per kilogram of carcass weight, the early maturing animals were found to have a higher proportion.

Overall, only minor breed differences are found in weight distribution among muscles, groups of muscles and wholesale cuts (Charles and Johnson, 1976a). To achieve, this the growth coefficient for the muscle groups relative to the total muscle side must be similar (Shahin *et al.*, 1993). The differences between early maturing compared to late maturing breed types which have been isolated are as follows. Earlier maturing animals have a greater proportion of late maturing muscles (for example the abdominal and neck muscles) and the later maturing animals having a greater proportion of early maturing muscles (Mukhoty and Berg, 1973; Shahin *et al.*, 1993). These minor differences may be due to very small true breed differences, or it may simply reflect allometric growth differences within the musculature brought about by the stage of growth reached by two breeds of cattle of differing maturity type (Charles and Johnson, 1976a). It can be concluded that similarity of muscle weight distribution in the different types of carcasses studied shows that carcass shape is not associated with differences in the distribution of muscle weight in wholesale cuts.

Comparisons between maturity types on physiological age basis should remove all differences except those of a true genetic nature. A number of researchers have attempted this. The problem lies however with their method of determining physiological age. LeVan *et al.* (1979) and Barber *et al.* (1981) compared late and early maturing steers slaughtered at equal percentages of mature cow weight. The use of this technique is complicated by possible genetic improvements (selection or heterosis), different rearing techniques between the cow herds of the maturity types, large variation in weight within cow herds (due to condition or maturity type). With the use of this technique, LeVan *et al.* (1979) and Barber *et al.* (1981) found larger *longissimus* muscle areas for the late

maturing animals, but the muscle area increased for both maturity types as carcass weights increased. Discrepancies in the results indicate that this method did not fully compensate animals to an equal physiological age. For example higher fore-quarter and lower hind-quarter percentages were observed in the early maturing breed at all but the heavy weight class (Barber *et al.*, 1981). The conclusion reached was that this was due to advanced fattening of the early maturing breed. Considering that the animals should be at equal fattening rates due to their equal physiological ages, either the animals were not of an equal physiological age, or some other factor limited fat deposition in the late maturing breed. Distributional anomalies with early maturing animals having a larger amount of late maturing tissues and vice versa is removed when compared at an equal physiological age. LeVan *et al.* (1979) found the percentage of retail lean to be the same between maturity types at assigned slaughter weights with there being no breed effect on retail lean distribution.

An alternative technique used to compare animals at an equal physiological age is that used by Jones *et al.* (1984). Animals are compared at the same proportion of dissected carcass subcutaneous fat. This technique is complicated with the following problems. As will be shown, breeds differ in their fat deposition areas, some breeds have been found to deposit a lower proportion of fat in the subcutaneous fat sites. A trial starting at a set weight or age will have the early maturing animals further along the growth curve and so their fat deposition will have had been affected to a greater extent by the pretrial factors. Potentially the most difficult point is that even though the fat depths are the same, the fat weight of the subcutaneous depot is not of the same proportion of the carcass. If not, then animals are unevenly matched as one has deposited more energy than the other as a percentage of its total carcass weight. Carcasses from large crossbred's were found to have a greater proportion of muscle, with generally no differences in the distribution of muscle within each of the wholesale cuts (Jones *et al.*, 1984). Even though distributional differences are removed with the use of this technique, the presence of a greater proportion of muscle indicated that the late maturing animals could be physiologically younger. As will be shown further evidence is found on examining the results for bone distribution.

The proportion and weight of bone in the carcass of a late maturing animal is expected to be higher, as the carcass will be less mature than an early maturing animals carcass. This is confirmed by Bond *et al.*, 1972, Butterfield, 1988 and Wheeler *et al.*, 1989 who found that, the growth coefficients for bone and muscle were similar between breeds. However comparisons at a common muscle to bone ratio resulted in significant breed differences. As the coefficient for growth was the same, these differences appear not to be caused by differences in relative growth over the growth period represented, but already established at an earlier stage of growth (Mukhoty and Berg, 1971). Wheeler *et al.* (1989) discovered that there were differences in the proportional changes of bone between maturity types. Carcasses from early maturing animals decreased in bone percentage from 0 to 128 days and then levelled off. However, late maturing cattle decreased from 0 to 77 days then stabilised. The decrease in bone percentage is due to the higher proportional increase in muscle. The late maturing animals are expected to have a greater length of time during which the bone percentage decreases due the higher proportional muscle weight to be deposited by the animals. Possible explanations for this discrepancy are that the late maturing animals deposited the required amount of muscle at faster than expected rate or that the late maturing animals have a greater proportion of bone in the carcass. Butterfield (1988) provides support for late maturing animals having more bone by reporting that the major limb bones are proportionately heavier.

On an equal physiological age basis, LeVan *et al.* (1979), found the total bone percentage from the four major wholesale cuts was greater for Charolais (late maturing breed) steers at all weights. This work supports that of Butterfield (1988) as it was also performed on a physiological age basis. The work by Jones *et al.* (1984) contradicts this by showing that the carcasses from large crossbred's have less bone than small crossbred's. Bone distribution, however, did vary significantly across several wholesale cuts with carcasses from large animals having lower proportions of bone (in the hip, loin, rib, chuck and shank) than those from small animals. These areas of bone development are late maturing, indicating that with this technique the late maturing animals were physiologically younger.

Korver *et al.* (1987), concluded however that regardless of the method used breed differences in carcass composition still existed after adjusting for degree of maturity. This may however be due to breed rather than maturity differences. As with Berg and Butterfield's (1976), statement that breeds differ in muscle measured as total muscle weight relative to bone weight. This is due to breeds that were selected for body thickness or for draft usage generally exceed those selected for dairy character in muscle:bone ratio. The superior muscling that occurs does so early in the post-natal period and breeds with high muscle:bone ratio remain superior throughout life, barring periods of weight loss. However breed plays a relatively minor role in relative growth within the musculature and therefore in muscle weight distribution. Extensive studies have failed to reveal any differences of muscle weight distribution of sufficient magnitude to be of commercial importance (Berg and Butterfield, 1976).

The level of fatness is a controlling force in the decision as to when an animal is ready for slaughter. Fat being considered the tissue that dilutes the proportion of the other tissues making up the carcass, comparison of tissue proportions rely heavily on the comparative level of fatness between animals. Breeds which are slaughtered at a low level of fatness will appear superior in yields of high-priced cuts. However when comparisons of yields of high-priced cuts are made at equal levels of fatness it is unlikely that any breed will show an advantage (Berg and Butterfield, 1976).

The level of fat in a carcass is due to a balance between maturity type and the level of nutritional intake (Butterfield, 1966). An early maturing animal being further along the growth curve at a set age than a late maturing one would have a higher fat deposition rate irrespective of nutritional plane. In general the use of animals of different maturity types within experiments illustrates that early maturing animals have a higher level of fat content at set weights, independent of the plane of nutrition (Bond *et al.*, 1972; Lalande and Fahmy, 1975; Barber *et al.*, 1981; Coleman and Evans, 1986; Coleman *et al.*, 1993; Lowman *et al.*, 1994). Mukhoty and Berg (1971) and Coleman *et al.* (1993) demonstrated that the growth coefficients for fat differed significantly between breed groups. Having different growth coefficients for fat it is likely that this is due to the breeds differences in time of onset of the fattening phase.

The work by Wheeler *et al.* (1989) shows the maturity types differing response to fattening. Percentage of total carcass fat and fat thickness increased linearly across days on feed for the carcasses from early maturing cattle. However, on the carcasses from late maturing cattle, percentage of total carcass fat increased slightly up to 77 days and remained relatively constant thereafter. The linear increase in fat deposition by the early maturing animals follows that described in normal growth patterns. The late maturing animals increase up to 77 days illustrates the increase in feed intake and the subsequent extra energy available for fat deposition over that of muscle growth. The fat deposition does not increase further as the animal is still following a high muscle growth phase in order to achieve the same muscle to bone ratio as that in the early maturing animals. An increase in metabolisable energy affected the early maturing animals. Their fat deposition rate increased whereas that of the late maturing animals did not. Barber *et al.* (1981) suggested that this implied a breed x diet interaction. If the late maturing animals were consuming as much feed per day as they were at the low energy level then the extra energy must be used for something. This could be for muscle deposition or for fat deposition in areas not accounted for by Barber *et al.* (1981) for example intramuscular fat.

Differences in fat deposition and partitioning between breeds is complicated by maturity type as well. Distribution particularly of subcutaneous fat, seems to be associated with the shape of animals, that is traditional beef breeds having a higher proportion. Along this vein it has been found that dairy breeds (Holsteins) have a greater proportion of their total fat in the visceral site as compared to beef breeds (Jones *et al.*, 1985). The differences seen in the partitioning between depots may be eliminated by comparing in terms of the total amount of fat present relative to the mature content of the breed. In this case larger breeds at the same weight of subcutaneous fat, have more intermuscular fat, however at the same proportion of their final weight they may not be different (Berg and Butterfield, 1976).

An extensive trial conducted by Charles and Johnson (1976b) to determine breed differences in amount and distribution of bovine carcass dissectible fat, is confounded by maturity type differences. Some breed differences do seem to be revealed though.

Herefords were found to have deposited significantly more subcutaneous fat and significantly less kidney plus pelvic fat than the other breeds. A general trend however for all the breeds was a constant change in subcutaneous fat depth as total dissected fat changed. With an increasing total dissected fat the subcutaneous fat increased in proportion, and the kidney plus pelvic fat decreased. However in the Charolais x carcasses both depots increased, to make up for this the depot which did decrease in the Charolais as compared to the other breeds was that of intermuscular fat.

Comparison on an equal physiological age by Jones *et al.* (1984), carcasses from large crossbred's had similar proportions of total fat to small crossbred's. The work by LeVan *et al.* (1979) however showed that the Angus and Charolais were not different in kidney fat percentage or fat thickness except that middle weight Angus had more 12th-rib fat thickness than their Charolais counterparts. Charolais steers had consistently less fat thickness per 100 kg of carcass than Angus. Total fat percentage was greater for Angus. Korver *et al.* (1987) concluded that, regardless of the method used, breed group differences in carcass composition still existed after adjusting for degree of maturity.

This conclusion seems to be justified in the context of fat distributional differences, but the argument cannot be settled until the animals are compared at a truly equal physiological age. From the literature covered an important gap that needs to be filled is the comparison between maturity types and compensating versus non-compensating on an equal physiological age basis to determine whether the basic growth of the three main tissues are the same. Once this question is answered then examination of possible distributional differences is necessary. Overall these answers will remove senseless debate allowing producers and researchers to concentrate on fitting of maturity types to their best production possibilities within the markets constraints of preference for a 200 kg carcass. It is expected that the trial to be described will go some way to removing some of the ambiguity that surrounds this topic.

CHAPTER TWO
PRE-FEEDLOT TREATMENT

2.1 INTRODUCTION

The effect of nutrition on carcass composition (Chapter One), is particularly relevant in the South African context because grazing animals are subject to recurring nutritional depressions. The latter results from cold dry winters and periodic droughts (Joubert, 1954) which has led to retarded growth and losses in body weight. Winchester *et al.* (1957) reported on the retardation in growth experienced by a large number of range cattle which usually occurs during the autumn and winter seasons, and in most cases gains were completely interrupted or the animals were subjected to an actual loss of weight. The concern raised was that this retardation may have resulted in a later loss of at least part of the potential for the production of high quality beef (Winchester *et al.*, 1957).

The market requirement has been for a carcass of roughly 200 kg. To achieve this a growing phase for cattle is usually imposed for a period between weaning and finishing in a feedlot. During the growing phase, body development is allowed to continue before fattening to a slaughter finish at the desired carcass weight. Nutritional stress is often placed on the cattle during the growing phase, thus allowing for the cattle to exhibit compensatory growth when placed on full feed (Sainz *et al.*, 1995). Two of the most important components of net efficiency, in a beef production system, is that of post-weaning growth and feed efficiency (Smith *et al.*, 1976). Wilson and Osbourn (1960) concluded that there is no difference in efficiency (in terms of weight gain and feed conversion) between a restricted and a re-alimentated animal than a continuously grown animal. This conclusion is valid on conclusion that the animal does not lose weight and is allowed to express increased appetite during re-alimentation by *ad libitum* feeding. This allows for the utilization of cheaper winter foods due to the compensating animal making more efficient use of the more expensive finishing rations subsequently.

The characterisation of cattle breeds into different maturity types and physiological ages is an important consideration when determining their potential postweaning performance. This is of particular relevance in feedlot feeding, where those animals that are likely to exhibit compensatory growth are expected to achieve improved feed conversion efficiencies (FCE), average daily gains (ADG) and reduced cumulative feed intake, as compared to animals of a similar chronological age but of a later physiological age. This will, however, be offset by the animals remaining in the feedlot for a greater length of time to reach a given end-point (e.g. a perceived bodyfat of twenty per cent). Therefore, investigation of the advantages shown by animals exhibiting compensatory growth, over those following a normal growth pattern, as well as the comparison between animals of differing maturity types due to their different physiological ages at a given chronological age will be beneficial. This is so as to determine the type of animal which is likely to achieve the best performance in a given situation.

Animals that have the potential to exhibit compensatory growth will perform more efficiently than corresponding animals of equal chronological age, but of a more mature physiological age. The combination of compensatory growth and early maturity should theoretically, provide the best class of animal for feedlot feeding (providing a carcass of roughly 200 kg).

2.2 MATERIALS AND METHODS

2.2.1 EXPERIMENTAL DESIGN

To answer the question on growth with respect to maturity type the experiment used two extreme maturity types *viz* late and early maturing. Comparison of the potential for compensatory growth was achieved, by splitting each maturity type into two pre-feedlot planes of nutrition (Figure 19).

Figure 19. Diagrammatic representation of the experimental design.

EARLY MATURING (n = 48)	FAT (n = 24)	REP 1 (n = 12)
		REP 2 (n = 12)
	THIN (n = 24)	REP 1 (n = 12)
		REP 2 (n = 12)
LATE MATURING (n = 48)	FAT (n = 24)	REP 1 (n = 12)
		REP 2 (n = 12)
	THIN (n = 24)	REP 1 (n = 12)
		REP 2 (n = 12)

FAT : those animals gaining weight during the pre-feedlot period (0.5 Kg/day).
THIN : those animals maintaining weight during the pre-feedlot period (0.0 Kg/day).

This resulted in four treatments :

Early maturing fat (EF); Late maturing fat (LF); Early maturing thin (ET); Late maturing thin (LT).

2.2.2 RESEARCH ANIMALS

All the animals were purchased from a country auction at the same sale. This has the advantage of reducing any effect of pre-weaning treatment and age as all the animals obtained came from the same farming system. Although most animals were crossbreeds the early maturing animals were dominated by the Hereford, Sussex and Angus breeds, with the late maturing animals being made up of the Simmentaler and Charolais breeds.

It was attempted to purchase animals differing in body condition. This meant that in each case there were animals that were thin or fat within each maturity group. On arrival at the farm, the animals were conditioned scored (2.2.4.2), with the thin animals (ET and LT) being placed in a separate group to the fat animals (EF and LF).

2.2.3 PRE-FEEDLOT PLANE OF NUTRITION

Two pre-feedlot treatments were imposed on the animals. The first treatment was that of an average daily gain of approximately 0.5 kg per day, with the other treatment being that of an average daily gain of 0.0 kg per day i.e. maintenance of weight. The treatment period was intended to last for about 100 days.

Those animals intended for weight gain during the pre-feedlot period were placed on kikuyu (*Pennisetum clandestinum*) pasture. These steers always has first access to the pasture. As conditions during winter deteriorated and the quality of the pasture dropped, a supplement was necessary to ensure the required weight gains.

Those animals destined to maintain their weight over the pre-feedlot period (*restriction*) were given access to the same pastures as the first group but only after the pasture had been grazed severely. A supplement was also found to be necessary towards the end of winter to prevent weight loss.

2.2.4 EXPERIMENTAL MEASUREMENTS

2.2.4.1 Liveweight

Animals were weighed individually during the winter period at twenty one day intervals, to ensure that target weight gains were achieved. The animals were weighed on a full body weight basis, that is no restriction in terms of water or feed was imposed before each weighing.

2.2.4.2 Condition

Animals were condition scored using a combination of visual evaluation and physical touch. The animals were rated according to a scale where a 1 was considered emaciated and a 5 over fat. The condition scoring was performed on all animals at the time of weighing.

2.2.5 STATISTICAL METHODS

The GENSTAT V statistical programme (Lawes Agricultural Trust, Rothamstead) was used for all statistical analyses. Regression models were fitted to the data, where relevant. A selection of models was used; linear, quadratic, cubic. The model that provided the best statistical fit was chosen. Before a model was chosen, however, its use had to be justified, that is, its parameters must meet with logical understanding, in line with existing principles in Animal Science. Significant differences between means were determined from analysis of variance tables with the use of the *Students' t test* (Steel and Torrie, 1980).

Canonical variate analysis (CVA) was performed on the liveweight and condition of the four treatments over time. This method allows for the separation of two or more groups of individuals given measurements for these individuals on several variables (Manly, 1991).

2.3 PRE-FEEDLOT RESULTS

The canonical variate analysis for liveweight (Table 5), and condition (Table 6), show that the animals were correctly allocated to their status treatments, that is, all the animals within the fat or thin group's performed consistently, as expected. Even though some animals were switched between maturity types, no significant differences were found between the growth rates of maturity types within a status group (2.3.1 and 2.3.2).

Table 5. Canonical variate analysis of liveweight over the pre-feedlot period

TREAT[a]	NEW TREATMENT				
	EF	LF	ET	LT	COUNT
EF	21	3	0	0	24
LF	3	21	0	0	24
ET	0	0	17	7	24
LT	0	0	7	17	24
COUNT	24	24	24	24	96

[a] = Treatment group (2.2.1)

Table 6. Canonical variate analysis of condition over the pre-feedlot period

TREAT[a]	NEW TREATMENT				
	EF	LF	ET	LT	COUNT
EF	13	11	0	0	24
LF	6	18	0	0	24
ET	0	0	12	12	24
LT	0	0	9	15	24
COUNT	19	29	21	27	96

[a] = Treatment group (2.2.1)

The pre-feedlot period extended for 103 days, with the following growth responses.

2.3.1 LIVEWEIGHT

Table 7. Analysis of variance results for pre-feedlot periodic liveweights changes

Source of Variation	d.f.	s.s.	m.s.	v.r.	F pr.
Type[a]	1	70131.7	70131.7	231.66	<.001
Status[b]	1	406352	406352	1342.3	<.001
Time	4	72929.7	18232.4	60.23	<.001
Lin[c]	1	53774.4	53774.4	177.63	<.001
Quad[d]	1	16925.6	16925.6	55.91	<.001
Cub[e]	1	1915.8	1915.8	6.33	0.012
Deviations	1	313.9	313.9	1.04	0.309
Type.Status	1	2367.4	2367.4	7.82	0.005
Type.Time	4	439.8	109.9	0.36	0.835
Type.Lin	1	237.1	237.1	0.78	0.377
Deviations	1	0.4	0.4	0.00	0.969
Status.Time	4	12428.7	3107.2	10.26	<.001
Status.Lin	1	8279.7	8279.7	27.35	<.001
Status.Quad	1	4120.6	4120.6	13.61	<.001
Deviations	1	28.4	28.4	0.09	0.760
Type.Status.Time	4	108.4	27.1	0.09	0.986
Type.Status.Lin	1	2.6	2.6	0.01	0.926
Deviations	2	28.6	14.3	0.05	0.954
Residual	460	139257	302.7		
Total	479	704015			

[a] : Maturity type (early or late), (2.2.2)
[b] : Pre-feedlot treatment (fat or thin), (2.2.3)
[c] : Linear model
[d] : Quadratic model
[e] : Cubic model

All three variables (Table 7), were found to be significant components of the model (Type, Status and Time). The interaction of Status and Time (i.e. the change in status over time) produced a significant ($P < 0.001$) quadratic response. With the nutritional treatments applied (2.2.3) this was expected to be linear. The quadratic response was due to a large increase in liveweight between the first and second measurement. The animals were weighed on a starved body weight basis at the first measurement, and a full body weight basis from then on. Only after the first measurement did the animals in their respective statuses follow their pre-determined growth pattern (2.2.3). The interaction of Type and Time (i.e. the change in type over time) showed a non-significant response. Those animals that were early maturing remained early maturing and those that were late maturing remained late maturing, in other words the differences between maturity types remained constant. The interaction of Type, Status and Time (i.e. the change in status of each type over time) was non-significant. Each treatment followed similar growth patterns, in that the fat groups (EF vs LF), and the thin groups (ET vs LT), were not significantly different.

The liveweight's of the animals exposed to the different treatment's were significantly different at the beginning and at the end of the pre-feedlot period (Table 8). The animals were significantly different in liveweight at the beginning of the pre-feedlot period because they were allocated to their treatments according to their condition (2.2.4.2), on arrival at the farm. The change in liveweight over the 103 days of the pre-feedlot period were significantly different between treatments (fat vs thin), but non-significantly different within treatments (EF vs LF and ET vs LT).

Table 8. Liveweights and average daily gains of steers during the pre-feedlot period

ITEM	TREATMENT			
	EF	LF	ET	LT
Initial Weight (kg)	219.3[b]	243.6[a]	184.3[d]	200.7[c]
Final Weight (kg)	256.3[b]	286.0[a]	196.5[d]	216.3[c]
ADG (kg/day)	0.360[a]	0.412[a]	0.100[b]	0.152[b]

[a,b,c] Values in the same row with different superscripts are different $P < .05$.

2.3.2 CONDITION

Table 9. Analysis of variance results for pre-feedlot condition changes, against type, status and time

Source of Variation	d.f.	s.s.	m.s.	v.r.	F pr.
Type[a]	1	0.40833	0.40833	6.55	0.011
Status[b]	1	57.4083	57.4083	921.20	<.001
Time	4	224.226	56.0565	899.51	<.001
Lin[c]	1	204.465	204.465	3280.9	<.001
Quad[d]	1	5.16999	5.16999	82.96	<.001
Cub[e]	1	13.6683	13.6683	219.33	<.001
Deviations	1	0.92253	0.92253	14.80	<.001
Type.Status	1	0.35208	0.35208	5.65	0.018
Type.Time	4	0.28437	0.07109	1.14	0.337
Type.Lin	1	0.14323	0.14323	2.30	0.130
Deviations	1	0.10797	0.10797	1.73	0.189
Status.Time	4	5.24271	1.31068	21.03	<.001
Status.Lin	1	1.31093	1.31093	21.04	<.001
Status.Quad	1	0.24611	0.24611	3.95	0.047
Deviations	1	1.46985	1.46985	23.59	<.001
Type.Status.Time	4	0.05937	0.01484	0.24	0.917
Type.Status.Lin	1	0.01979	0.01979	0.32	0.573
Deviations	2	0.03951	0.01976	0.32	0.728
Residual	460	28.6667	0.06232		
Total	479	316.648			

[a] : Maturity type (early or late), (2.2.2)
[b] : Pre-feedlot treatment (fat or thin), (2.2.3)
[c] : Linear model
[d] : Quadratic model
[e] : Cubic model

As with liveweight, Type, Status and Time are significant components of the model (Table 9). The aim of the pre-feedlot period was to split the animals into two groups, according to their growth during this period. The nutritional treatments imposed (2.2.3) were also intended to affect the condition of the animals. The interaction of Status and Time (i.e. the change in condition over time) showed a significant ($P < 0.001$) quadratic response. The condition of the animals decreased over time until the last measurement, before the feedlot period, when it increased. This increase was due to the condition scoring being performed by two scorer's rather than the one used prior to this time. The result was a consistently higher condition score for all animals regardless of treatment, as that given at the previous measurement. The interaction of Type and Time (i.e. change in type over time) showed a non-significant response. This was expected as no change in type over time should occur. An important consideration was that the treatments for each type of animal be non-significantly different. This was shown with the interaction of Type, Status and Time or the change in status for each type over time being non-significant.

The condition score between the animal status's (fat vs thin) were significantly different at the beginning of the pre-feedlot period. The animals within a maturity type were placed on treatments, based on their condition score on arrival at the farm. The thin animals within a maturity type being allocated to the thin treatment. This significant difference in condition was maintained over the pre-feedlot period, with the fat animals ending the period on a significantly higher condition score than the thin animals (Table 10). The condition scores were however non-significantly different within the status treatments (EF vs LF and ET vs LT). Non-significant differences between treatments regarding the rate of loss of condition score indicated that even though the animals had differing growth rates the growth rate of the fat group was insufficient to maintain or increase their condition (subcutaneous fat deposition). The exception to this was that of the LF vs ET animals. The ET animals were found to lose condition significantly faster.

Table 10. Change in condition of steers over the pre-feedlot period

ITEM	TREATMENT			
	EF	LF	ET	LT
Initial CS	3.729[a]	3.542[a]	3.125[b]	3.063[b]
Final CS	2.083[a]	2.021[a]	1.396[b]	1.417[b]
Change in CS	-1.646[ab]	-1.521[b]	-1.729[a]	-1.646[ab]

[a,b,c] Values in the same row with different superscripts are different $P < .05$.

Examples, of the four treatments, at the end of the pre-feedlot period are given in Plate's 1, 2, 3 and 4.

Plate 1. Early maturing fat (EF)

Plate 2. Late maturing fat (LF)

Plate 3. Early maturing thin

Plate 4. Late maturing thin

2.4 DISCUSSION

The target length of the restriction period was achieved, allowing for 103 days of controlled growth before the feedlot period. This equated to a 60 kg weight difference (Table 6), at the start of the feedlot trial. Thus, the thin animals (ET and LT), entered the feedlot period 25 per cent lighter than their corresponding fat counterparts (EF and LF).

A point of concern was the grouping of animals according to condition on arrival at the farm. This led to the question of whether the animals were thin due to previous nutritional restriction, illness or genetic factors. To exclude these factors from the equation the following was taken into account. No single group of animals was purchased from one source, thus reducing any one of the factors being responsible. As the groups could be affected by more than one of the factors, it was assumed that one factor would not have the same affect on an animal as another. Canonical variate analysis was performed (2.3), showing that the animals' growth over the pre-feedlot period was consistent within their status treatments. The animals were therefore correctly allocated to the fat or thin treatments, according to their performance, compared to the performance, of all other animals in the trial. Therefore, this excluded the factors mentioned, from being responsible for the thinness of the animals.

From the results (2.3.1 and 2.3.2), it is apparent that the only variable to change significantly over time was that of status. The quadratic terms for both variables (liveweight and condition) have been explained, leaving the expected linear change over time. The ADG was below the target for the fat groups and above the target for the thin groups. They were however significantly different, and resulted in large differences in liveweight between treatments at the end of the period.

The pre-feedlot period achieved all expectations allowing for the groups to have significantly different liveweights and condition scores on entering the feedlot period of the trial. The occurrence of compensatory gain depends on the length and severity of restriction (O'Donovan, 1984). Comparison with published results shows that

compensatory growth could be expected from the treatments imposed during the pre-feedlot period. Similar treatments have been imposed with positive results with respect to compensatory growth (Meyer *et al.*, 1965; Fox *et al.*, 1972 and Folman, *et al.*, 1974).

The fat groups were shown to be gaining in weight, but losing condition. Thus, the animals did not maintain their natural growth (1.2.1.4) with respect to tissue deposition, as their level of fat (subcutaneous) was decreasing over time. This is indicative of *mild restriction* (1.2.1.5), which could subsequently result to some degree of compensation could have been expected. This expectation derives from the work by Hironaka *et al.* (1979), who achieved positive compensation after a restriction at 0.5 kg per day.

CHAPTER THREE
MEASUREMENT OF BODY COMPOSITION

3.1 INTRODUCTION

Prior *et al.* (1977) suggested that there was a need for research on the influence of nutrition, mature size and rate of growth on carcass composition. The requirement has been for animals comparative growth rates to be compared at a point of equality (mature in terms of growth). Maturity, is defined as the anatomical equilibrium reached when an animal has ceased to grow (Butterfield, 1988). With the determination of the composition of a mature animal, it was possible to relate the whole body or part, of an immature animal, in terms of the mature body or part i.e. the degree of maturity that the comparative part has achieved. Reference must again be made to the quote from Butterfield (1988), that is, "it is, therefore, likely that any comparisons made at equal age, equal weight, or equal anything, other than degree of maturity, will give comparisons which have little bearing on the genetic body composition of the animals being compared".

The ash and protein content in an animals body is little affected by restriction (1.2.1.5.5 and 1.2.1.5.7), thus the amounts of ash and protein are considered good measures of maturity. The degree of maturity in terms of protein is expressed as the weight of protein in the body over the weight of protein in the body at maturity. Animals can then be compared at equal degrees of maturity, for factors such as maturity type and previous nutritional differences.

3.2 MATERIALS AND METHODS

3.2.1 CHEMICAL BODY COMPOSITION

The aim was to determine the chemical composition of the steers over time i.e. as they progressed through their fattening phase. Two animals from each pen were examined using the urea dilution technique. A pair of animals were randomly selected every two

weeks until all animals had been examined. Different animals had to be chosen at each measurement as the technique has been found to have a significant effect on the feedlot performance of tested steers compared to those steers not yet examined.

3.2.1.1 The Urea Dilution Technique

The technique used was that described by Preston and Kock (1973). However, a number of practical changes were made due to availability of materials. The animals were starved by denying access to feed and water for 18 hours in order to measure empty body weight. The volume of solution injected was measured to achieve approximately 130 mg of urea per kg live weight. The infusion solution contained 20% urea dissolved in 0.9% saline. This was introduced into the jugular vein of the steer over a two minute period. The solution was infused via an 18 gauge needle with a 50 ml syringe. A sample of the infusion solution was taken each day for analysis. Jugular blood samples from alternate sides were taken prior to and 12 minutes after the mean infusion time. Plasma was removed following centrifugation for urea analysis.

The method described by Preston (unpublished) was used to determine urea nitrogen and is based on the reaction of ammonia with sodiumphenate and hypochlorite to produce a blue colour (Berthelot, 1859). The method described by Preston (unpublished), has been modified to run on a Technicion Autoanalyzer. All reagents and buffers were the same concentrations as those of the original publication. The whole method has been automated, from sampling the raw plasma, through incubation at 37°C to additions of Phenol and Hyperchlorite to final colour reaction at 660nm. The total automation has removed all error due to time and pippeting between samples and standards.

Two control samples were run with each group of animals sampled. The high control was Precinorm U from Boehringer Mannheim

Urea Nitrogen	= 22.80 mg/dl
Urea S	= 49.02 mg/dl
Our result Urea Nitrogen	= 22.11 mg/dl
Standard error %	= 4.83%

The low control was QCS from Ciba-Corning

Urea Nitrogen	= 13.00 mg/dl
Urea	= 27.95 mg/dl
Our result Urea Nitrogen	= 14.33 mg/dl
Standard error %	= 3.63%

3.2.1.2 Calculation of Urea Space

The following formula was used to calculate urea space as a percentage of live weight (Kock and Preston, 1979) : Urea space (US) (%) = Volume infused[a] X concentration of solution[b] ÷ PUN[c] ÷ live weight in kg; where a = volume of urea infused (ml); b = concentration of urea solution infused (mg urea-N/100ml) and c = difference in plasma urea nitrogen (PUN) taken from blood sample prior to and after urea infusion (mg urea-N/100ml).

3.2.1.3 Criterion For Acceptance of Data

Adjustment of the technique as discussed (3.2.1.1), could lead to large human error. This can be manifested in two ways :

a) Due to the needle not remaining within the jugular, the required quantity of infusion solution to enter the blood stream is not met. This results in the difference in PUN concentrations to be small, indicating a large urea space.

b) Contamination of the second blood sample could result from a collection of the urea solution in the tissue around the infusion site. This results in the difference in PUN concentrations to be large, indicating a small urea space.

The range of US % recorded by Bartle *et al.* (1987) = 39.8 to 59.4. The equations presented in 3.2.1.4, predict the point at which the fat percentage is zero to be reached at a US % of 64.08. With this in mind the following criterion were imposed on the data.

The limits imposed on the US % =

$$20\ \% < \text{ACCEPTED} < 64.08\ \%$$

3.2.1.4 Equations for Estimation of *In Vivo* Body Composition

These equations are all on a percentage basis (Bartle *et al.*, 1987) :

Water (%) = 12.4 + 0.95*US[a] $r^2 = 0.67$

Fat (%) = 80.1 - 1.25*US $r^2 = 0.67$

Protein (%) = (0.91 + 0.040*US)*6.25[b] $r^2 = 0.67$

Ash (%) = 100 - (Water (%) + Fat (%) + Protein (%))

[a] US = Urea space as a percentage of liveweight

[b] 6.25 = Conversion factor for nitrogen to protein

3.2.1.5 Calculation of tissue weights

The mean proportions for each tissue (3.2.1.5), were then multiplied by the corresponding mean liveweight of the pen (Table 11). The mean liveweight of the pen was used, as the animals measured, were randomly selected to be representative of the whole treatment.

Table 11. Mean liveweights of treatment's at the time of each urea dilution

	MEAN WEIGHT (kg) FOR EACH TREATMENT			
TIME[a]	EF	LF	ET	LT
3	266.38	302.46	216.74	245.67
5	286.63	325.87	239.17	268.25
7	305.21	353.21	265.17	296.67
9	329.25	375.87	294.61	325.37
11	355.75	404.33	313.65	345.08
13	355.21	411.83	336.81	366.67

[a] : Time (weeks) from the start of the feedlot period.

3.2.2 STATISTICAL METHODS

The GENSTAT V statistical programme (Lawes Agricultural Trust, Rothamstead) was used for all statistical analyses. Regression models were fitted to the data, where relevant. A selection of models was used; linear, quadratic, cubic, gomperitz, linear exponential and broken stick. The model that provided the best statistical fit was chosen. Before a model was chosen, however, its use had to be justified, that is, its parameter must meet with logical understanding in line with existing principles in Animal Science.

Within the regression equations -

T = Time in weeks, from the start of measuring the data in question.

T.T = Quadratic term (Time squared), as above.

3.3 RESULTS

A large amount of data failed to meet the criterion set (3.2.1.3). Examination of the data (Tables 12, 13, 14 and 15), shows that this was predominately due to the urea space being too large (> 64.08%). This was as a result of the difference between the two plasma urea nitrogen values being small. In other words there was a large volume of water for the urea to diffuse into. As explained in 3.2.1.3, this could well be due to an incomplete infusion of the urea solution into the jugular.

A proposal for the opposite effect, namely a very large difference between the two plasma urea nitrogen levels, was that of kidney malfunction (Preston, *pers comm.*). This would result in the animal's body already being saturated with urea, thus the infusion of urea would result in very little diffusing into the body. However, those animals which did follow this error, did not have high plasma urea nitrogen levels at the beginning of the infusion. Thus indicating that the error was rather due to that given in 3.2.1.3.

Overall forty four of the possible ninety six readings were removed due to erroneous results. During the ninth week from the start of the trial all four measurements were removed completely, due to their erroneous nature. This occurred for both the thin treatments (ET and LT).

The mean urea space for each treatment at each time interval was then used in the equations (3.2.1.4), to generate estimates for the body composition proportions over time. These are presented in Tables 16, 17, 18 and 19.

The tissue proportions showed a large amount of variation over time. Examination of the prediction equations (3.2.1.4), revealed that US % makes up a large proportion of the equation for some of the tissues. In an increasing order, protein (0.040), water (0.95) and fat (1.25). If variation was due to the proportion of the prediction made up for by the US %, then the variation will be greater in those tissues with a large response to US %.

Table 12. The variables in the calculation of, Urea Space (%), for animals in the early maturing fat treatment

			PUN[a] CONCENTRATION				
T[b]	SLW[c]	INFUSION AMOUNT	1st	2nd	UREA SOLUTION	US	C[d]
	(kg)	(ml)	mg/ml	mg/ml	mg/ml	%	Y / N
3	249	165	19.07	43.15	18427.16	50.71	Y
3	252	167	23.33	57.28	18427.16	35.97	Y
3	271	179	17.89	44.94	18427.16	45.00	Y
3	248	164	16.10	40.29	18427.16	50.38	Y
5	327	216	24.55	40.23	17990.60	75.82	N
5	304	201	24.10	45.84	17990.60	54.72	Y
5	253	167	13.67	33.50	17990.60	59.91	Y
5	264	175	17.59	109.13	17990.60	13.03	N
7	266	176	23.80	55.51	17253.99	36.00	Y
7	310	205	0.00	0.00	17253.99	0.00	N
7	299	198	16.06	41.34	17253.99	45.19	Y
7	254	168	24.14	52.40	17253.99	40.40	Y
9	336	222	23.76	32.21	16509.92	129.1	N
9	315	208	25.76	47.79	16509.92	49.47	Y
9	342	226	34.87	54.74	16509.92	54.92	Y
9	322	213	26.77	49.82	16509.92	47.38	Y
11	333	220	21.95	23.09	16423.95	952.2	N
11	341	225	24.38	25.41	16423.95	1050	N
11	300	198	22.68	32.40	16423.95	111.5	N
11	316	209	19.11	46.78	16423.95	39.26	Y
13	340	225	23.44	26.19	13129.60	315.7	N
13	307	203	23.48	28.68	16253.36	206.5	N
13	315	208	27.76	51.64	16253.36	44.93	Y
13	376	249	31.69	39.90	13129.60	105.8	N

[a] : Plasma urea nitrogen (mg urea-N / 100ml).
[b] : Time (weeks) from the start of the feedlot period.
[c] : Starved liveweight.
[d] : Criterion for acceptance (3.2.1.3), Yes or No.

Table 13. The variables in the calculation of, Urea Space (%), for animals in the late maturing fat treatment

			PUN[a] CONCENTRATION				
T[b]	SLW[c]	INFUSION AMOUNT	1st	2nd	UREA SOLUTION	US	C[d]
	(kg)	(ml)	mg/ml	mg/ml	mg/ml	%	Y / N
3	288	190	15.37	36.83	18427.16	56.66	Y
3	309	204	21.80	246.37	18427.16	5.42	N
3	290	192	18.90	44.18	18427.16	48.25	Y
3	286	189	14.96	35.11	18427.16	60.45	Y
5	320	212	17.65	31.99	17990.60	83.11	N
5	311	206	20.62	46.48	17990.60	46.07	Y
5	306	202	24.64	39.17	17990.60	81.72	N
5	321	212	19.84	37.07	17990.60	68.99	N
7	364	241	27.07	30.85	17253.99	301.9	N
7	335	222	30.68	49.99	17253.99	59.22	Y
7	320	212	24.44	52.09	17253.99	41.34	Y
7	335	222	23.74	44.68	17253.99	54.60	Y
9	358	237	27.05	35.22	16509.92	133.8	N
9	371	245	27.63	34.87	16509.92	150.5	N
9	329	218	33.54	37.30	16509.92	290.8	N
9	372	246	16.92	42.25	16509.92	43.11	Y
11	398	263	16.75	37.24	16423.95	52.97	Y
11	377	249	19.01	22.12	16423.95	348.0	N
11	384	254	20.68	31.05	16423.95	104.8	N
11	388	256	21.95	41.37	16423.95	55.82	Y
13	360	238	32.04	55.19	13129.60	37.49	Y
13	398	263	31.63	37.69	16253.36	177.1	N
13	346	229	27.52	31.73	16253.36	255.3	N
13	352	233	31.91	38.36	13129.60	134.7	N

[a] : Plasma urea nitrogen (mg urea-N / 100ml).
[b] : Time (weeks) from the start of the feedlot period.
[c] : Starved liveweight.
[d] : Criterion for acceptance (3.2.1.3), Yes or No.

Table 14. The variables in the calculation of, Urea Space (%), for animals in the early maturing thin treatment

T[b]	SLW[c]	INFUSION AMOUNT	PUN[a] CONCENTRATION 1st	2nd	UREA SOLUTION	US	C[d]
	(kg)	(ml)	mg/ml	mg/ml	mg/ml	%	Y / N
3	203	134	18.55	47.77	18427.16	41.63	Y
3	213	141	15.57	42.55	18427.16	45.21	Y
3	211	140	19.91	50.93	18427.16	39.41	Y
3	210	139	12.69	34.66	18427.16	55.51	Y
5	205	136	20.86	31.80	17990.60	109.1	N
5	228	151	17.63	45.41	17990.60	42.89	Y
5	231	153	32.21	63.75	17990.60	37.78	Y
5	195	129	17.57	44.85	17990.60	43.62	Y
7	247	164	21.63	353.42	17253.99	3.45	N
7[e]	***	***	*****	*****	********	****	N
7	266	176	19.50	52.83	17253.99	34.26	Y
7	221	146	24.04	67.64	17253.99	26.14	Y
9	268	177	23.18	28.66	16509.92	198.8	N
9	248	164	22.12	26.42	16509.92	253.9	N
9	301	199	28.32	29.71	16509.92	781.1	N
9	315	208	27.99	32.53	16509.92	240.3	N
11	319	211	15.72	37.13	16423.95	50.73	Y
11	347	229	23.54	44.61	16423.95	51.44	Y
11	297	196	18.92	27.05	16423.95	133.4	N
11	352	233	29.11	52.70	16423.95	46.09	Y
13	324	214	24.12	29.18	16253.36	212.5	N
13	332	220	26.12	32.53	13129.60	135.8	N
13	298	197	32.44	54.09	16253.36	46.63	Y
13	293	194	23.01	46.38	13129.60	37.20	Y

[a] : Plasma urea nitrogen (mg urea-N / 100ml).
[b] : Time (weeks) from the start of the feedlot period.
[c] : Starved liveweight (kg).
[d] : Criterion for acceptance (3.2.1.3), Yes or No.
[e] : Excluded from the trial (illness).

Table 15. The variables in the calculation of, Urea Space (%), for animals in the late maturing thin treatment

			PUN[a] CONCENTRATION				
T[b]	SLW[c]	INFUSION AMOUNT	1st	2nd	UREA SOLUTION	US	C[d]
	(kg)	(ml)	mg/ml	mg/ml	mg/ml	%	Y / N
3	229	152	14.32	38.51	18427.16	50.57	Y
3	223	148	20.30	43.04	18427.16	53.76	Y
3	222	147	17.22	54.42	18427.16	32.80	Y
3	227	150	13.98	42.96	18427.16	42.01	Y
5	266	176	15.37	39.80	17990.60	48.74	Y
5	248	164	22.40	36.85	17990.60	82.34	N
5	232	154	22.96	47.79	17990.60	48.09	Y
5	280	185	14.32	719.78	17990.60	1.69	N
7	249	165	22.36	49.97	17253.99	41.42	Y
7	247	164	24.83	32.12	17253.99	157.2	N
7	268	177	25.99	31.56	17253.99	204.6	N
7	249	165	18.06	38.72	17253.99	55.34	Y
9	330	218	27.31	39.73	16509.92	87.76	N
9	362	239	25.76	39.17	16509.92	81.25	N
9	297	196	17.67	34.01	16509.92	66.68	N
9	266	176	26.19	43.19	16509.92	64.23	N
11	299	198	22.23	37.02	16423.95	73.53	N
11	321	212	22.68	42.53	16423.95	54.66	Y
11	380	251	26.70	49.49	16423.95	47.60	Y
11	388	256	19.78	30.38	16423.95	102.2	N
13	342	226	25.31	49.34	16253.36	44.68	Y
13	325	215	24.14	31.18	16253.36	152.9	N
13	240	159	18.88	41.90	16253.36	46.76	Y
13	400	264	31.00	57.21	13129.60	33.06	Y

[a] : Plasma urea nitrogen (mg urea-N / 100ml).
[b] : Time (weeks) from the start of the feedlot period.
[c] : Starved liveweight.
[d] : Criterion for acceptance (3.2.1.3), Yes or No.

The proportional composition (Tables 16, 17, 18, 19) showed a relatively constant pattern of change, across treatments, that is the US % decreased. This resulted in a decrease in the proportion of the body made up from water and protein. Fat however was found to be increasing. Ash decreased as a proportion, due to the proportion of fat increasing at rate faster than the decrease in the proportion made up from water and protein.

Table 16. Body composition proportions (means) of the early maturing fat animals

TIME (week)	UREA SPACE (% of liveweight)	BODY COMPOSITION (%)			
		WATER	FAT	PROTEIN	ASH
3	45.51	55.64	23.20	17.07	4.09
5	57.32	66.85	8.45	20.01	4.69
7	40.53	50.90	29.44	15.82	3.84
9	50.59	60.46	16.86	18.33	4.35
11	39.26	49.69	31.02	15.50	4.24
13	44.93	55.08	23.94	16.92	4.06

Table 17. Body composition proportions (means) of the late maturing fat animals

TIME (week)	UREA-SPACE (% of liveweight)	BODY COMPOSITION (%)			
		WATER	FAT	PROTEIN	ASH
3	55.12	64.76	11.20	19.47	4.57
5	57.53	56.17	22.51	17.21	4.11
7	51.72	61.54	15.45	18.62	4.39
9	43.11	53.35	26.22	16.46	3.97
11	54.40	64.07	12.11	19.28	4.54
13	37.49	48.01	33.24	15.06	3.73

Table 18. Body composition proportions (means) of the early maturing thin animals

TIME (week)	UREA-SPACE (% of liveweight)	BODY COMPOSITION (%)			
		WATER	FAT	PROTEIN	ASH
3	45.44	55.57	23.30	17.05	4.08
5	41.43	51.76	28.31	16.04	3.89
7	30.20	41.09	42.35	13.24	3.32
9	*[a]	*[a]	*[a]	*[a]	*[a]
11	49.42	59.35	18.32	18.04	4.29
13	43.41	53.64	25.83	16.54	3.99

[a] : Due to the criterion (3.2.1.3), all data for this treatment at this particular time interval were removed for not meeting the requirements.

Table 19. Body composition proportions (means) of the late maturing thin animals

TIME (weeks)	UREA-SPACE (% of liveweight)	BODY COMPOSITION (%)			
		WATER	FAT	PROTEIN	ASH
3	44.78	54.95	24.11	16.88	4.06
5	48.42	58.40	19.58	17.79	4.23
7	48.38	58.36	19.63	17.78	4.23
9	*[a]	*[a]	*[a]	*[a]	*[a]
11	51.13	60.97	16.19	18.47	4.37
13	41.50	51.83	28.22	16.06	3.89

[a] : Due to the criterion (3.2.1.3), all data for this treatment at this particular time interval were removed for not meeting the requirements.

The conversions of the proportional make up of the selected animals (Tables 16, 17, 18, 19), to the weight of tissues of the individual treatments (3.2.1.5), are presented in Tables 20, 21, 22. As with the proportion, the mean weights of the tissue showed a large amount of variation. The weights of the individual tissues for each treatment were regressed on time. This was done in order to derive prediction equations. The prediction equation for the change in protein weight over time, was then to be used (together with the maximum protein weight at maturity for each maturity type), for the estimation of the animals physiological age over the period measured. The regressions are presented in Figure's 20 and 21 for water, Figure's 22 and 23 for fat and Figure's 24 and 25 for protein.

Table 20. Mean weight of water in the body per treatment at the time of each urea dilution

	MEAN WEIGHT OF WATER (kg) FOR EACH TREATMENT			
TIME[a]	EF	LF	ET	LT
3	148.2	195.9	120.4	135.0
5	191.6	183.0	123.8	156.6
7	155.4	217.4	109.0	173.1
9	199.1	200.5	*[b]	*[b]
11	176.8	259.1	186.2	210.4
13	195.6	197.7	180.7	190.0

[a] : Time (weeks) from the start of the feedlot period.

[b] : Due to the criterion (3.2.1.3), all data for this treatment at this particular time interval were removed for not meeting the requirements.

Table 21. Mean weight of fat in the body per treatment at the time of each urea dilution

	MEAN WEIGHT OF FAT (kg) FOR EACH TREATMENT			
TIME[a]	EF	LF	ET	LT
3	61.81	33.88	50.50	59.24
5	24.23	73.35	67.71	52.54
7	89.85	54.56	112.30	58.24
9	55.51	98.55	*[b]	*[b]
11	110.35	48.96	57.47	55.87
13	85.04	136.89	87.00	103.49

[a] : Time (weeks) from the start of the feedlot period.

[b] : Due to the criterion (3.2.1.3), all data for this treatment at this particular time interval were removed for not meeting the requirements.

Table 22. Mean weight of protein in the body per treatment at the time of each urea dilution

	MEAN WEIGHT OF PROTEIN (kg) FOR EACH TREATMENT			
TIME[a]	EF	LF	ET	LT
3	45.46	58.88	36.95	41.48
5	57.37	56.08	38.37	47.72
7	48.28	65.76	35.10	52.75
9	60.36	61.87	*[b]	*[b]
11	55.14	77.98	56.59	63.74
13	60.10	65.02	55.71	58.90

[a] : Time (weeks) from the start of the feedlot period.

[b] : Due to the criterion (3.2.1.3), all data for this treatment at this particular time interval were removed for not meeting the requirements.

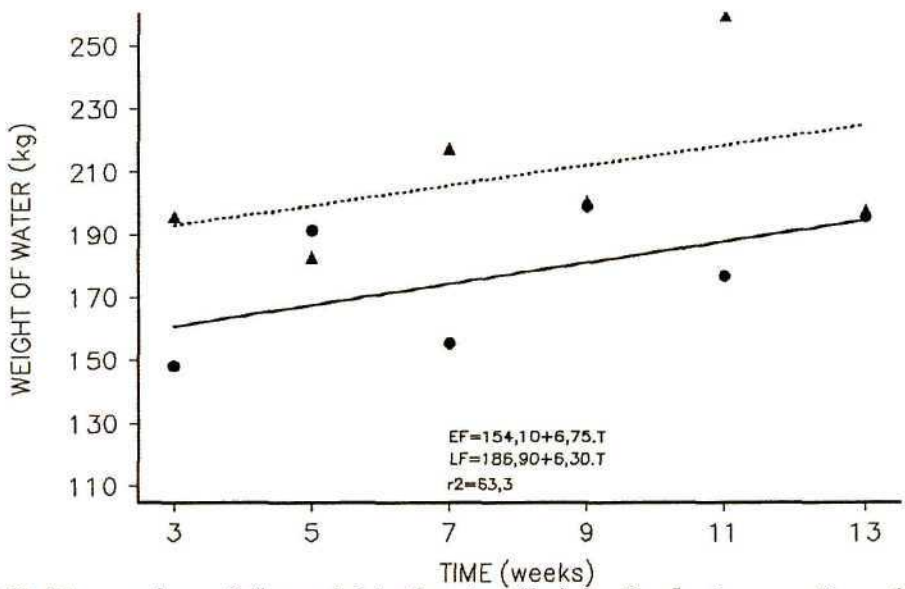

Figure 20. Regression of the weight of water (kg) in the body over time (weeks). Data points = EF : ●, LF : ▲. Regression models = Early Maturing : ———, Late Maturing = · · · ·

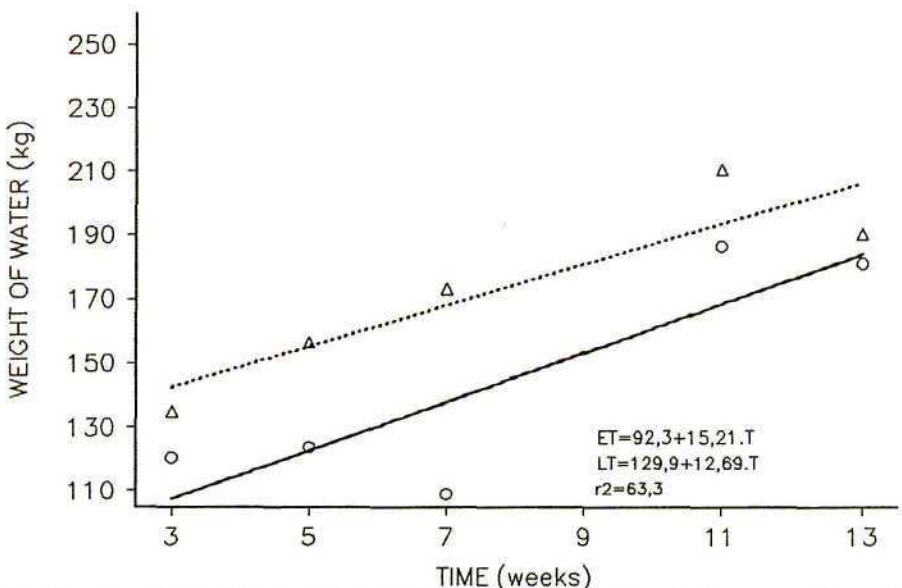

Figure 21. Regression of the weight of water (kg) in the body over time (weeks). Data points = ET : ○, LT : △. Regression models = Early Maturing : ———, Late Maturing = · · · ·

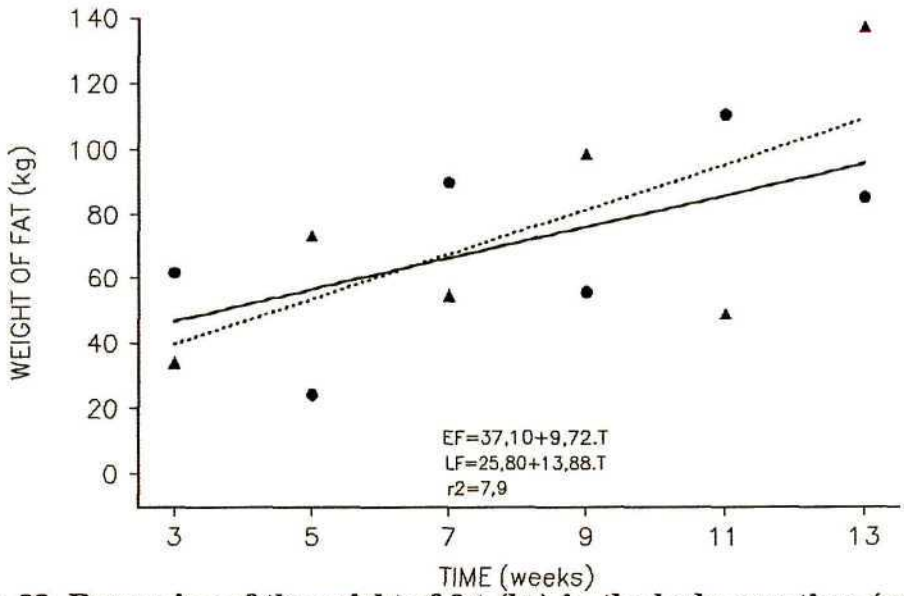

Figure 22. Regression of the weight of fat (kg) in the body over time (weeks). Data points = EF : ●, LF : ▲. Regression models = Early Maturing : ————, Late Maturing = · · · ·

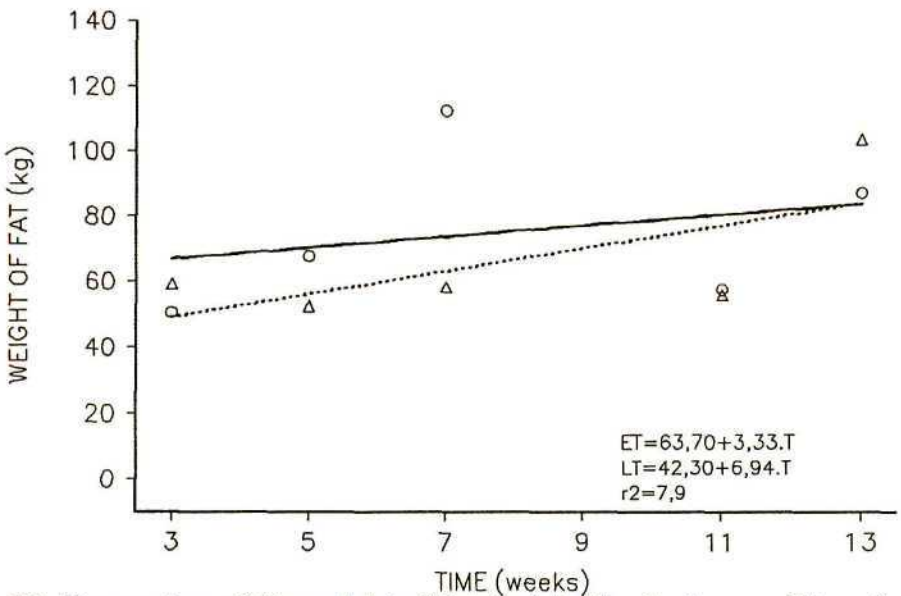

Figure 23. Regression of the weight of fat (kg) in the body over Time (weeks). Data points = ET : ○, LT : △. Regression models = Early Maturing : ————, Late Maturing = · · · ·

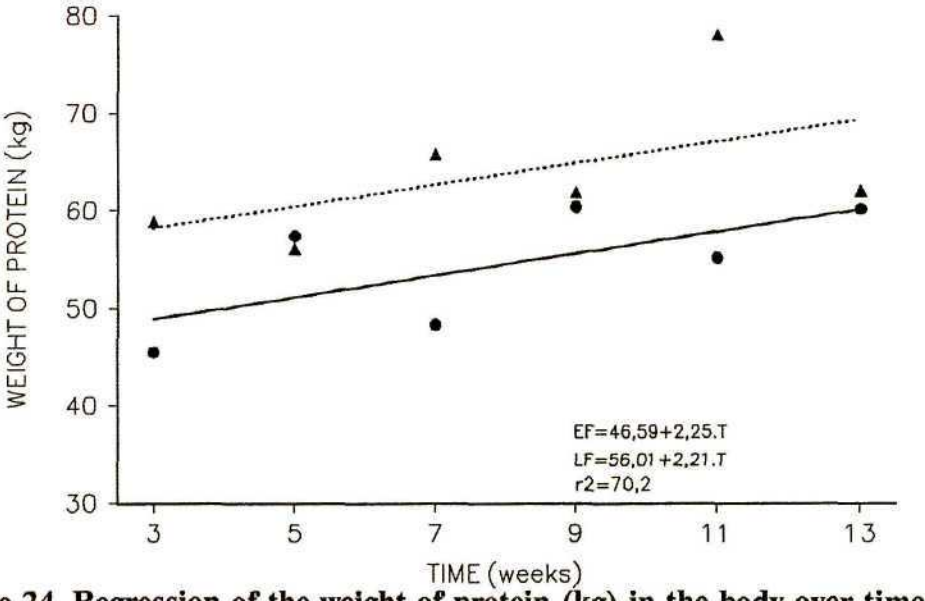

Figure 24. Regression of the weight of protein (kg) in the body over time (weeks). Data points = EF : ●, LF : ▲. Regression models = Early Maturing : ————, Late Maturing = · · · ·

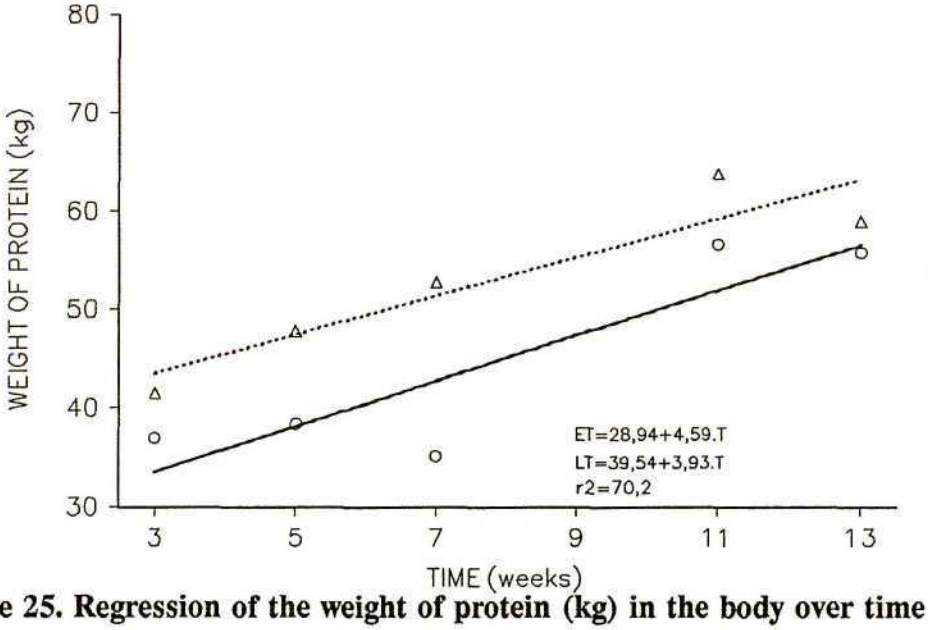

Figure 25. Regression of the weight of protein (kg) in the body over time (weeks). Data points = ET : ○, LT : △. Regression models = Early Maturing : ————, Late Maturing = · · · ·

The regression of the weight of the body tissues over time generated linear models, as those to have the best fit (Figures' 20, 21, 22, 23, 24 and 25). The full breakdown of the models are in Appendix 1. The fit of the models was affected by the amount of variation that they could account for. The R^2 was highest for the model predicting protein, followed by water and then fat. The protein figures were generated from the prediction equations with the lowest US % contribution. As explained earlier the lower the contribution by US %, the lower the variation due to the measurement. The variation in the weight of fat within the body was large, resulting in a poor accounting for variation by the regression model (R^2 = 7.9). Water and protein varied to a lesser degree than fat had a lot more accounted for by their respective regression equations (R^2 = 63.3 and R^2 = 70.2 respectively).

Table 23. Regression equations of the weight of water (kg) in the animals body on time (weeks)

TERM	TREATMENT			
	EF	LF	ET	LT
Constant	154.1[a]	186.9[a]	92.3[b]	129.9[ab]
Linear	6.75[a]	6.30[a]	15.21[a]	12.69[a]

[a,b,c] Values in the same row with different superscripts are different $P < .05$.

Table 24. Regression equations of the weight of fat (kg) in the animals body on time (weeks)

TERM	TREATMENT			
	EF	LF	ET	LT
Constant	37.1[a]	25.8[a]	63.7[a]	42.3[a]
Linear	9.72[a]	13.88[a]	3.33[a]	6.94[a]

[a,b,c] Values in the same row with different superscripts are different $P < .05$.

Table 25. Regression equations of the weight of protein (kg) in the animals body on time (weeks)

TERM	TREATMENT			
	EF	LF	ET	LT
Constant	46.59[a]	56.01[a]	28.94[b]	39.54[a]
Linear	2.25[a]	2.21[a]	17.66[a]	3.93[a]

[a,b,c] Values in the same row with different superscripts are different $P < .05$.

Tables 23, 24, and 25 give the breakdown of the regression models and the statistical comparison of their components between treatments.

Statistical differences between treatments with respect to the constant term were found. The statistical differences were observed for those tissues that showed the least amount of variation. The early maturing thin animals had significantly ($P<0.005$) smaller

constant, against all the other treatments. With respect to water, the constant for the early maturing thin animals, was significantly different from the fat treatments only. There were no significant differences between treatments, with regards the constant, for the regression of fat. Even though significant differences were observed, the differences between those treatments found to be non-significantly different were also large.

Irrespective of treatment or tissue, no significant differences were observed for the rate of change of the tissues over time (the linear component of the models). This was improbable, as has already been shown (6.3), the rate of fat deposition was significantly greater for the thin than for the fat treatments. This was also shown to a limited extent by the greater gain in eye-muscle diameter (lean tissue), by the early maturing thin animals over the early maturing fat animals (5.3.2).

3.4 DISCUSSION

The large amount of variation attributable to the US % measurements, made comparisons between treatments impossible. Although the technique is cost effective for the determination of tissue composition *in vivo*, there are a number of drawbacks. The infusion of all of the solution into the animal has to be guaranteed. Large volumes are required for heavy animals which make the infusion process more difficult to achieve. This is complicated by the large infusion volume. The more solution that has to be infused over the two minutes, and the greater the need to fully restrain the animals leading to increased stress levels, the greater the chance of inaccurate measurements.

It can be concluded that the use of the urea dilution technique did not achieve the intended objective. The elimination of roughly 50 % of the measurements before analysis already placed a possible bias on the remaining data. The attempts to use the remaining data were hampered by the large variations between measurements. This was found to vary between tissues, due to the degree of varying influence on the equation's that the US % had.

The water content of the body has been found to vary from between 80 to 40 per cent (Berg and Butterfield, 1976). The predicted water contents in this trial fell within this range. Water deposition was credited to those tissues that were actively growing (1.2.1.4.4). Protein showed a steady increase relative to liveweight, but decreased as a proportion of the carcass (Seebeck and Tulloh, 1969). The results follow this trend. Protein increased at relatively constant rate irrespective of maturity type or pre-feedlot plane of nutrition. Fat increased at a linear rate, before following an accelerated rate of deposition (1.2.1.4.6). As the animals were in a feedlot trial, with *ad lib* access to high energy feed, it was expected that they would experience this accelerated rate of fat deposition. The results however found the fat deposition to be of a linear trend throughout the animals time within the feedlot.

With the comparison of maturity types, the late maturing animals were expected to have a greater deposition rate of water and protein, due to their higher growth rate in lean tissue (1.2.1.7). Similarly, the earlier maturing animals were expected to have a higher fat deposition rate during the initial period in the feedlot. This appears to have happened in part (6.3), but is not supported with the results form the urea dilution.

Compensatory growth, irrespective of maturity type, provides for an increased rate of tissue deposition. The animals had a higher nutrient intake per kilogram of liveweight (see chapter 4), during realimentation, and are therefore able to exhibit a higher tissue deposition rate. The high energy intakes should definitely be expressed in the form of higher fat deposition rates with respect to non-compensating animals. Fat deposition rates was significantly greater for the thin animals over the fat animals were found (6.3), but again this was not supported by the urea dilution.

CHAPTER FOUR

LIVEWEIGHT GAINS AND FEED INTAKES DURING FEEDLOT PERIOD

4.1 INTRODUCTION

An important consideration in the minds of producers, is whether the use of low cost winter fodder, with the associated reduction in growth, is offset by increased gains and efficiencies in the feedlot. In a comprehensive review of compensatory growth Wilson and Osbourn (1960), concluded that animals are able to recover from periods of undernutrition by (a) prolonging the growth period and (b) increasing appetite and rate of weight gain. A true comparative measure of dry matter intake (DMI), is the weight of feed eaten (kg) per unit of liveweight (kg). In this respect when compared on a liveweight basis, the compensating animals being lighter than the controls, have a higher DMI per kilogram of liveweight. However, compensating animals, have been found to have similar daily intake's as the controls.(Allden, 1970).

Hicks *et al.* (1990a) proposed that DMI is controlled by the body composition of the animals. The fat content exerts a negative feedback control on DMI. Consequently the DMI expressed per unit live weight of an animal will decrease as it approaches a slaughter condition. The DMI by compensating animals matched the controls until twelve weeks into the realimentation period, from when DMI was higher for the compensating animals (Ryan *et al.*, 1993). This is an illustration of the feedback mechanism, as the control animals would have been closer to a slaughter condition, that is they had a higher fat content at an equal chronological age (1.2.1.5.6).

Since the compensating animals eat more per kilogram liveweight, an important consideration would be the efficiency of growth (kilograms of feed per kilogram of weight gain). In order to have matched or bettered the efficiency of growth achieved by the controls, the compensating animals rate of gain has had to be superior. Improved efficiency of feed utilisation during the rehabilitation period has been attributed by Allden (1970), to a lowered basal metabolism or (more possibly) an increase in efficiency of energy utilisation for weight gain.

Hicks *et al.* (1990a) stated that cattle of different genders differ in the weight at which they reach a given degree of carcass and inter-muscular lipid. Since DMI is regulated by body composition, DMI differed between gender (Hicks *at al.*, 1990). Similarly as maturity types differ in body composition due to their degree of maturity (1.2.1.7), it is postulated that DMI would differ between maturity types. Previous nutritional history that affects body composition (1.2.1.5) will also affect future DMI on re-alimentation.

4.2 MATERIALS AND METHODS

4.2.1 LIVEWEIGHT

At the beginning of the feedlot period the animals were weighed individually on an empty body weight basis after denying access to feed and water for 18 hours. Every seven days during the feedlot period individual weights were obtained on a full body weight basis. On reaching a slaughter condition (6.2.1), each steer was weighed again on an empty body weight basis.

4.2.2 FEED INTAKE

The animals were fed on a pen basis after bunker scoring twice daily. This allowed for the animals to be fed according to their intake (*ad lib*), while removing the risk of them exhibiting the "*roller coaster effect*" common in feedlots. The ration was mixed weekly and consisted of the following:

Table 26. The feedlot ration's ingredient's and their proportions

INGREDIENT	QUANTITY (kg's)
Hominy Chop :	585.00
Broiler Chicken Litter :	240.00
Molasses :	120.00
Premix :	50.00
- Hominy Chop	23.63
- Romensin	0.12
- Tylan 100	0.10
- Vitamin A and Mineral Concentrate	0.71
- Broiler Chicken Litter	4.85
- Feedlime	10.50
- Salt	5.05
- Urea	5.05
Total	995.00

4.2.3 **FEED COMPOSITION**

An important consideration was that of the feed's nutrient composition changing over time. To check for this, samples of each of the ingredients as well as that of the mixed ration (hand snatches from at least ten bags), were taken every two weeks. The results of which are presented in Table 27. The samples were analyzed for : crude protein (CP), calcium, phosphorous, fat (EE), ash, moisture, neutral detergent fibre (NDF), acid

detergent fibre (ADF) and crude fibre (EE). The digestible energy (DE) content of the feed was calculated using the equations from Dunbar *et al.* (1991).

$$DE = 3{,}729697 + 0{,}0080470.CP + 0{,}0458200.EE - 0{,}0393000.ASH - 0{,}0392000.CF \quad Mcal/kg$$

Metabolisable energy was then calculated using NRC (1984).

$$ME = 0.82*DE \quad Mcal/kg$$

Converted to MJ/kg by multiplying by a factor of 4.18

Table 27. Composition of calculated mixed ration

CALCULATED MIXED RATION[1]		
COMPOSITION	MEAN %	CV %
CRUDE PROTEIN	14.5	4.34
CALCIUM	1.8	7.78
PHOSPHOROUS	0.7	12.86
FAT	6.7	15.52
ASH	9.1	4.18
MOISTURE	16.8	5.18
N.D.F	28.2	11.38
A.D.F.	12.8	8.20
CRUDE FIBRE	10.3	11.26
M.E. (MJ/kg)	11.6	2.07

[1] : The composition of the calculated mixed ration = the sum of the composition of the trial ration ingredients in their respective proportions.

The full breakdown of the nutrient concentrations for the ingredients and mixed ration are in Appendix 2. The composition means for each of the ingredients, multiplied by the proportion of the ingredient in the diet, generated the calculated mixed ration. The calculated mixed ration was chosen as the indicator for any changes in the ration. This was due to the inclusion of broiler chicken litter in the ration (Table 26). A sample of the mixed ration (even though it was made up from at least ten bags), had a high chance of including a large clump of chicken litter. In Appendix 2, it can be seen that the variation between measurements was higher for the mixed ration than for the calculated mixed ration.

No nutrient differed significantly over time with respect to the calculated mixed ration. This means that irrespective of the length of time an animal spent in the feedlot, the ration composition never became a factor regulating growth responses.

4.2.3.1 Net Energy Available for Growth

The net energy for maintenance (NE_m) was calculated using :

$$NE_m = 0.077 * W^{0.75} * 4.1855 \text{ (MJ/day)}$$

where : $W^{0.75}$ is the metabolic weight of the animal (NRC, 1984).

Metabolisability of the energy in the feed (q_m) :

$$11.6 / 18.4 = 0.63$$

Efficiency of utilisation of metabolisable energy for maintenance (k_m) :

$$k_m = 0.35 * q_m + 0.503$$

$$k_m = 0.35 * 0.63 + 0.503 = 0.72$$

Equations from McDonald *et al.* (1990).

Net energy available for growth :

$$NE_g(\text{MJ/Week}) = (\text{ME in feed consumed per week} * k_m) - (NE_m * 7)$$

$$NE_g(\text{MJ/Week}) = (11.6\text{MJ/Kg} * \text{Feed Intake Kg/Week} * 0.72) - (NE_m * 7)$$

4.2.4 VACCINATIONS AND IMPLANTS

At the start of the pre-feedlot period all the animals were inoculated against quarter evil, botulism and anthrax. On starting the feedlot period the animals were dosed with Panicure (a general deworming medicine) and had Ralgro implants (growth stimulants) implanted.

4.2.5 STATISTICAL METHODS

The GENSTAT V statistical programme (Lawes Agricultural Trust, Rothamstead) was used for all statistical analysis. Regression models were fitted to the data, where relevant. A selection of models was used; linear, quadratic, cubic, gomperitz, exponential, linear exponential and broken stick. The model that provided the best statistical fit was chosen. Before a model was chosen however its use had to be justified, that is, its parameters must meet with logical understanding in line with existing principles in Animal Science. Statistical differences between means were determined from analysis of variance tables with the use of the *Students' T test* (Steel and Torrie, 1980)

Within the regression equations -

T = Time in weeks, from the start of measuring the data in question.

T.T = Quadratic term (Time squared), as above.

Z1 = The first linear component of the broken stick model. (5(1);12(0)) multiplied by Time in weeks.

Z2 = The second linear component of the broken stick model. (5(0);12(1)) multiplied by Time in weeks.

Z3 = The constant term for the second linear model. (5(0);12(1).

L = the natural log of the liveweight at a point in time.

4.3 RESULTS

4.3.1 ILLNESS

One animal was excluded within twenty four hours, after injuring itself trying to escape from the feedlot. Another animal was excluded during the trial, after receiving bullying related injuries to it's back. Four weeks into the feedlot period eighteen animals developed scours, had reduced weight gains and appeared weak. These animals were examined by a veterinarian. The diphtheric membrane underneath the tongue, where the tongue is in contact with the mucous membrane was covered with unusual lesions. These appeared to be chemical burns. On analysis they were diagnosed as uremic lesions caused by the animal not being able to excrete urea. The symptoms cleared up within two weeks. It was shown that this outbreak was unrelated to the urea dilution technique, as half of the animals affected had not had the technique performed on them as yet. This illness did however increase the variation in liveweight and feed intake during the period of outbreak. The effect was felt by all animals from all treatments.

4.3.2 LIVEWEIGHT

As the animals were slaughtered at a set condition score (6.2.1), a point had to be decided upon, from which the remaining animals were no longer representative of the treatment as a whole. This was taken as the sixteenth week of the trial.

The liveweight measurements were regressed on time. A quadratic model was found to fit the data best (R^2 = 99.3). The models and the data are illustrated in Figure's 26 and 27, with the model components and their significant differences between treatments in Table 28. Appendix 3 provides the breakdown of the mean weights, number of observations and regression models.

Significant differences were found between maturity types with respect to their starting masses (the constants). The early maturing treatments were significantly lighter than late maturing treatments (EF vs LF and ET vs LT). The difference in liveweight between

maturity types was expected, with early maturing animals being lighter than late maturing animals (1.2.1.7). The pre-feedlot period (2.3.1), had a significant effect on the treatments, as the thin animals, were all significantly lighter than the respective fat animals (EF vs ET and LF vs LT).

The growth rates are given in Table 28. The growth rates of the compensating animals (the linear component of the regression models), were significantly higher than those for their respective non-compensating animals (EF vs ET and LF vs LT). This higher rate of growth by the compensating animals, would have allowed for any abnormalities within composition to be corrected. This however can not be shown, due to the urea dilution technique not providing consistent results (Chapter 3).

Significant growth differences between maturity types were also expected, with late maturing animals growing at a faster rate than the early maturing animals (1.2.1.7). This only occurred between the fat animals (EF vs LF). The compensating early maturing animals (ET), grew at a similar rate ($P > 0.05$) to either of the late maturing treatments. This could indicate that the growth rate achieved by the late maturing thin animals was their maximum achievable. As their growth demands, due to maturity type, are higher than those for the earlier maturing animals, they should have grown at a greater rate.

The quadratic component was fitted as it was a significant term (3). There was however no significant differences between the quadratic terms for the different treatments. Thus the growth rate was declining at a similar rate for treatments.

Table 28. Components and their significant differences, for the regression models of liveweight (kg) over time (weeks)

TERM	TREATMENT			
	EF	LF	ET	LT
Constant	231.81[b]	263.78[a]	177.09[d]	202.40[c]
Linear	12.591[c]	14.103[b]	14.585[ab]	15.144[a]
Quadratic	-0.1312[a]	-0.2215[a]	-0.2115[a]	-0.2574[a]

[a,b,c] Values in the same row with different superscripts are different $P < .05$.

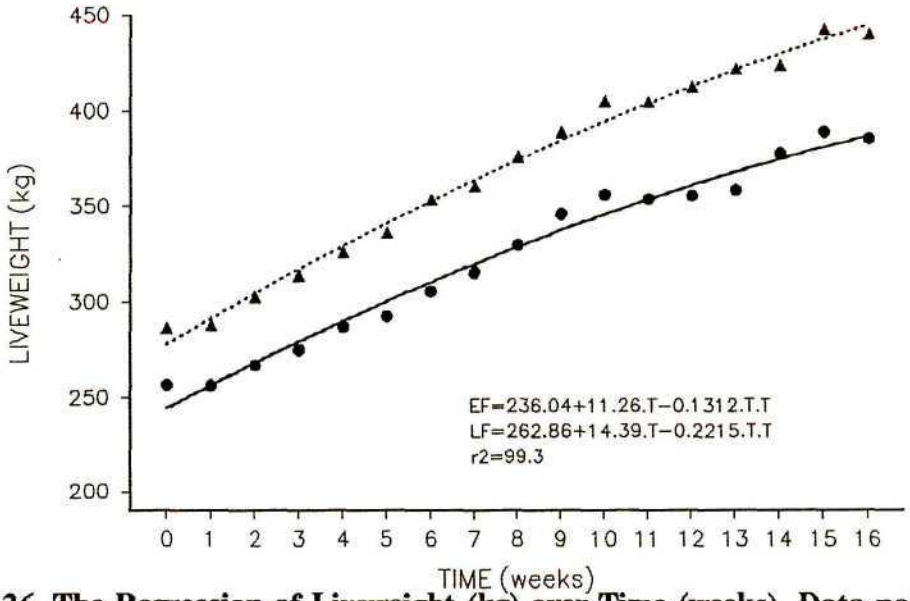

Figure 26. The Regression of Liveweight (kg) over Time (weeks). Data points = EF : ●, LF : ▲. Regression models = Early maturing : ———, Late maturing = · · · ·

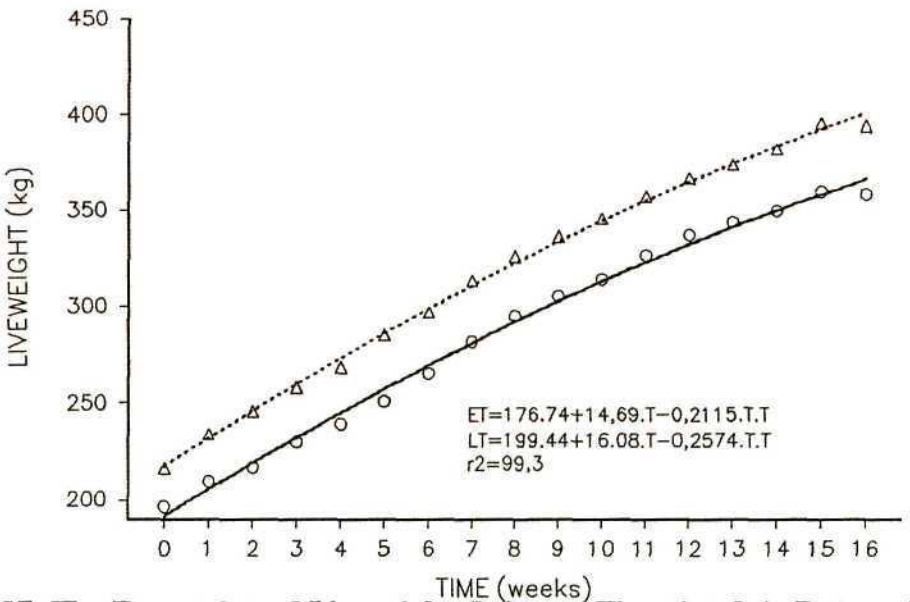

Figure 27. The Regression of Liveweight (kg) over Time (weeks). Data points = ET : ○, LT : △. Regression models = Early maturing : ———, Late maturing = · · · ·

The performance of the animal's over the feedlot period is given in Table 29. All treatments started the feedlot period at significantly different weights. The early maturing treatments (EF and ET) finished the feedlot period at similar ($P > 0.05$) live weights. The early maturing thin animals spent a significantly longer period of time in the feedlot, but they gained live significantly faster than the fat early maturing ones. The late maturing treatments followed similar trends. The late maturing thin animals however finished the feedlot period significantly lighter, despite spending a significantly longer period of time in the feedlot and gaining weight at a significantly greater rate.

Comparison of treatments on a maturity type basis, showed that between the fat treatments (EF vs LF), the late maturing animals gained a significantly greater amount, at a significantly greater rate, over a significantly longer period of time in the feedlot. Within the thin treatments however (ET vs LT), the only significant differences to come out of the feedlot period was that of the amount of weight gained. The late maturing animals gained a significantly greater amount of liveweight. This was achieved by growing at a faster ($P < 0.05$) rate and over a longer ($P < 0.05$) period of time than the early maturing animals. The difference between the growth rate of maturity types is expected, due to the respective positions of the maturity types on their growth curves (1.2.1.7). The similarity between the compensating treatments could be an indication that , under the conditions of the experiment, their rate of growth was at a maximum. Thus the late maturing thin animals could not express any faster rate of growth that they may have been possible on for example, a higher energy ration.

Table 29. Liveweight performance of steers over the feedlot period, showing significant differences between treatments

ITEM	TREATMENT			
	EF	LF	ET	LT
Initial Weight (kg)	256.3[b]	286.0[a]	196.5[d]	216.3[c]
Final Weight (kg)	363.6[c]	439.9[a]	353.7[c]	396.8[b]
Time (days)	95.8[b]	118.2[a]	117.2[a]	124.1[a]
Weight Gain (kg)	107.2[c]	153.9[b]	157.3[b]	180.5[a]
ADG (kg/day)	1.131[c]	1.303[b]	1.359[ab]	1.461[a]

[a,b,c] Values in the same row with different superscripts are different $P < .05$.

4.3.2.1 Efficiency of Gain

Efficiency of gain is defined as the change in liveweight between weekly measurements (on a daily basis; average daily gains), as a proportion of the liveweight at the beginning of the week. This provides a measure of the animals growth rate in relation to its liveweight. As seen in 4.3.2, an animal's change in liveweight decreases over time. Thus the efficiency of gain should decrease over time as well. This was true for all treatments, with quadratic models being found to fit the data best. The models of the respective treatments are illustrated in Figure's 28 and 29. Appendix 4 has the full breakdown of the regression models.

The efficiency of gain was significantly greater (Table 30), for the compensating treatments (ET and LT vs EF and LF). This was due to the compensating animals having greater rate of gain and lighter liveweights, than the non-compensating animals (4.3.2). As the animals grew, their rate of growth decreased and their liveweights increased,

which resulted in a decrease in efficiency of gain. This decrease in efficiency of gain was however non-significantly different between treatments. This was not expected as the compensating animals should have had a faster rate of decline in efficiency of gain than the non-compensating animals. From 4.3.2 it was seen that the compensating treatments were growing at a significantly greater rate than their respective fat treatments. However, the rate of decline in growth was non-significantly different between treatments. Thus on a weekly basis the compensating animals were gaining a significantly greater amount in liveweight. According to the definition of efficiency of gain the compensating animals should have a significantly greater decline in efficiency of gain than the non-compensating treatments. A reason for this not having occurred, could be that the fitted models did not account for enough of the variation between the data points within a treatment (R^2 = 39.1).

Table 30. Components and their significant differences for the regression of treatments average daily gains (kg/day) / liveweight (kg) over time (weeks)

TERM	TREATMENT			
	EF	LF	ET	LT
Constant	0.00133[b]	0.00296[b]	0.00664[ab]	0.00954[a]
Linear	0.000845[a]	0.000564[a]	0.000281[a]	-0.000448[a]
Quadratic	-.0000520[a]	-.0000409[a]	-.0000366[a]	-.0000005[a]

[a,b,c] Values in the same row with different superscripts are different $P < .05$.

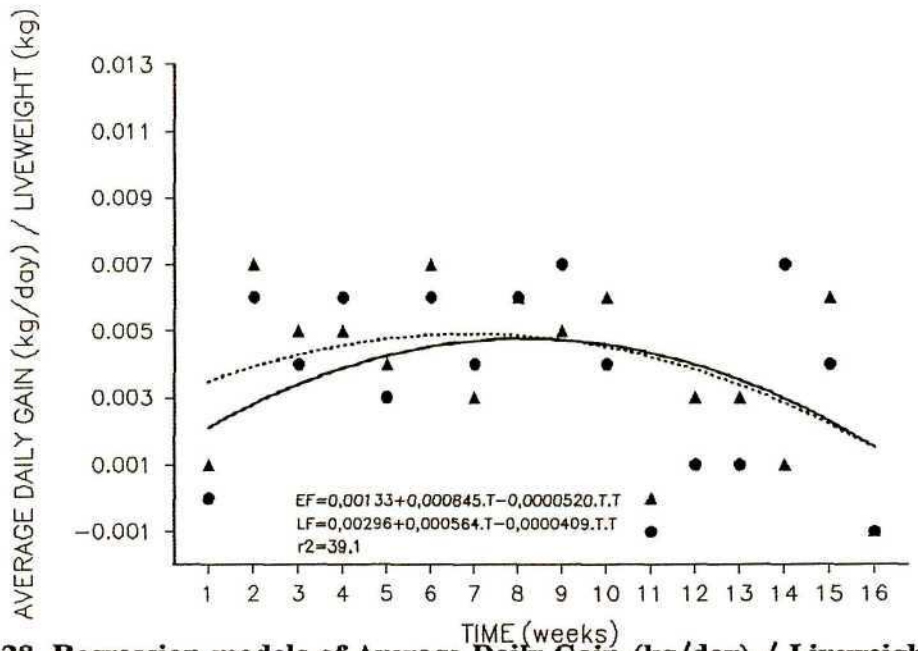

Figure 28. Regression models of Average Daily Gain (kg/day) / Liveweight (kg) over Time (weeks). Data points = EF : ●, LF : ▲. Regression models = Early maturing : ———, Late maturing = · · · ·

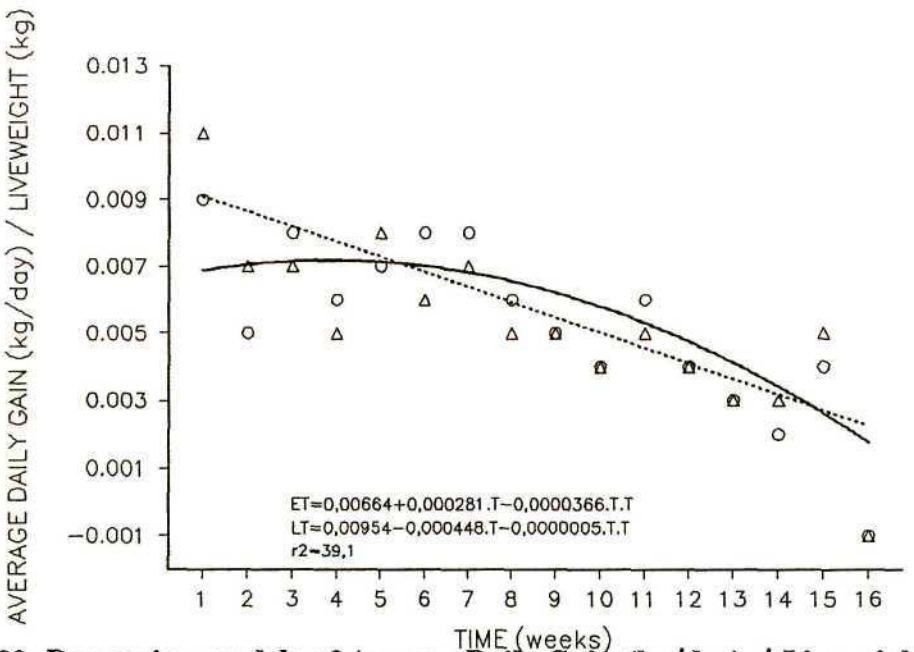

Figure 29. Regression models of Average Daily Gain (kg/day) / Liveweight (kg) over Time (weeks). Data points = ET : ○, LT : △. Regression models = Early maturing : ———, Late maturing = · · · ·

4.3.3 FEED INTAKE

During the initial weeks in the feedlot, the feed intake of the animals should increase at a decreasing rate. This increase is due to the adaptation of the ruminant to the characteristics and quantity of feed available. Once the animal is adapted to the diet its feed intake should match its requirements, or as close as the animal is able to get it, with respect to the factors that control voluntary feed intake.

Table 31. Regression equations, for the quadratic models, of feed intake (kg) per week over time (weeks)

TERM	TREATMENT			
	EF	LF	ET	LT
Constant	34.20[a]	39.16[a]	30.61[a]	37.08[a]
Linear	5.03[a]	6.34[a]	6.11[a]	6.09[a]
Quadratic	-0.1489[a]	-0.2526[a]	-0.2616[a]	-0.2523[a]

[a,b,c] Values in the same row with different superscripts are different $P < .05$.

The regression models fitted to the data (Appendix 5), were allowed to extend to a quadratic term which was significant. The breakdown of the regression models is in Appendix 6. The fitted models accounted for a large amount of the variation (R^2 = 82.0). The quadratic models for the differing treatments are illustrated in Figure's 30 and 31. The models are given in Table 31.

Regression models show that treatments had similar starting intakes (constants), linear rate of increase in feed intake and similar rates of decrease in the increasing feed intake (quadratic).

Examination of the data points and the quadratic models, for the differing treatments, over time, exposed some discrepancies. The data appeared to follow a linear trend up to a peak intake point. Beyond this point the data appeared to plateau in a linear manner. The quadratic models could not account for the peak feed intake. Thus these models would underestimate the peak feed intake. The peak feed intakes for the different treatments to occur at the same point of time. This was unexpected, as the treatments were made up of two maturity types and two pre-feedlot nutrition planes, thus providing animals of widely differing liveweights and nutritional requirements. It was thus decided to fit an alternative model, a broken stick. This was to query the two linear trends of feed intake over time and the appearance of the peak feed intake occurring at the same point of time irrespective of treatment.

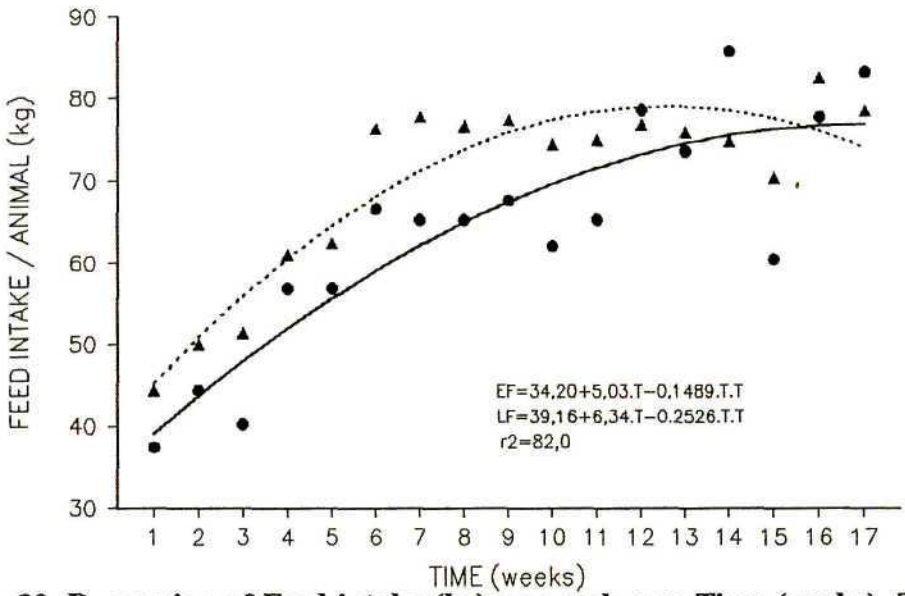

Figure 30. Regression of Feed intake (kg) per week over Time (weeks). The models fitted are quadratic. Data points = EF : ●, LF : ▲. Regression models = Early maturing : ———, Late maturing = · · · ·

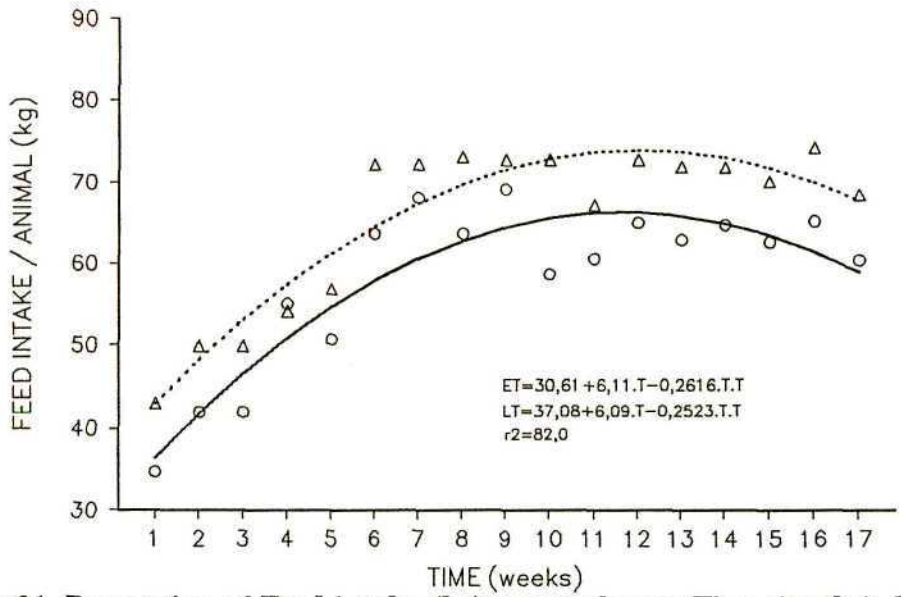

Figure 31. Regression of Feed intake (kg) per week over Time (weeks). The models fitted are quadratic.Data points = ET : ○, LT : △. Regression models = Early maturing : ———, Late maturing = · · · ·

The underlying assumption of a broken stick model is that at some point the direction of regression changes drastically. For example, the assumption in using a broken stick model with the feed intake data is that, the feed intake increases at a linear rate until a peak intake is reached, from this point on the feed intake follows a plateau.

Table 32. Regression equations, for the broken stick models, of feed intake (kg) per week over time (weeks)

TERM	TREATMENT			
	EF	LF	ET	LT
Constant	27.32^{c}	41.90^{a}	33.32^{bc}	41.46^{ab}
Linear Z1	6.34^{a}	4.10^{ab}	3.96^{ab}	3.11^{b}
Linear Z2	1.063^{a}	0.225^{b}	-0.120^{b}	-0.137^{b}
Z3	31.62^{a}	31.62^{a}	31.62^{a}	31.62^{a}

^{a,b,c} Values in the same row with different superscripts are different $P < .05$.

Fitting a broken stick model to the feed intake data increased the coefficient of determination (R^2 = 88.3; Appendix 7). This implies that the broken stick models are a better fit of the data than the quadratic models (Figure's 32 and 33). With the broken stick models differences in the feed intakes were found between treatments (Table 32). The early maturing fat animals had a significantly lower feed intake at the start of the trial, as compared to the late maturing treatments. The feed intake for the early maturing thin animals was greater ($P>0.05$) than that of the early maturing fat treatment. The high initial feed intake by the early maturing thin animals resulted in the

compensating treatments being non-significantly different (ET and LT). The differences in feed intake between the late maturing treatments was not significant.

The first linear component (Z1), showed that the early maturing fat animals had the greatest rate of intake, which was significantly different from the late maturing thin treatment. Thus the early maturing animals started at the lowest feed intake, but made up for this by increasing their intake at the fastest rate.

The point at which peak feed intake was reached was tested on an individual treatment basis and on a between treatment basis. There was no difference between treatments as to when the peak feed intake point occurred. It consistently occurred at the sixth week (42 days). The treatments were expected to have differing nutrient requirements. However, the feed intake results don't match this expectation. If the later maturing animals (especially the compensating animals), had greater nutrient requirements due to their larger metabolic weight and greater growth requirements, then their feed intake should start at a greater amount or increase at a greater rate. As this did not occur consistently, then the rate of feed intake increase should have continued for a greater period of time. Similarly the feed intake for the thin treatments would be expected to increase at a greater rate or over a longer period of time compared to the fat treatments. For all treatments the change in feed intake pattern occurred at 42 days into the feedlot period. This implies that a restriction of some kind was limiting the animals ability to satisfy their nutrient requirements.

The plateau stage (Z2), was not at a constant feed intake but rather one that intake either slightly increased or decreased according to treatment. The fat animals had a positive change in feed intake beyond the peak intake point, with the early maturing fat animals being significantly different from the other treatments. The thin treatments were not significantly different from the late maturing fat animals.

Thus it seemed from the analyses that the treatments started and increased their feed intakes at a maximum possible rate. Feed intake was restricted in all treatments at about 42 days.

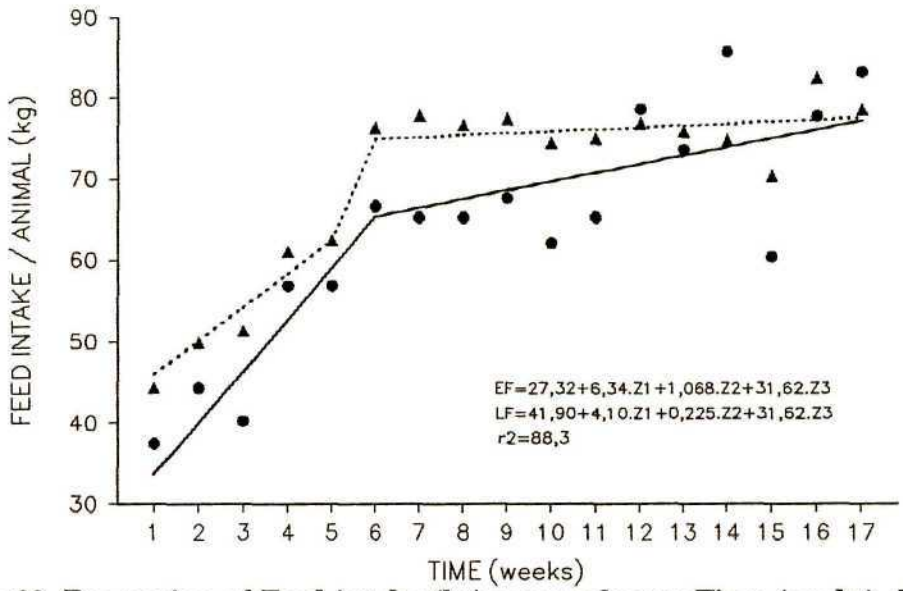

Figure 32. Regression of Feed intake (kg) per week over Time (weeks). The models fitted are broken stick. Data points = EF : ●, LF : ▲. Regression models = Early maturing : ———, Late maturing = · · · ·

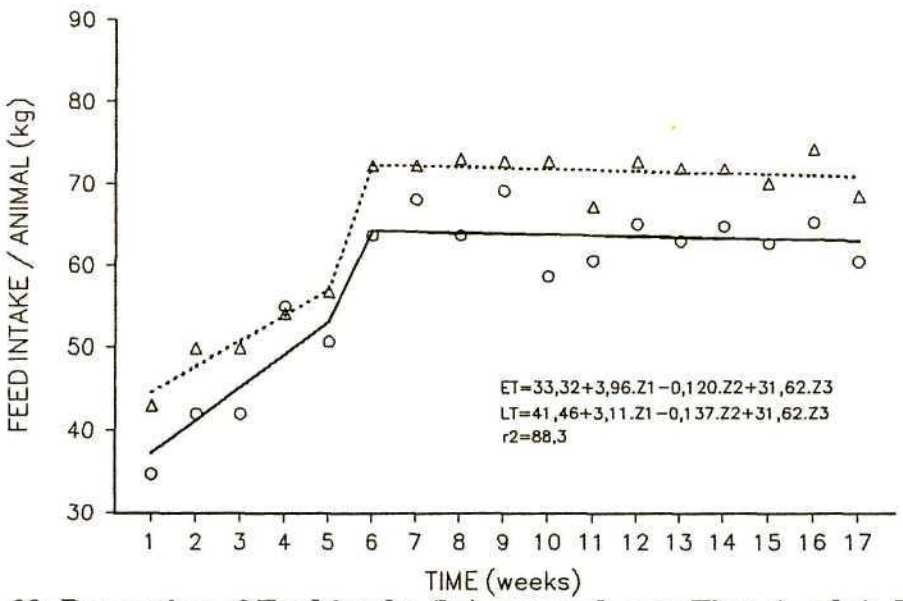

Figure 33. Regression of Feed intake (kg) per week over Time (weeks). The models fitted are broken stick. Data points = ET : ○, LT : △. Regression models = Early maturing : ———, Late maturing = · · · ·

Accepting either the quadratic or the broken stick model, feed intake increased to a point and then either decreased or followed a plateau. The liveweights followed a quadratic model over time. Thus the feed intakes as a proportion of liveweight, should increase to a point and then decrease as the feed intake either remains constant or decreases and the liveweight, continues to increase, at a decreasing rate.

Fitting a quadratic model to these data (Appendix 6), resulted in an R^2 = 42.9. Thus the model accounted for a low proportion of the variation. The model components are presented in Table 33. No significant differences were found between treatments for any components of the regression models. Again the models did not account for the peak feed intake to liveweight proportion i.e. the model cut off the top of the data. Thus it was decided to fit a broken stick model to the data.

Table 33. Regression equations, for the quadratic models of feed intake (kg) per week / metabolic weight ($W^{0.75}$) over time (weeks), showing significant differences between treatments, with respect to model components

TERM	TREATMENT			
	EF	LF	ET	LT
Constant	0.1473[a]	0.1498[a]	0.1727[a]	0.1813[a]
Linear	0.00947[a]	0.01049[a]	0.01101[a]	0.00895[a]
Quadratic	-0.000390[a]	-0.000186[a]	-0.000709[a]	-0.000585[a]

[a,b,c] Values in the same row with different superscripts are different $P < .05$.

The broken stick models (Appendix 7), accounted for a greater amount of variation (R^2 = 66.5). Again the break between the two linear models occurred at exactly the same point of time irrespective of treatment (Figure's 34 and 35).

Table 34. Regression equations, for the broken stick models of feed intake (kg) per week / metabolic weight ($W^{0.75}$) over time (weeks)

TERM	TREATMENT			
	EF	LF	ET	LT
Constant	0.4750[c]	0.5993[b]	0.6513[ab]	0.7162[a]
Linear Z1	0.0705[a]	0.0408[ab]	0.0384[ab]	0.0231[b]
Linear Z2	0.00035[a]	-0.01274[b]	-0.01815[b]	-0.02023[b]
Z3	0.3929[a]	0.3029[a]	0.3929[a]	0.3929[a]

[a,b,c] Values in the same row with different superscripts are different $P < .05$.

Components of the broken stick models (Table 34), differed significantly between treatments. The constant for the early maturing fat animals, was significantly lower than for the other treatments. This was due to the low feed intake at the beginning of their feedlot period, and their heavier liveweight relative to the thin treatments. The late maturing thin animals had the highest starting feed intake and lowest liveweight. The performance of the late maturing fat animals was non-significantly different to the early maturing thin animals.

The early maturing fat animals' feed intake to liveweight proportions increased the most over time. This was a combination of having had the highest rate of increase in feed intake and the lowest increase in liveweight. Similar trends to those that occurred for the feed intake models occurred for the remaining treatments. This was a combination of

non-significant differences in rate of increase in feed intake and higher increases in liveweight for the late maturing thin animals.

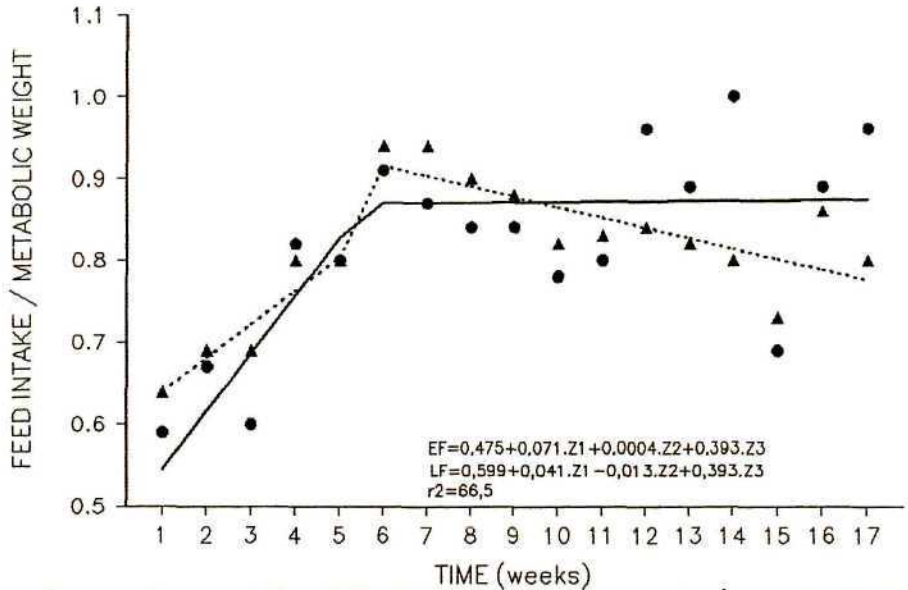

Figure 34. Regression models of Feed intake (kg) per week / Metabolic Weight ($W^{0.75}$) over Time (weeks). The models fitted are broken stick. Data points = EF : ●, LF : ▲. Regression models = Early maturing : ————, Late maturing = · · · ·

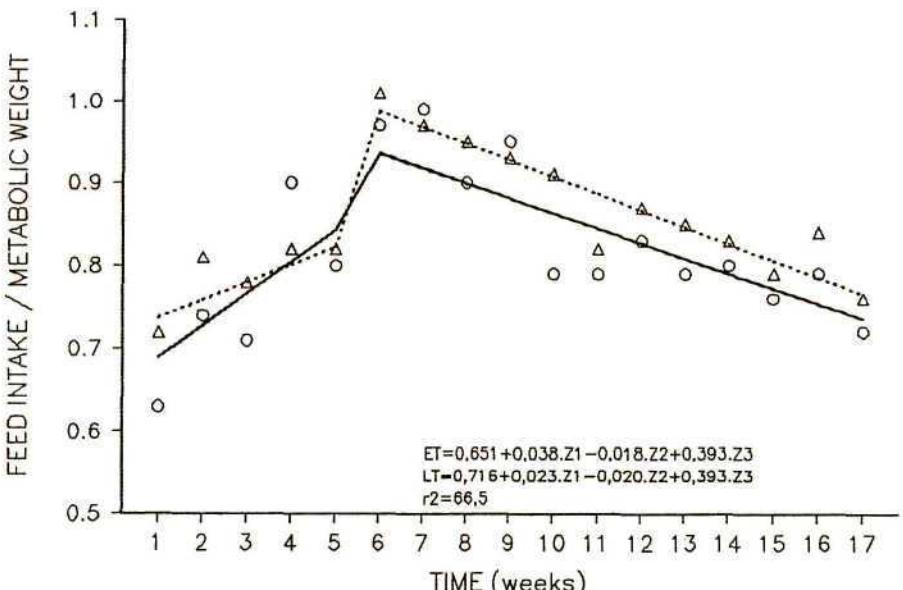

Figure 35. Regression models of Feed intake (kg) per week / Metabolic Weight ($W^{0.75}$) over Time (weeks). The models fitted are broken stick. Data points = ET : ○, LT : △. Regression models = Early maturing : ————, Late maturing = · · · ·

An important measure for beef producers is that of feed conversion efficiency. This is the amount of feed (kg) an animal must eat to gain a kilogram of liveweight. This is the simple breakdown of costs (feed) and returns (increased liveweights). In order to measure this the log of the cumulative feed intake was regressed on the log of the liveweight. This provided an indication of the change in feed intake for a set change in liveweight.

Table 34. Regression equations, for the models of the log of the cumulative feed intake (kg) per week over the log of the liveweight (kg)

TERM	TREATMENT			
	EF	LF	ET	LT
Constant	-37.18[a]	-36.48[a]	-25.02[b]	-26.83[b]
Linear	7.411[a]	7.147[a]	5.413[b]	5.652[b]

[a,b,c] Values in the same row with different superscripts are different $P < .05$.

The regression models (Appendix 8; Figure's 36 and 37), and their components (Table 35), illustrate that differences occurred between treatments. The fat treatments had a significantly higher feed conversion efficiency (the linear component of the model). This meant that the thin treatments, gained more weight, for an equal amount of feed. No significant differences were found between the fat treatments (EF vs LF), or the thin treatments (ET vs LT). The differences in the intakes (late maturing having greater intakes) were offset by the late maturing animals greater liveweight gains.

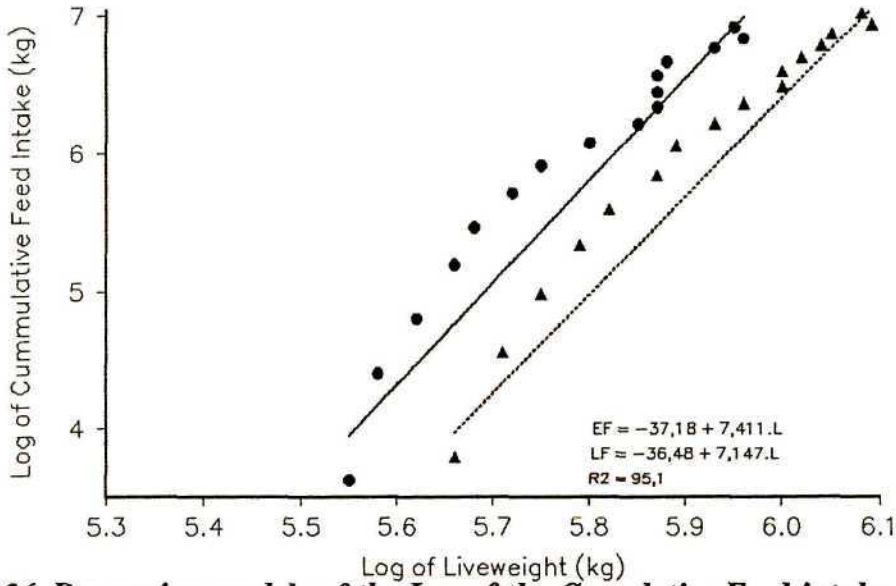

Figure 36. Regression models of the Log of the Cumulative Feed intake over the Log of the Liveweight. Data points = EF : ●, LF : ▲. Regression models = Early maturing : ———, Late maturing = · · · ·

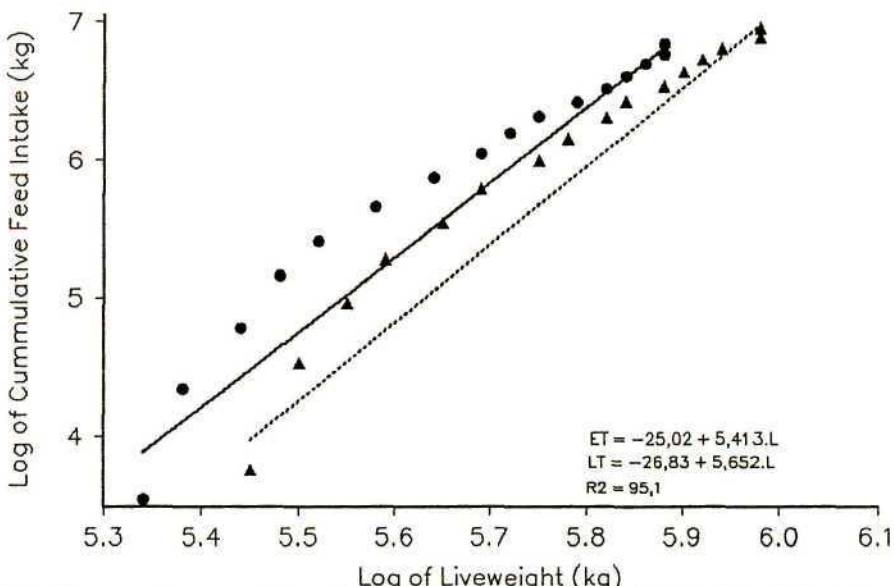

Figure 37. Regression models of the Log of the Cumulative Feed intake over the Log of the Liveweight (weeks). Data points = ET : ○, LT : △. Regression models = Early maturing : ———, Late maturing = · · · ·

4.3.4 NET ENERGY AVAILABLE FOR GROWTH (NEg) (MJ)

A ration was made up of a number of different ingredients. This is due to the necessity to provide a balanced ration and that no single ingredient can fulfil this role. As ingredients have different price tags, an indication of the efficiency of utilisation of the constituents of the ration is an important consideration in practical feedlot nutrition. From the data available, it was possible to examine net energy. The net energy for growth (NEg) was calculated by subtracting the maintenance requirements from the total energy intake (4.2.3.1).

The trend for the availability of net energy for growth was one of an increasing amount as feed intake increased. Then a decreasing amount as feed intake remained relatively constant, while the liveweight increased. Feed intake provided an estimate of the intake of net energy and liveweight provided an estimate for the maintenance requirements. The quadratic models fitted (data in Appendix 9), are shown in Figure's 38 and 39. The full breakdown of the models can be seen in Appendix 10.

Table 35. Regression equations, for the quadratic models, of the net energy available for growth (NEg) (MJ) per week over time (weeks)

TERM	TREATMENT			
	EF	LF	ET	LT
Constant	188.9[a]	222.3[a]	177.4[a]	226.7[a]
Linear	42.4[a]	54.3[a]	51.6[a]	51.4[a]
Quadratic	-1.298[a]	-2.286[a]	-2.358[a]	-2.276[a]

[a,b,c] Values in the same row with different superscripts are different $P < .05$.

The variation accounted for was less (R^2 = 74.0) than that achieved with the quadratic model fitted to the feed intake data (R^2 = 82.0). The added variation could be attributable to the maintenance requirements i.e a factor of the liveweight changes over time.

The quadratic model again failed to predict any significant differences between treatments (Table 35). This was despite the differences that were expected to occur between treatments. The thin treatments (ET and LT), were predicted to have higher feed intakes, thus high net energy intakes. Their liveweights were also significantly lighter, therefore their maintenance requirements should have been lower, with a resultant higher net energy availability for growth. With respect to maturity type, the later maturing animals were expected to have a higher net energy intake, to account for their higher maintenance requirements (being heavier animals, 1.2.1.7) and higher growth expectations.

As with the feed intake (4.3.3) quadratic model did not seem to fit the net energy available for gain trends (Figure's 38 and 39). Thus, broken stick models were fitted.

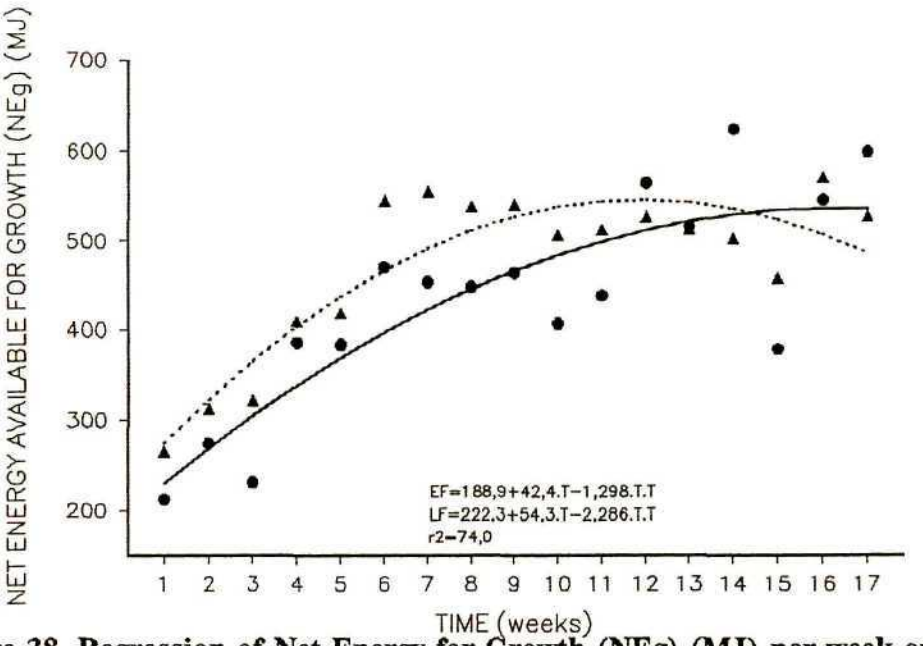

Figure 38. Regression of Net Energy for Growth (NEg) (MJ) per week over Time (weeks). The models fitted are quadratic. Data points = EF : ●, LF : ▲. Regression models = Early maturing : ————, Late maturing = · · · ·

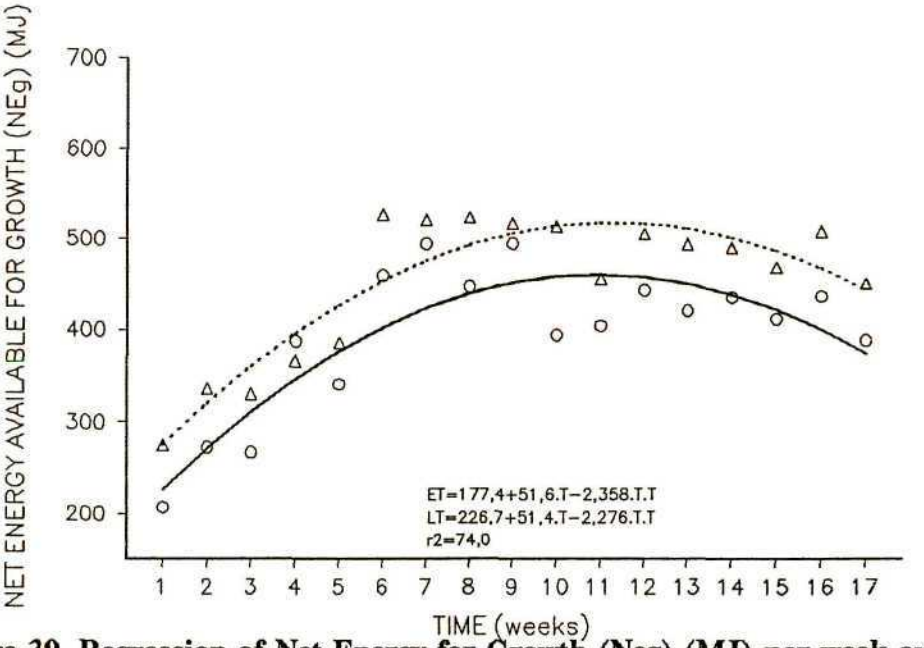

Figure 39. Regression of Net Energy for Growth (Neg) (MJ) per week over Time (weeks). The models fitted are quadratic. Data points = ET : ○, LT : △. Regression models = Early maturing : ————, Late maturing = · · · ·

The broken stick models, accounted for a greater amount of variation than the quadratic models (R^2 = 82.9). The full statistical breakdown of the model is in Appendix 11. The models appeared to account for the trends in the data (Figure's 40 and 41). That is the models did not underestimate the peak intake for growth. The break in the models again occurred at the same point in time, irrespective of maturity type or pre-feedlot plane of nutrition.

Table 36. Regression equations, for the broken stick models, of the net energy for growth (NEg) (MJ) per week over time (weeks)

TERM	TREATMENT			
	EF	LF	ET	LT
Constant	120.3^{b}	245.8^{a}	198.7^{ab}	265.4^{a}
Linear Z1	56.4^{a}	34.4^{ab}	33.2^{ab}	24.6^{b}
Linear Z2	7.40^{a}	-1.15^{b}	-4.72^{b}	-5.04^{b}
Z3	289.1^{a}	289.1^{a}	289.1^{a}	289.1^{a}

[a,b,c] Values in the same row with different superscripts are different $P < .05$.

The significant differences between treatments, for their components of the broken stick models (Table 36), was similar to that for feed intake (Table 32). The early maturing fat animals started with a significantly lower NEg, this was because of their low feed intake and high liveweight. The low liveweight of the thin treatments (ET and LT) increased their NEg, to the extent that the early maturing animals (ET) was no longer significantly lower than the late maturing fat animals (LF), and the late maturing thin animals (LT), started with the highest estimated intake of NEg. The first linear component (Z1), again showed the early maturing fat animals to have a significantly higher increase in intake of NEg, as compared to the late maturing thin animals. This was due to the early

maturing animals significant higher increase in feed intake and the late maturing animals significantly higher increase in liveweight and hence maintenance requirements. The second linear component (Z2), had a positive gradient for the early maturing fat treatment, due to a combination of their low liveweight gains and their increasing feed intakes (significantly different to the other treatments). The remaining treatments (LF, ET and LT) all had a negative Z2 component. This resulted from their low increase or slight decrease in feed intake, with their high increases in liveweight.

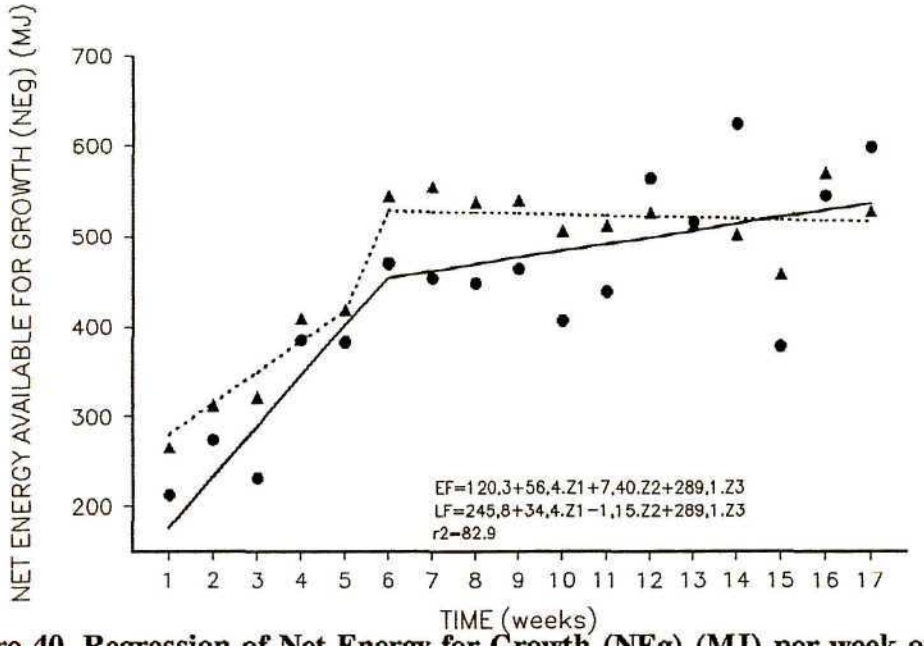

Figure 40. Regression of Net Energy for Growth (NEg) (MJ) per week over Time (weeks). The models fitted are broken stick. Data points = EF : ●, LF : ▲. Regression models = Early maturing : ————, Late maturing = · · · ·

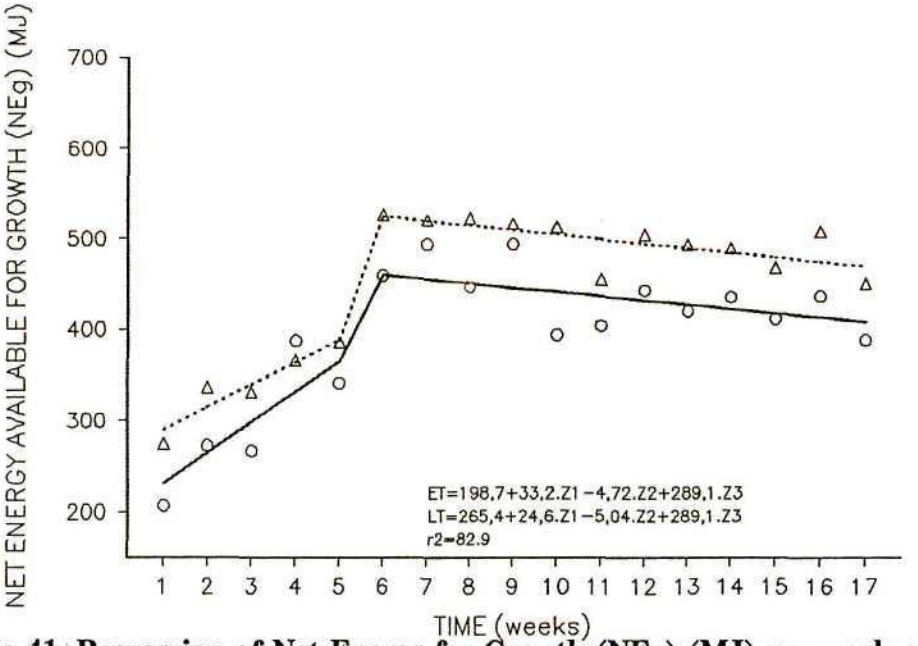

Figure 41. Regression of Net Energy for Growth (NEg) (MJ) per week over Time (weeks). The models fitted are broken stick. Data points = ET : ○, LT : △. Regression models = Early maturing : ————, Late maturing = · · · ·

The comparison of the NEg as a proportion of metabolic weight ($W^{0.75}$), between treatments, with quadratic models, provided a poor fit (Figure's 42 and 43). No significant differences were apparent between treatments model components (Table 37 and Appendix 10). The quadratic models did not follow the data trend. They underestimated the peak proportion and in some instances over estimated it towards the end of the feedlot period (EF and LF).

Table 37. Regression equations, for the quadratic models, of the net energy available for growth (NEg) (MJ) per week / metabolic weight ($W^{0.75}$) over time (weeks)

TERM	TREATMENT			
	EF	LF	ET	LT
Constant	3.249[a]	3.512[a]	3.808[a]	4.328[a]
Linear	0.451[a]	0.516[a]	0.546[a]	0.477[a]
Quadratic	-0.01688[a]	-0.02568[a]	-0.03054[a]	-0.02640[a]

[a,b,c] Values in the same row with different superscripts are different $P < .05$.

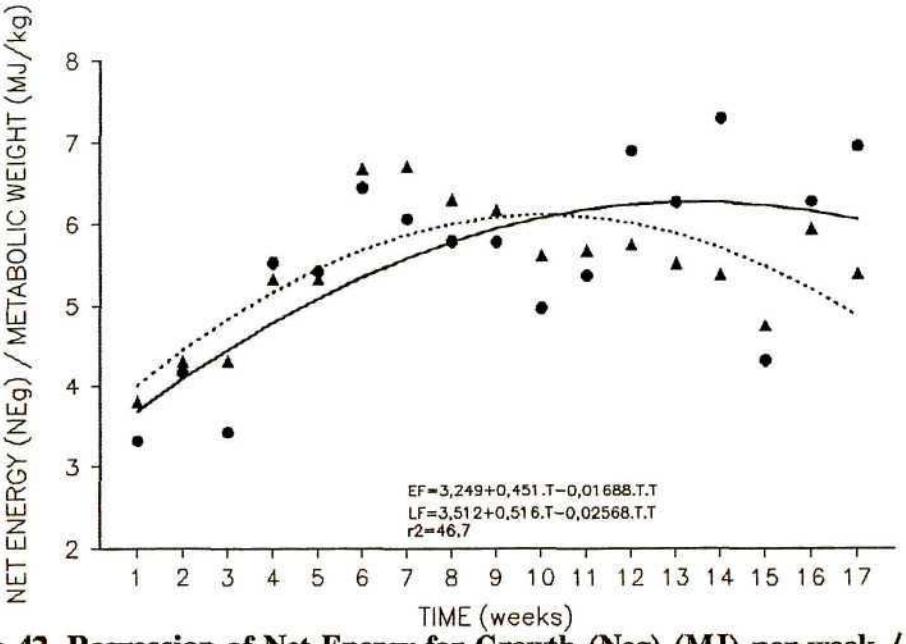

Figure 42. Regression of Net Energy for Growth (Neg) (MJ) per week / Metabolic Weight ($W^{0.75}$) over Time (weeks). The models fitted are quadratic. Data points = EF : ●, LF : ▲. Regression models = Early maturing : ———, Late maturing = · · ·

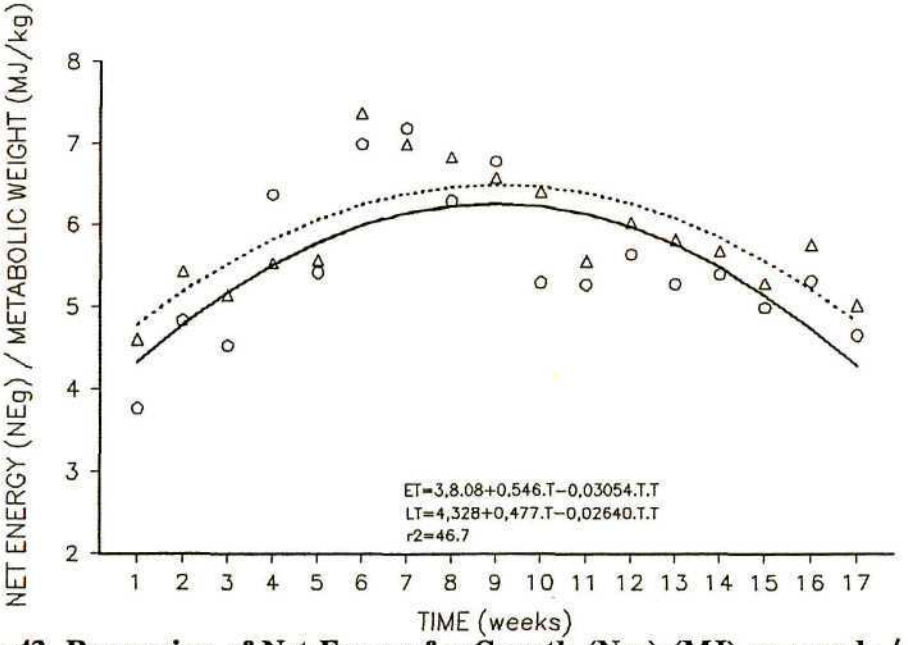

Figure 43. Regression of Net Energy for Growth (Neg) (MJ) per week / Metabolic Weight ($W^{0.75}$) over Time (weeks). The models fitted are quadratic. Data points = ET : ○, LT : △. Regression models = Early maturing : ———, Late maturing = · · ·

The broken stick models fitted the data with a greater accountancy of the variation (R^2 = 66.5). The models accounted for the peak intake of NEg, without over, or under, estimating, the intake of NEg, during the feedlot period (Figure's 44 and 45).

Table 38. Regression equations,for the broken stick models, of the net energy available for growth (NEg) (MJ) per week / metabolic weight ($W^{0.75}$) over time (weeks)

TERM	TREATMENT			
	EF	LF	ET	LT
Constant	2.273^{c}	3.459^{b}	3.955^{ab}	4.573^{a}
Linear Z1	0.673^{a}	0.389^{ab}	0.366^{ab}	0.220^{b}
Linear Z2	0.0033^{a}	-0.1214^{b}	-0.1731^{b}	-0.1929^{b}
Z3	3.747^{a}	3.747^{a}	3.747^{a}	3.747^{a}

^{a,b,c} Values in the same row with different superscripts are different $P < .05$.

Significant differences between treatments were found (Table 38 and Appendix 11). The high metabolic weights ($W^{0.75}$) of the fat treatments (EF and LF), reduced their NEg to metabolic weight proportions. Thus the early maturing fat animals, were significantly different to the early maturing thin animals. The late maturing fat and the early maturing thin remained non-significantly different, and the late maturing thin animals became significantly different from their respective fat animals. The increase and decrease (Z1 and Z2) followed the same trends as that for NEg. The difference is in the steepness of the slopes, which are due to the respective animals increase in liveweight.

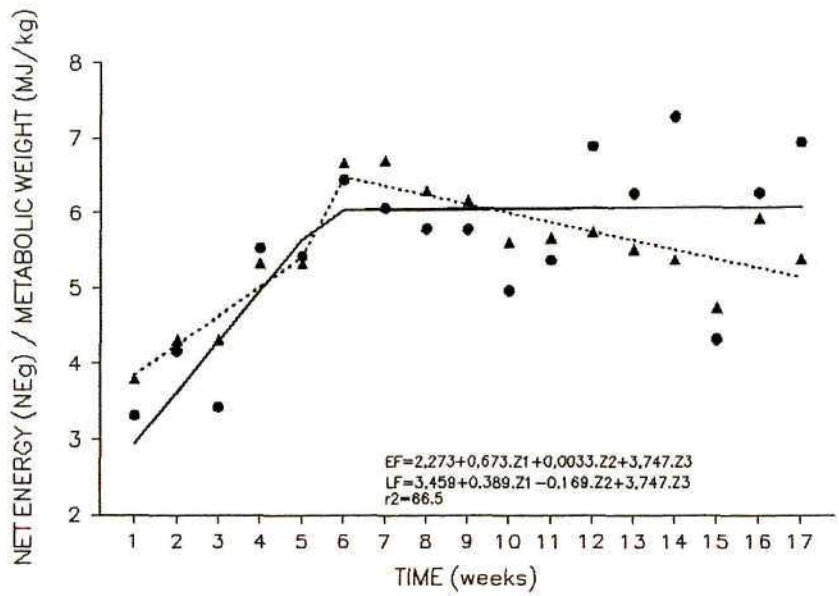

Figure 44. Regression of Net Energy for Growth (NEg) (MJ) per week / Metabolic Weight ($W^{0.75}$) over Time (weeks). The models fitted are broken stick. Data points = EF : ●, LF : ▲. Regression models = Early maturing : ————, Late maturing = · · ·

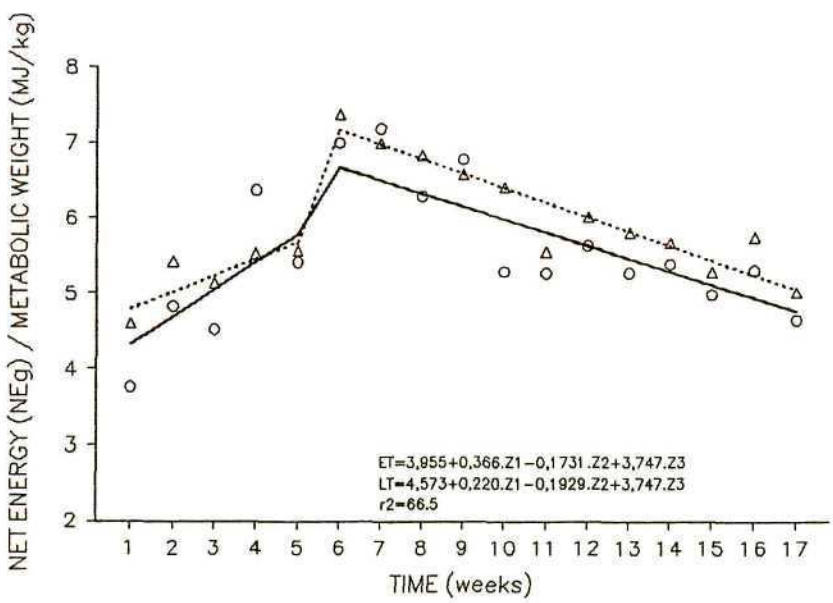

Figure 45. Regression of Net Energy for Growth (Neg) (MJ) per week / Metabolic Weight ($W^{0.75}$) over Time (weeks). The models fitted are broken stick. Data points = ET : ○, LT : △. Regression models = Early maturing : ————, Late maturing = · · ·

Having established that the treatments have differing NEg availabilities and differing growth rates, the NEg conversion efficiency is a good comparative measure between treatments (Figure's 46 and 47). Significant differences were found (Table 39 and Appendix 12), between the pre-feedlot treatments (EF and LF vs ET and LT). The thin treatments conversion of NEg was significantly more efficient than that achieved by the fat treatments.

Table 39. Regression equations, for the models of the log of the cumulative net energy for growth (NEg) (MJ) per week over the log of the liveweight (kg), showing significant differences between treatments, with respect to model components

TERM	TREATMENT			
	EF	LF	ET	LT
Constant	-37.80[a]	-36.46[a]	-24.37[b]	-25.78[b]
Linear	7.838[a]	7.460[a]	5.631[b]	5.802[b]

[a,b,c] Values in the same row with different superscripts are different $P < .05$.

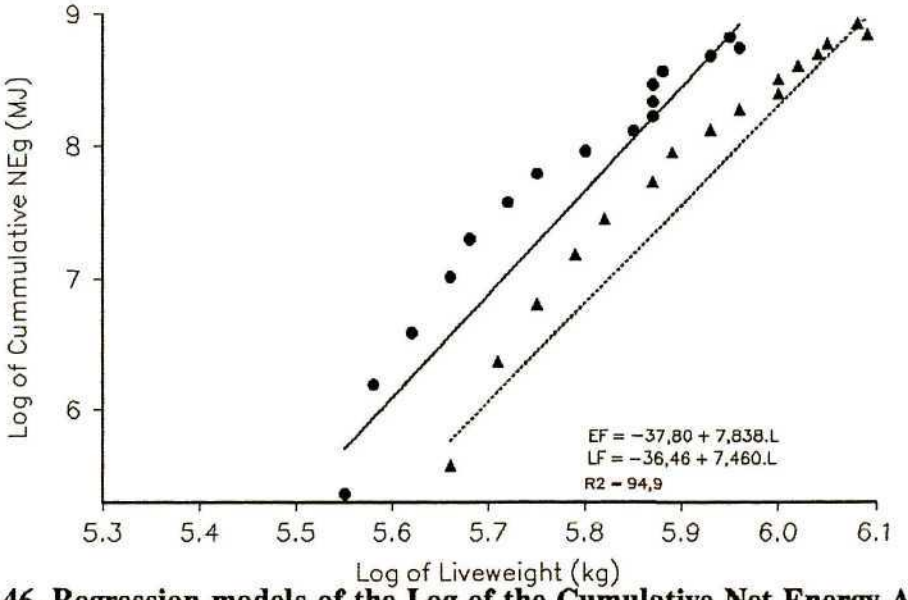

Figure 46. Regression models of the Log of the Cumulative Net Energy Available for Growth (NEg) over the Log of the Liveweight. Data points = EF : ●, LF : ▲. Regression models = Early maturing : ————, Late maturing = · · · ·

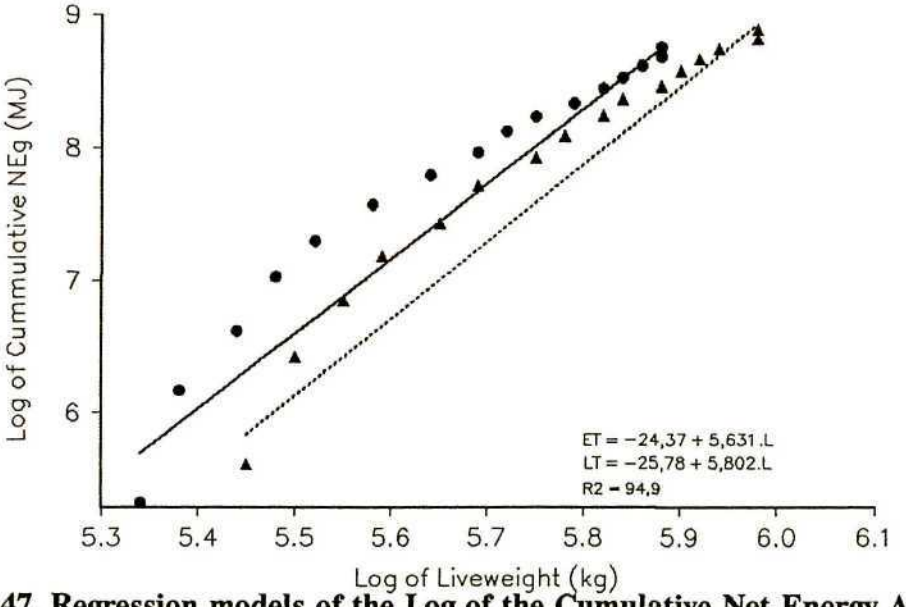

Figure 47. Regression models of the Log of the Cumulative Net Energy Available for Growth (NEg) over the Log of the Liveweight (weeks). Data points = ET : ○, LT : △. Regression models = Early maturing : ————, Late maturing = · · · ·

4.4 DISCUSSION

Compensating animals have typically taken a longer period of time to attain a set condition or liveweight as compared to controls (Meyer *et al.*, 1965; Allden, 1970; Fox *et al.*, 1972; Coleman and Evans, 1986; Phillips *et al.*, 1991; Patterson *et al.*, 1995). This was attributed by Allden (1970), to an animals capacity to grow not being controlled by chronological age. Thus the compensation achieved is on the lines of G3 rather than G2 as described in 1.2.1.6. In order to achieve these targets a higher growth rate must be achieved for the compensating animals (Fox *et al.*, 1972; Patterson *et al.*, 1995). The results achieved in the trial described (4.3.2) follow the general trend achieved by other researchers. Comparison of maturity type growth rates, showed that, the later maturing animals, have a higher growth rates, similar to those achieved in 4.3.2 (Ferrell *et al.*, 1978).

Comparisons between compensating versus non-compensating animals with respect to feed intake, show that there exists little difference in their levels of feed intake (Allden, 1970; Ryan *et al.*, 1993). This lack of difference however only persisted for the initial twelve weeks of recovery, from then on the compensating animals consumed more. This is similar to what was found in the trial, except that when comparisons were based on the broken stick models differences were apparent although slight. On a maturity type basis, the feed intakes for later maturing animals was higher (Ferrell *et al.*, 1978).

A more accurate comparison between treatments with respect to differing liveweights is that of food intake / metabolic weight $^{0.75}$. as in the results of this trial, compensating animals, by eating the same amount as non-compensating, will have significantly higher proportions of feed intakes to metabolical weights during the compensating period (Patterson *et al.*, 1995). Dry matter or ME intake did not differ between breed groups when expressed on metabolic body size ($W^{0.75}$) basis. Weight gain so expressed slightly favoured the small-type steers even though weight gains of the small-type steers apparently contained a higher percentage of fat and lower percentage of protein and water (Farewell *et al.*, 1978).

The maturity type feed efficiencies, despite differences in growth rates, are similar (Ferrell *et al.*, 1978). However, when comparing compensating animals, which are slaughtered at a common end point, compensatory gain is reflected in improved efficiency (Meyer *et al.*, 1965; Fox *et al.*, 1972; Coleman and Evans, 1986; Phillips *et al.*, 1991; Patterson *et al.*, 1995). These are reflected in the trial results. Fox *et al.* (1972), showed that as with the results from this trial, the improved efficiency of feed utilisation by compensating animals, could be extended to that of improved efficiency of NEg utilisation.

The point of interest, generated by the results in this chapter, is the use of the broken stick model to generate prediction equations. The use of a broken stick model has to be justified, that is there has to be a biological reason for the animals to suddenly change their pattern of behaviour (i.e. their increasing feed intake). The reason appears from the analysis to be a form of restriction, occurring at the point of inflection. Similar patterns of feed intake have been published (Hicks *et al.*, 1990a and b). Further evidence for the use of the broken stick model or an alternative to the general quadratic model generally fitted is found in the papers of Hicks *et al.* (1990a and b). Not only did the peak feed intake occur at the same time irrespective of season but also irrespective of maturity type and sex. This should not be possible as all these respective groups have differing nutrient requirements over time, as well as liveweights and growth rates. Examination of more appropriate models to fit the data and the possible reason for the restriction on feed intake is necessary.

CHAPTER FIVE

SHOULDER HEIGHT AND EYE-MUSCLE DIAMETER

5.1 INTRODUCTION

The height of an animal is closely related to the growth of it's long bones. From the literature reviewed in Chapter One it can be concluded that only under *severe restriction* (1.2.1.5), when actual weight loss occurs (1.2.1.5.1), will bone growth be inhibited. As the restriction imposed on the trial animals can be classified only as *restriction* (1.2.1.5), no differences in height or rate of growth should have been observed between the fat and thin groups. Growth differences due to maturity type could however be present due to the early maturing animal being physiologically more mature, and therefore exhibiting lower bone growth.

The eye-muscle (*longissimus dorsi*) is situated in the loin area of the carcass. This area has a high impetus of growth (1.2.1.4.2), and is therefore highly susceptible to nutritional restriction (1.2.1.5.2). Berg and Butterfield (1976) allocated this muscle group to the high-average impetus muscles with a growth rate nearly equal to that of total muscle. Thus the growth of the eye-muscle could well mirror the growth of muscle overall within the carcass. The measurement of this muscle is complicated by the deposition of intermuscular, fat making protein deposition difficult to measure.

5.2 MATERIALS AND METHODS

5.2.1 HEIGHT

The height over the shoulders of individual steers was measured fortnightly. Three boards, with height markings on, were attached towards the front end of the crush. Two of these boards were placed 30 cm apart on the near side of the steer, with the remaining board located between the previous two, but on the far side of the steer. By eye-balling across the shoulders of the steer, and lining up the equivalent measurement on a near side, and the far side board, the height of the animal could be determined.

5.2.2 EYE-MUSCLE DIAMETER

Half of the animals in each pen were randomly selected to have their eye-muscle (*longissimus dorsi*) diameters (cm's), determined every two weeks. The eye-muscle diameter was measured using a Scanner 200 (Piemedical, Holland). The scanner was used in B- mode with the eye-muscle diameter being measured with a rectal probe at 5 mHz with a linear array. The measurements were taken between the twelfth and thirteenth rib on the left handside of the steer. The site was prepared by clipping excess hair away before applying ultrasound gel. The probe was then placed on the surface of the skin, at 90° to the midline and the diameter read 50 mm from the midline.

5.2.3 STATISTICAL METHODS

The GENSTAT V statistical programme (Lawes Agricultural Trust, Rothamstead) was used for all statistical analyses. Regression models were fitted to the data, where relevant. A selection of models was used; linear, quadratic, cubic, gomperitz, linear exponential and broken stick. The model that provided the best statistical fit was chosen. Before a model was chosen however its use had to be justified, that is, its parameters must meet with logical understanding in line with existing principles in Animal Science. Statistical differences between means were determined from analysis of variance tables with the use of the *Students' t test* (Steel and Torrie, 1980).

Within the regression equations -

T = Time in weeks, from the start of measuring the data in question.

T.T = Quadratic term (Time squared), as above.

5.3 RESULTS

5.3.1 HEIGHT

From 1.2.1.6.6, later maturing animals were observed to be taller than earlier maturing animals at an equal chronological age. This was also found to be true in this trial (Table 40). The later maturing animals were significantly taller than the earlier maturing animals, at the start of the pre-feedlot period (LF vs EF). An anomaly exists in that the late maturing, thin animals (LT) were significantly shorter than the late maturing, fat animals (LF). This was not observed between the early maturing treatments (EF vs ET), the thin animals were shorter, but not significantly so. The thin animals still followed the theory of maturity type, as the later maturing animals were taller than the earlier maturing animals (LT vs ET).

The difference in height could have been created by the effect of the pre-feedlot periods on the long bone growth, that is, the plane of nutrition followed by the thin animals reduced their growth in terms of height. This is unexpected, as the restriction was not expected to be severe enough to affect bone growth, as compared to a previous trial reported (1.2.1.5.1).

The height gained during the feedlot period followed a predictable trend, with the thin groups growing more than their respective fat groups, and the later maturing growing more than the earlier maturing groups. However, the rate of growth was significantly different between the LT group and the other three. If the reason for the difference in height between the late maturing treatments, was due to reduction in growth, caused by the pre-feedlot nutritional plane, then the LT animals may have exhibited a degree of compensatory growth. In spit of this significantly higher growth rate, the LT group was unable to catch up with the LF group and finished significantly shorter, but with a smaller difference than at the start of the feedlot period.

Table 40. Heights of steers over the feedlot period

TERM	TREATMENT			
	EF	LF	ET	LT
Initial Height (cm)	109.17^{bc}	117.21^{a}	107.96^{c}	110.37^{b}
Final Height (cm)	115.50^{c}	125.37^{a}	116.91^{c}	121.92^{b}
Height Gain (cm)	6.33^{c}	8.17^{b}	8.96^{b}	11.54^{a}
ADG (cm/day)	0.0851^{b}	0.0903^{b}	0.1011^{b}	0.1228^{a}

^{a,b,c} Values in the same row with different superscripts are different $P < .05$.

Illustration of the growth in height of the groups over time can be seen in Figure's 48 and 49. The breakdown of the regression models and the significant differences between components within the models is presented in Table 41. The full statistical breakdown of the models can be seen in Appendix 13, along with the means and CV % for the treatments.

Table 41. Regression equations for height (cm) over time (weeks)

TERM	TREATMENT			
	EF	LF	ET	LT
Constant	108.973[b]	117.172[a]	106.292[c]	108.453[b]
Linear	0.542[b]	0.332[b]	1.712[a]	2.023[a]
Quadratic	0.0886[a]	0.1112[a]	-0.0326[b]	-0.0447[b]

[a,b,c] Values in the same row with different superscripts are different $P < .05$.

Having shown that the pre-feedlot period could have restricted growth in terms of height, for the thin treatments (ET and LT), a degree of compensatory growth may have been expected. Thus, their rate of growth over time (the linear component of the models), should be higher for the thin treatments. This in fact occurred with the thin treatments (ET and LT), starting with a significantly faster growth rate than that of the fat treatments (EF and LF). The decrease in growth, as the tissue reaches a higher degree of maturity, should result in a negative quadratic component for all treatments. However, the quadratic component shows that the fat treatments rate of growth was increasing over time, whereas that of the thin treatments was decreasing over time.

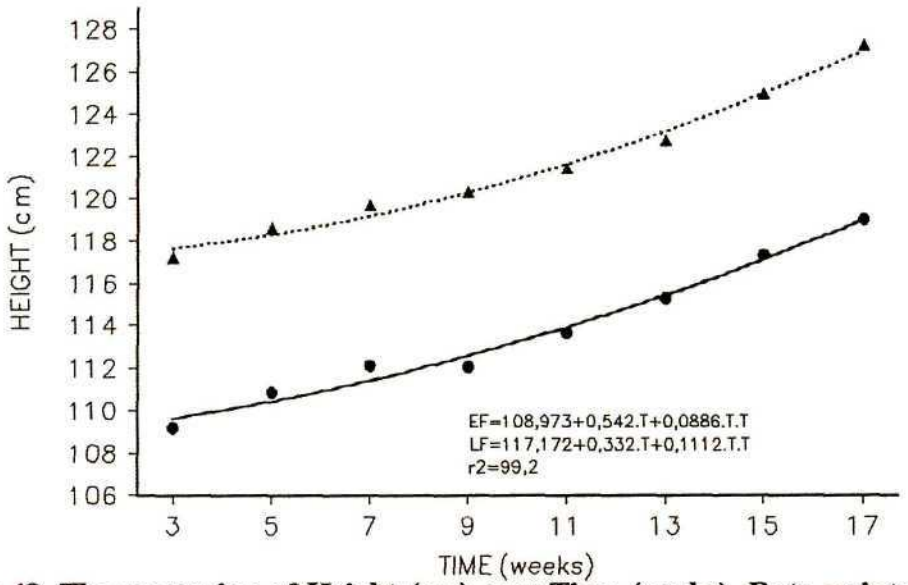

Figure 48. The regression of Height (cm) over Time (weeks). Data points = EF : ●, LF : ▲. Regression models = Early maturing : ———, Late maturing = · · · ·

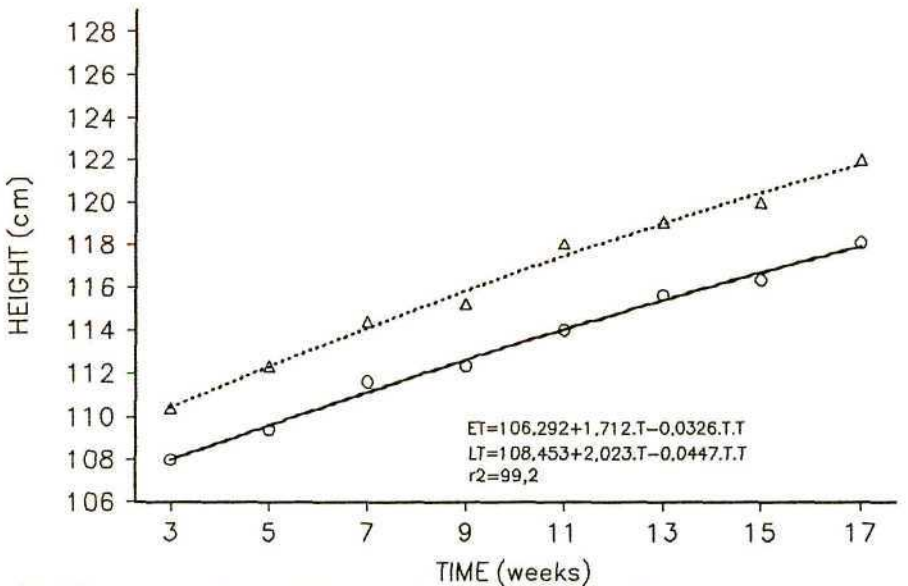

Figure 49. The regression of Height (cm) over Time (weeks). Data points = ET : ○, LT : △. Regression models = Early maturing : ———, Late maturing = · · · ·

5.3.2 EYE-MUSCLE DIAMETER

The eye-muscle was chosen due to its high impetus for growth and thus its susceptibility to nutritional restriction (5.1). If the pre-feedlot period affected the growth rate of muscle tissue, then it should be observable from comparative measurements of the eye-muscle. The fat treatments (EF and LF), should have a wider eye-muscle diameter than their respective thin treatments (ET and LT). At the start of the feedlot period (Table 42), the thin groups were significantly smaller, as regards their eye-muscle diameters, than their respective fat groups. The differences between maturity types is confounded by later maturing animals being larger, and earlier maturing animals being more mature at an equal chronological age. The lack of significant differences between maturity types is therefore not surprising.

Compensatory growth, or a growth rate greater than that achieved by the controls, should result in the restricted treatments (ET and LT) making up differences on those animals that were not as restricted (EF and LF). The differences were non-significant between the EF and ET groups by the end of the feedlot period due to a significantly greater gain in diameter by the ET group. The difference between the LF and LT group remained significant, with both groups attaining non-significantly different increases in diameter. The large variation between measurements (Appendix 14), resulted in there being no significant differences between treatments, with respect to rate of growth, even though large differences existed.

Table 42. Changes in Eye-muscle diameter of steers, comparison between treatments over the feedlot period

TERM	TREATMENT			
	EF	LF	ET	LT
Initial Diameter (cm)	8.47[a]	7.89[ab]	6.52[c]	6.74[bc]
Final Diameter (cm)	13.01[b]	14.91[a]	13.23[b]	13.15[b]
Diameter Gain (cm)	4.54[b]	7.02[a]	6.71[a]	6.41[a]
ADG (cm/day)	0.0562[a]	0.0735[a]	0.0723[a]	0.0654[a]

[a,b,c] Values in the same row with different superscripts are different $P < .05$.

Figure's 50 and 51, represent the changes in eye-muscle diameter over time, with their respective regression models. Appendix 14 includes the full statistical breakdown of the regression models. The breakdown of the regression models and the significant differences between components within the models is presented in Table ??.

Table 43. Regression equations for eye-muscle diameter (cm) over time (weeks)

TERM	TREATMENT			
	EF	LF	ET	LT
Constant	1.7271[a]	1.6534[ab]	1.4675[c]	1.5710[b]
Linear	0.1176[b]	0.1404[a]	0.1476[a]	0.1345[ab]
Quadratic	-0.00382[a]	-0.00160[a]	-0.00551[a]	-0.00877[a]

[a,b,c] Values in the same row with different superscripts are different $P < .05$.

Growth in the eye-muscle diameter will be due to deposition of protein as well as inter-muscular fat (5.1). The growth rate of the eye-muscle will also depend on the degree of maturity of the animals in question. Earlier maturing animals are more mature at an equal chronological age than later maturing animals. This was illustrated by the EF treatment (Table 43), having the lowest rate of growth (or linear component within the models). All three other treatments were non-significantly different in their rate of growth, even though their maturity types and previous history of growth differed. This could have well been due to the fact that this growth rate was the maximum attainable by the animals under the circumstances.

The normal pattern of growth for a muscle (1.2.1.4.2), is a reduction over time as its mature size is approached. Thus, it was expected that a quadratic term, would need to be included in the model's as a significant term. It was however found that the rate of decrease in growth was non-significantly different between treatments.

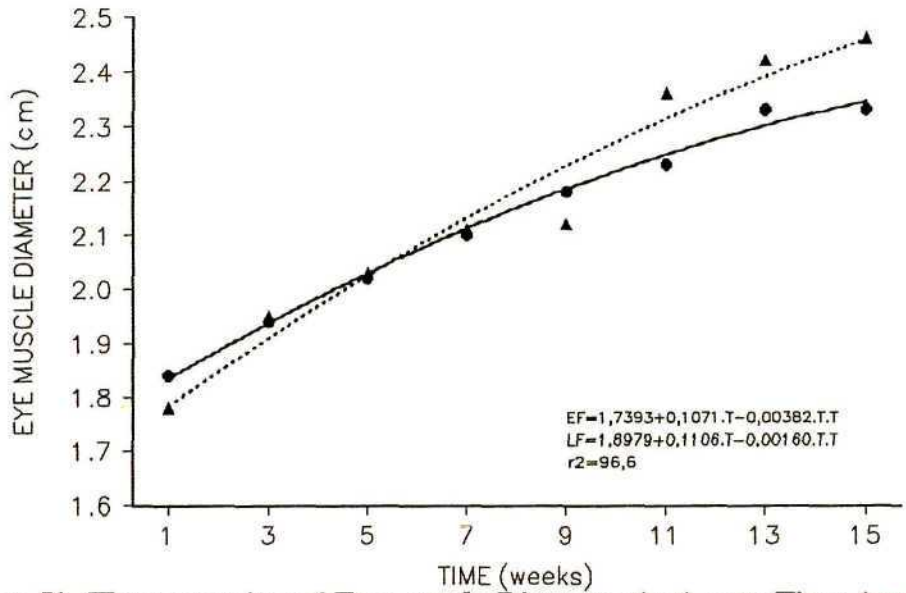

Figure 50. The regression of Eye-muscle Diameter (cm) over Time (weeks). Data points = EF : ●, LF : ▲. Regression models = Early maturing : ————, Late maturing = · · · ·

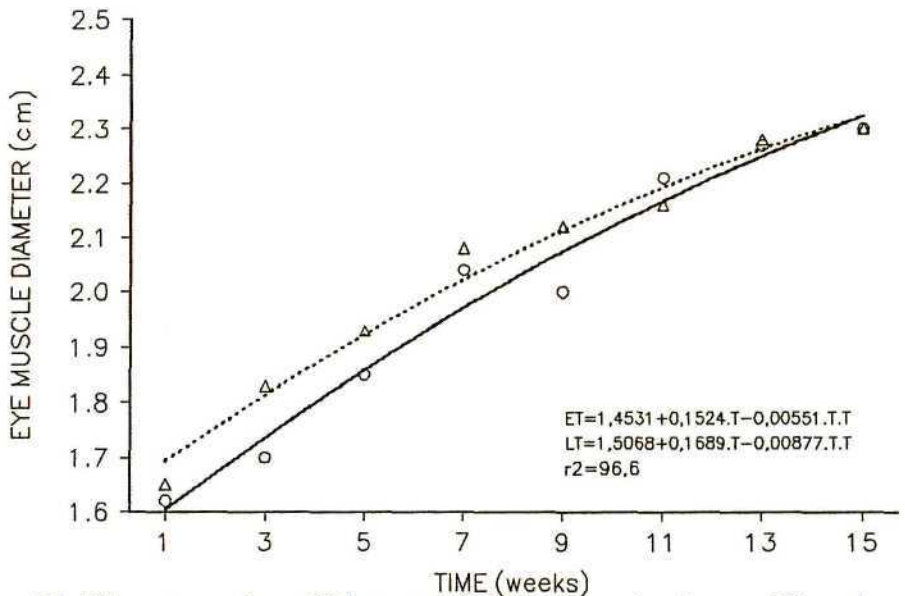

Figure 51. The regression of Eye-muscle Diameter (cm) over Time (weeks). Data points = ET : ○, LT : △. Regression models = Early maturing : ————, Late maturing = · · · ·

5.4 DISCUSSION

Differences between breeds with respect to size are due partially to differences in size of the skeleton (Owens *et al.*, 1993). Hence, late maturing animals should be taller at an equal chronological age than early maturing animals and indeed is the case in this study (Figure's 48 and 49).

As predicted from the literature covered (1.2.1.5.1), the planes of nutrition imposed during the pre-feedlot period (2.2.3), were not expected to affect bone growth. However, the fact that the LT treatment started the feedlot period significantly shorter than the LF treatment indicates that inhibition may have occurred.

The question arises that if the pre-feedlot period affected the growth with respect to height of the LF treatment, why did it not equally affect the ET treatment. Possibly, this could have been due to the level of maturity of bone growth between the maturity types. Bone development of the early maturing animals was at a higher level of maturity and therefore less susceptible to nutritional restriction (1.2.1.5.1).

The rate of growth in height during the feedlot period by the thin treatments was significantly higher than the fat treatments, indicating a lower level of maturity in the thin treatments. Even though literature (1.2.1.6.1) points to limited compensatory growth existing within bone tissue it seems to have occurred in this case.

It was expected that the fat animals would follow a constant or slightly decreasing growth in height, under the assumption that the animals are growing in height, in an unrestricted manner, on entering the feedlot. This was however not the pattern of growth seen. An explanation for the increase in their rate of growth over time was not apparent. Hammond's (1960) explanation of growth gradients within the body would place the shoulders in an early maturing region (1.2.1.1). This means that as a site of growth, it would not have an increasing growth rate as the animal matured. However, at the end of the feedlot period the rate of growth was still higher for the thin animals and so the

possibility of the fat and thin animals meeting and following a similar growth plane was not observed.

The LT treatment, within the feedlot period was also unable to exhibit *complete compensation* if, in fact, they were exhibiting compensatory growth. Thus, a longer period of growth was required for the animals to reach the same height.

Since the eye-muscle diameters were less for the thin animals than for the respective fat animals, it is clear that growth was restricted during the pre-feedlot period. The restriction of growth would have resulted in a combination of a decrease in protein deposition and intermuscular fat deposition. Due to their lower degree of maturity, protein deposition during the feedlot period would have been at a maximum rate for the thin animals. As the degree of maturity increased the amount of deposition attributable to intermuscular fat increased, while the overall rate decreased. This is illustrated by the EF treatment growing at a slower rate during the feedlot period, indicating a higher level of maturity and therefore decreased muscle deposition and increased fat deposition. The ET treatment gained a significantly greater amount (at a non-significantly different rate) in comparison to the EF treatment. This indicates that the ET treatment showed compensatory growth by achieving an amount of growth greater than that of animals of equal maturity type and chronological age.

There was no significant difference between the amount or rate of growth with respect to eye-muscle diameter for the LF and LT treatments. Being of a lower degree of maturity than the earlier maturing animals the rate of protein deposition is at a maximum in the region measured, limiting the LT treatment's ability to exhibit compensatory growth over the LF treatment.

Published results have concentrated on the change in eye-muscle area, which has an obvious relation to that of eye-muscle diameter. The reason why eye-muscle diameter was not measured in this case was that the length of the probe was insufficient to cover the length of the eye-muscle.

Similar results have however been recorded when measuring eye-muscle area, as for those recorded for the eye-muscle diameter. The rate of change was found to be non-significantly different across frame size over time (Duello *et al.*, 1990). The rate of change was found by Hamlin *et al.* (1995), to be a function of age or weight. The rate at which muscle grows is dependent on the physiological maturing rate of the particular animal and consequently the muscle area is affected by the age or weight of the animal. The degree of maturity is therefore an important indication of the rate of growth within the eye-muscle. This is further supported by a negative quadratic term providing the best account of variation (Duello *et al.*, 1990 and Hamlin *et al.*, 1995). The eye-muscle's growth is therefore expected to decrease as the animal matures.

CHAPTER SIX

CHANGES IN BODY CONDITION AND CARCASS MEASUREMENTS

6.1 INTRODUCTION

Comparison of performances within the feedlot period are incomplete without suitable comparisons of the product produced. In the case of steers this is the carcass. A trial was designed to impose differing pre-feedlot nutritional regimes, and then compare all animals on the same plane of nutrition. The animals were destined for slaughter at a set condition. The aim was to mimic the practical situation. No producer will intentionally send to slaughter animals that are too fat or too thin. The set condition of slaughter was that dictated by the market, i.e. the aim was to achieve a carcass of an A2+ to an A3 (roughly a 20% fat content), as these carcasses received the premium pricing at the time of the trial. By keeping all animals at the same degree of finish, a true comparison of ADG, FCE and length of time in the feedlot was possible.

6.2 MATERIALS AND METHODS

Some of the measurements, for example condition score, that were taken were subjective. In order to reduce any bias two condition scorers and the same carcass grader was used throughout the trial. The objective of these measurements was to show that the animals were slaughtered at an equal degree of finish.

6.2.1 CONDITION

Animals were condition scored using a combination of visual evaluation and physical touch. The animals were rated according to a scale where a 1 was considered emaciated and a 5 over fat. The condition scoring was performed on all animals, every two weeks. The condition score was the basis of selection for slaughter, as all animals were slaughtered at a set condition score (4). Towards the end of the feedlot period, condition scoring on a weekly basis became necessary as more animals became ready for slaughter.

See Plates 5 and 6 for examples of the condition of the early and late maturing animals at the time of slaughter.

6.2.2 CARCASS MEASUREMENTS

Steers considered ready for slaughter were transported to the abattoir after determination of their empty body weight. The animals were slaughtered within twenty four hours of arrival at the abattoir. The carcasses were then measured for the following factors after being kept in a cold room for twenty four hours at four degrees celsius. Plates 7 and 8 illustrate the points on the carcass where measurements were taken.

- Cold carcass mass (kg's).

- Dressing percentage = (cold carcass mass / empty body mass) X 100.

- Eye muscle width's and length's (cm), were taken on the left carcass half. The measurement was at the widest point of the eye muscle at the 9⅓ lumbar vertebrae, (see plate 8; 4 = cut at the 9⅓ lumbar vertebrae).

- Description of the carcass with respect to subcutaneous fat coverage, Table 44. This was determined for the overall carcass as well as for the hindquarters, loin and forequarters, (see plate 7; 1 = hindquarter, 2 = loin, 3 = forequarter).

Plate 5. Early maturing

Plate 6. Late maturing

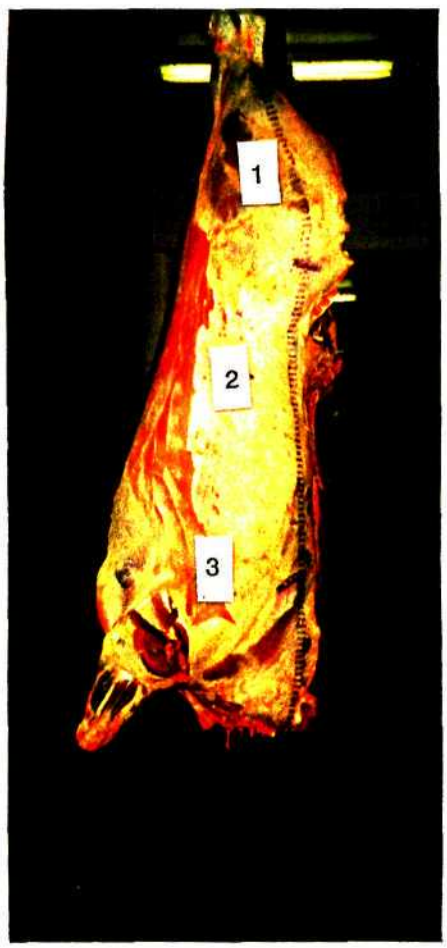

Plate 7. Subcutaneous fat coverage

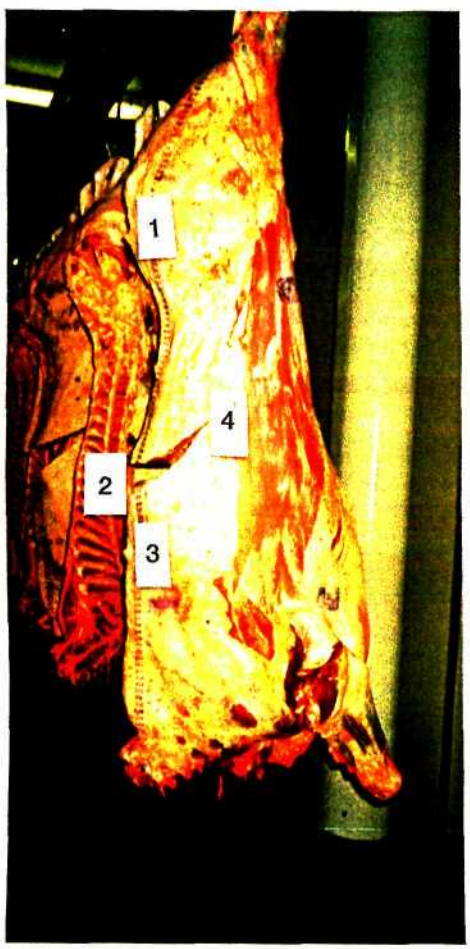

Plate 8. Subcutaneous fat depths

Table 44. Fatness classification of bovine carcasses.

DESCRIPTION OF FATNESS	FATNESS CLASS	THICKNESS OF SUBCUTANEOUS FAT LAYER
No Fat	0	Nil
Very Lean	1	Less than one
Lean	2	Between one and three
Medium	3	Between three and five
Fat	4	Between five and seven
Slightly over-fat	5	Between seven and ten
Excessively over-fat	6	More than ten

- Subcutaneous fat depths (mm) were measured at three sites : 2.5 cm lateral to the 13⅓ lumbar vertebrae (*1st*). 5.0 cm lateral to the 9⅓ lumbar vertebrae (*2nd*). 2.5 cm lateral to between the third and fourth lumbar vertebrae (*3rd*).

- Internal Fat, that is the fat deposited within the channel area and around the kidneys. This was subjectively scored on a scale of 1 to 6, with 1 showing very little deposition and 6 showing excessive deposition.

- The carcasses were classified on conformation (Agricultural Production Standards Act, 1990), Table 45.

Table 45. Conformation classification of bovine carcasses.

DESCRIPTION OF CONFORMATION	CONFORMATION CLASS	POINTS FOR CLASS
Very round	5	15
	5-	13
	4+	11
Round	4	9
	4-	7
	3+	5
Medium	3	3
Flat	2	3
Very flat	1	3

6.2.3 STATISTICAL METHODS

The GENSTAT V statistical programme (Lawes Agricultural Trust, Rothamstead) was used for all statistical analysis. Statistical differences between means were determined from analysis of variance tables with the use of the *Students' t test* (Steel and Torrie, 1980).

6.3 RESULTS

The animals started the feedlot period at different condition scores (2.3). The fat animals had a higher condition score than the thin animals. The animals were then slaughtered at a condition score of four. As the condition score was based on the visual coverage of fat over the animal, the rate of change in condition score was an indication of the rate of deposition of subcutaneous fat. Differences in fat deposition should have occurred

between treatments. The fat treatments should have deposited at a slower rate to the thin treatments (due to compensatory growth), and the earlier maturing animals at a faster rate to the later maturing (due to their maturing patterns). This was found to be partly true (Table 46). The thin animals deposited fat at a significantly faster rate than the fat animals. However, there was no significant difference with respect to maturity type within each pre-feedlot treatment (EF vs LF and ET vs LT).

As concluded by Wilson and Osbourn (1960) the compensating animals extended their growth period (Table 46). The thin early maturing animals required a significantly longer period of time to finish than the fat early maturing animals. There was also a large difference between the late maturing animals (LF vs LT). However, they were non-significantly different. With respect to maturity type, the earlier maturing fat animals being more mature, deposited more fat and finished more quickly than the later maturing animals.

Slaughter and carcass masses showed the later maturing animals to be heavier than the earlier maturing animals (Table 46). The difference between the fat and thin animals (EF vs ET and LF vs LT), at the start of the feedlot period was around 60 kg (2.3.1). Compensatory growth would have occurred if this difference had been reduced over a shorter period of time than the restrictive period (1.2.1.6). This occurred for both thin treatments. The early maturing animals (ET) achieved a 100% compensation, by reaching a non-significantly different weight to their corresponding fat animals (EF) by slaughter condition. The late maturing thin animals were however, significantly lighter than the late maturing fat animals, although the difference was reduced as compared to their masses at the start of the feedlot period (compensation < 100%).

Dressing percentages were relatively constant, the exception being that of the EF group which had a lower value than that attained by the late maturing groups.

Table 46. Slaughter mass and carcass return at the end of the feedlot period as influenced by treatments

TERM	TREATMENT			
	EF	LF	ET	LT
Condition Score Change / Day	0.00970[b]	0.00900[b]	0.01186[a]	0.01143[a]
Time in Feedlot (days)	95.8[b]	118.2[a]	117.2[a]	124.1[a]
Slaughter Mass (kg)	363.71[c]	439.92[a]	358.55[c]	396.79[b]
Cold Carcass Mass (kg)	205.67[c]	254.79[a]	203.91[c]	229.67[b]
Dressing %	56.46[b]	57.88[a]	57.00[ab]	57.92[a]

[a,b,c] Values in the same row with different superscripts are different $P < .05$.

The objective measurement of subcutaneous fat depths were taken to provide a more accurate assessment of the animals equality at the time of slaughter. The result (Table 47), was that there were no significant differences between treatments. This was irrespective of site of measurement. From these results it was concluded that the animals were slaughtered after having reached a similar degree of finish.

Table 47. Fat depth comparisons between treatments

TERM	TREATMENT			
	EF	LF	ET	LT
Fore-quarter (mm) (*1st*)	4.867[a]	4.588[a]	4.705[a]	3.654[a]
Loin (mm) (*2nd*)	4.338[a]	4.179[a]	5.514[a]	5.025[a]
Buttock (mm) (*3rd*)	2.125[a]	2.162[a]	2.850[a]	1.975[a]

[a,b,c] Values in the same row with different superscripts are different $P < .05$.

As the previous results (Table 47) only covered a specific site on each of the three areas of the carcass, the fat coverage over the whole of an area was assessed to give an indication of uniformity of deposition. Differences were found between treatments when compared on a fat coverage basis (Table 48). The earlier maturing thin animals were consistently fatter than the later maturing thin animals. When split to evaluate where on the carcass they were fatter, the results corresponded to Hammond's (1960) areas of early maturity (loin and fore-quarter). However, the fat coverage over the later maturing region of the buttock was found to be non-significantly different between the two groups of animals. The remaining two treatments (EF and LF) were non-significantly different fatter than either of the previous two treatments.

The degree of marbling (indicating intermuscular fat visible to the naked eye) was significantly lower for the EF group. Internal fat however resulted in the LT group having a significantly lower level than the other treatments.

Table 48. Fat coverage comparisons between treatments

TERM	TREATMENT			
	EF	LF	ET	LT
Class (A)	2.750^{ab}	2.708^{ab}	3.182^{a}	2.458^{b}
Fat Coverage	$3-^{ab}$	$3-^{ab}$	$3+^{a}$	3^{b}
Buttock	$3-^{a}$	$3-^{a}$	3^{a}	$2+^{a}$
Loin	3^{ab}	3^{ab}	$3+^{a}$	$3-^{b}$
Fore-quarter	3^{ab}	$3-^{ab}$	$3+^{a}$	$2+^{b}$
Marbling	1.042^{b}	1.458^{a}	1.545^{a}	1.458^{a}
Internal Fat	1.500^{a}	1.333^{ab}	1.409^{a}	1.083^{b}

[a,b,c] Values in the same row with different superscripts are different $P < .05$.

Conformation as described in Table 49, is a ranking according to the roundness and fullness of the carcass. This measurement is breed specific as some breeds tend to produce rounder and fuller carcasses. No benefit in terms of returns of product are observable for a fuller carcass. If there were, buyers would insist on a grading relative to this characteristics and pay a premium according to his choice of a better carcass. The later maturing animals were graded with a better conformation score than the earlier maturing animals (Table 49).

The eye-muscle measurements indicated that the early maturing thin animals had significantly smaller eye-muscle widths compared to the late maturing fat animals. Their lengths were also significantly shorter than all the other treatments.

Table 49. Conformation and eye-muscle width and length comparisons between treatments

TERM	TREATMENT			
	EF	LF	ET	LT
Conformation	3.417[b]	5.250[a]	4.091[b]	5.417[a]
Eye Muscle Width (mm)	58.63[ab]	61.08[a]	57.41[b]	59.29[ab]
Eye Muscle Length (mm)	125.21[a]	121.92[a]	115.32[b]	120.54[a]

[a,b,c] Values in the same row with different superscripts are different $P < .05$.

6.4 DISCUSSION

The early maturing fat animals had a significantly shorter period of time in the feedlot compared to the other treatments. The performance in the feedlot during this time had to be taken into account. Within this chapter the main area of performance measured was that of fat deposition. The compensating animals showed that during their time within the feedlot, they deposited significantly more fat at a significantly faster rate, as compared to the fat animals.

The surprising finding that there was no significant difference in fat deposition with respect to maturity type. The earlier maturing animal was expected to have higher fat deposition rate due to it being further along its growth curve (Butterfield, 1988). The level of fat within a carcass is due to a balance between maturity type and the level of nutrition (Butterfield, 1988). Animals in these two treatments started the feedlot period at the same condition score, they reached the slaughter condition over significantly different periods of time at non-significantly different rates. The reason for the non-

significantly different rates could be due to the large variation in slaughter times within the late maturing treatment.

The comparisons of the animals in terms of their masses is covered more fully in section 4.3.2 .i.e. the section concerning liveweight changes over the feedlot period. No explanation can be offered for the early maturing fat animals attaining a dressing percentage lower than that of the late maturing animals. All animals were treated the same with respect to empty body weight measurements and carcass preparation. The degree of fatness could have indicted that if the animals had been over fat, some of the excess might have been removed during the dressing of the carcass. this would have affected the dressing percentage. There was however no evidence of fat removal on examination of the carcasses and the early maturing animals are non-significantly fatter than the other treatments.

The sub-cutaneous fat depth measurements being non-significantly different between treatments implies that the goal of slaughtering at an equal degree of finish was achieved. Comparison of the results between sites illustrated the primary growth gradient within the body (1.2.1.1). The buttock site was found to have a lower level of fat than the other two sites. This result was first illustrated by Pálsson (1955).

The fat coverage results show that the objective was not achieved to perfection. It appears from the results that the compensating, early maturing animals (ET) were slaughtered at a slightly fatter level and the compensating late maturing animals (LT) at a slightly thinner level. This implies that the ET group could have been finished earlier and the LT group later than was the case in this study. The reasons for the discrepancies between the objective depth measurements and the subjective coverage scores is that the objective measurement describes the fat in only one place over the site. Fat is deposited unevenly over a site due to the growth gradients eg. Fat is deposited last over the lower rib area (1.2.1.4.3). Thus, a fatter animal, will have a larger proportion of fat in the this area of the site, than a thinner animal. The results relating to internal fat follow the same trend as for the subcutaneous fat. An animal has a constant fat deposition in the channel region over time (Figure 3), therefore fatter animal will have

deposited more fat in this region. Maturity-type differences are excluded due to the late maturing, fat group not being significantly different from the early maturing fat and early maturing thin group.

The eye-muscle diameters were significantly larger than those measured by ultrasound as described in Chapter 5. This could be due to distortion of the *longissimus dorsi* during halving and hanging of the carcass or the fact that the width measurement was taken at the widest point and not at a set distance from the midline. Temple *et al.* (1965), examining the location of scanning on the live animal and carcass, showed the sites to shift as much as five centimetres in relation to the skeleton, when the carcass was hung on the rail. The same trends are however found, as in Chapter 5. That is, the ET group having a significantly smaller diameter/width than the LF group. The EF group having a non-significantly different width and length of the eye-muscle, and a significantly lighter carcass, is showing a higher return of this high priced muscle per kg of carcass. This is also true for the ET group whereby the EF group has a significantly larger width and length of eye-muscle with a non-significantly different carcass mass. This however could be offset, by the LF, LT and ET groups having their eye-muscle extending for a greater length along the carcass. This could be true in the later maturing animals as they have a larger skeletal frame (1.2.1.7). This could also be true but due to a lesser extent for the ET group as by spending a significantly longer period of time in the feedlot they were allowed a longer period of time for skeletal growth. The answer to this question can be achieved only by measuring the length of the eye-muscle at slaughter.

GENERAL DISCUSSION

The object of the trial was to examine the interactions between maturity type and pre-feedlot plane of nutrition on the growth and performance of steers. The literature reviewed provided the basis for the comparison of feedlot performances, in terms of tissue deposition. Irrespective of chronological age, previous plane of nutrition or maturity type, an animal aims to attain a genetically determined body composition. The environmental factors have a potentially large effect as to whether this body composition is attained.

The literature review was split into four main sections, namely, normal growth, restricted growth, compensatory growth and breed and maturity type differences. Normal growth can be summarised using Figure 2, and Hammond's (1960) concepts of maturity gradients. Tissues have set growth patterns, with the following order of maturity bone, muscle then fat. Coupled with these growth patterns are the growth gradients within a tissue described as the primary and secondary growth waves by Hammond (1960).

The effect of restricted growth depended on the degree of restriction and the length of the restriction period. Tissues were affected in order of the growth impetus. That is the tissue that under normal growth would be growing at the highest rate, would be affected the most. Within a tissue, restricted growth affected the site of maximum deposition to a greater degree. The model of growth (1.2.1.2), by Berg and Butterfield (1976), showed that no tissue is affected independent of another. Compensatory growth has been found to result in the reverse of body composition differences imposed by restricted feeding. Thus tissues, whose growth was most severely affected during restriction, will have the greatest growth during compensation. The degree of compensation is dependent on the availability of nutrients and the length of the recovery period. The growth rates achieved during compensation are greater than those by unrestricted animals of an equal chronological age, and animals of an equal weight but younger chronologically (Baker *et al.*, 1985).

A large amount of variation has been reported in the literature concerning breed differences. A number of these can however be attributed to maturity type differences. Simply, an early maturing animal is further along its normal growth curve, as compared to a later maturing animal, at an equal chronological age. This results in the later maturing animal being heavier, larger at a set chronological age. The deposition rates of the tissues are offset by the animals maturity type, with the later maturing animal being larger and requiring a longer period of time to reach physiological maturity.

From the literature review it was apparent that there was a need to compare animals, of differing maturity type and pre-feedlot plane of nutrition, on a physiological age basis. Any differences at an equal physiological age can therefore be more directly related to genetic superiority.

The pre-feedlot period, achieved its objectives of restricting the growth of the thin treatments. A difference of sixty kilograms was achieved over 103 days of restriction, at the target growth rates. Thus on entering the feedlot period the thin treatments were roughly twenty five per cent lighter.

Examination of the animals' condition found that all treatments lost an equal amount of condition. Thus the fat treatments were also restricted, but to a lesser extent, by the pre-feedlot period. By having a positive growth gradient, the fat treatments would have been depositing all three tissues, but the tissues of highest growth impetus would have been deposited the least (Berg and Butterfield, 1976). Condition scores measure subcutaneous fat. As this was reducing for all treatments, and the fat animals were exhibiting positive growth, fat deposition must have occurred in alternative sites. These sites could have been those with a lower impetus for growth, i.e sites of deposition that were preferentially deposited in at a younger physiological age. Possible sites are the kidney, channel and intramuscular (1.2.1.4.3).

The urea dilution technique as urea space (US%), was adopted to determine the chemical composition, of the animals, over time, during the feedlot period. The results of the technique were disappointing. Roughly fifty per cent of the measurements had to

be discarded, due to their not meeting the criterion set for their acceptance (3.2.1.3). The variation was still high between the remaining measurements. The variation was found to be dependent on the degree of influence that the US % had on the prediction equations. If the US % use in the prediction equation was close to or greater than one, then the variation found in the US % measurements, was closely reflected in the tissue prediction. This was found to be the case with the prediction of fat. The high levels of variation, resulted in an inability, to find significant differences, with respect to weights and changes in weights of tissues. Unable to accurately predict the tissue composition of the animals over time, it was impossible to determine the treatments comparative physiological ages. Thus the treatments feedlot performances were not compared on a equal physiological basis.

Despite the limitations in the urea dilution data, it was shown that significant differences in tissue deposition occurred between treatments. Measurement of the animals height, showed that the compensating animals grew at a significantly greater rate. As height is correlated to long bone growth and thus bone tissue deposition the compensating animals had a greater deposition of bone tissue. No differences were however found between maturity types. Muscle growth in the form of the eye muscle diameter, showed that the growth rate, during the feedlot period was at a maximum, as no significant differences were found between the late maturing treatments or the early maturing thin animals. Significant differences between these treatments and that of the early maturing fat animals was predictable as these treatment were physiologically younger and thus had a higher protein deposition rate. The lack of differences between the three treatments, despite the late maturing thin animals expectation of the highest growth rate, could have been due to the rate of tissue deposition being the maximum possible under the prevailing environmental conditions. The rate of change in condition score gave an indication of the rate of change in subcutaneous fat deposition. The compensating animals deposited fat at a significantly greater rate. There was however a lack of difference in the fat deposition between maturity type, which could have been due to the late maturing animals higher net energy for gain (NEg) availability.

Differences were expected between maturity types with respect to skeletal size and bone growth. The results matched these predictions with the later maturing animals, being taller and having a higher growth in height, at an equal chronological age. The combination of the late maturing thin animals starting the feedlot period significantly shorter than the late maturing fat animals, and both compensating treatments (ET and LT), having significantly higher growth rates than the non-compensating treatments, is an indication of compensatory growth. The initial shortness, at the beginning of the feedlot period, was not however attributable to the pre-feedlot period. It could not be attributed to reduced bone growth, due to the planes of nutrition, as the animals were not measured for height during this period. Thus there is no means of knowing whether the animals came into the pre-feedlot period at significantly different heights or whether the significant differences were as a result of the pre-feedlot plane of nutrition.

Measurement of the eye-muscle diameters, showed that the lean tissue growth, of the restricted treatments, during the pre-feedlot period was significantly reduced. The rates of gain during the feedlot period, indicated that a maximum rate was being attained, due to the lack of difference between the treatments LF, ET and LT. The compensatory growth by the early maturing thin animals, resulted in a non-significant difference between them and the early maturing fat animals at the end of the feedlot period. These results were also mirrored in the carcass analysis, with the late maturing animals having significantly larger eye-muscles than the early maturing thin animals. The eye-muscles as a proportion of the carcass however, give the indication that the early maturing thin animals have a greater return.

The animal's liveweight change during the feedlot period, is one of the most important measurement to a producer. The late maturing animals gained weight at a significantly greater rate. As a proportion of their liveweight, this higher rate of gain is non-significantly different from that achieved by the lighter early maturing animals. Comparisons between the pre-feedlot treatments (EF vs ET and LF vs LT), showed compensating animals to have a superior rate of gain, which when coupled to the lighter weights resulted in a significantly greater gain as a proportion of their liveweight.

The division of the treatments into their production groups (rate of gain), requires an investigation into the cost of achieving these gains (feed intake and length of time in the feedlot). Analysis of the feed intake data presented problems due to the inadequate fit of the generally accepted quadratic model. The quadratic model, was found to underestimate, peak feed intake. This was due to the data seemingly following a linear increase to a peak point, from which point onwards the feed intake followed a more plateau trend. The quadratic model was therefore just finding the best fit through the two possible linear lines. A further point raised by the data was that the peak feed intake occurred at the same point of time irrespective of maturity type or condition. As the treatments have different growth expectations, their nutrient requirements are also expected to differ. It was therefore considered unlikely that the animals were able to attain their maximum feed intakes (and thus nutrient requirements) at the same point in time. Consequently a broken stick model was fitted. This model has the capacity to fit two linear models with significantly different linear components, as well as indicate whether the break (peak feed intake), or the point where the linear components change, are significantly different between treatments. The broken stick model improved the fit (R^2) of prediction models. In all cases the break in the data occurred at the same point of time, irrespective of maturity type.

Feed intakes were similar between treatments. Those treatments that started with low intakes, had greater rates of increase in intake over time. The feed intakes when compared as a proportion of metabolic weight $W^{0.75}$), found the compensating animals to have consumed a significantly greater amount per kilogram of their metabolic weight. The compensating animals were found to have increased liveweight gains and increased feed intakes, therefore an analysis of their efficiency of utilisation of the feed was performed. The compensating animals efficiency of utilisation of feed was significantly superior to that of that non-compensating animals. There were however no significant differences between maturity types.

The combination of knowing the animals net energy intake (energy content of the feed multiplied by the feed intake), and the prediction of the animals net energy for maintenance requirements, allowed for the analysis of the net energy available for

growth (NEg), over time. The results closely matched those attained for the feed intakes. Significant differences were however accentuated in some cases. This was because compensating animals' lower liveweights meant that their net energy maintenance requirements were lower. The compensating animals utilised the net energy available for growth with greater efficiency, again there was a lack of significant difference between maturity types.

The use of the broken stick model for the analysis of the feed intake data is controversial. A biological reason for the animals suddenly changing their feed intake trends must be found. Hicks *et al.* (1990a and b) examination of feed intake for season, sex and breed generated similar data as that examined in this trial. A further examination of the peak feed intake trends needs to be performed. Either the use of a better prediction model, or more precisely, an examination of what factor (s) could be limiting the animals feed intakes irrespective of maturity type, sex or season.

The animals in their respective treatments spent significantly different periods of time in the feedlot. That is the early maturing fat animals spent a significantly shorter period of time in the feedlot. There were no significant differences between the other treatments, although the late maturing thin animals were on average in the feedlot for a longer period of time. Analysis of the fat distribution data revealed that the compensating treatments were significantly different from each other. The early maturing thin animals could therefore have spent less time in the feedlot and the late maturing thin animals longer in the feedlot. Their differences in length of time in the feedlot could then have been significant.

Fat depth measurements of the carcass found the animals to be non-significantly different. This indicating that the animals had all attained an equal degree of finish in the feedlot. Analysis of the fat coverage however showed there to be significant differences between treatments. Those areas where significant differences were found, were those areas of early maturity with respect to subcutaneous fat deposition (1.2.1.4.3). That is the areas of fat deposition did not truly indicate differences with respect to overall subcutaneous fat deposition.

CONCLUSIONS

The pre-feedlot period successfully grouped the animals into the required feedlot treatments i.e a sixty kilogram difference in liveweight, between compensating and non-compensating treatments (EF vs ET and LF vs LT). Even though the fat treatments gained weight they experienced an equal loss in subcutaneous fat coverage, as measured by condition score.

The urea dilution technique was not successful in its application. Fifty per cent of measurements were rejected and the remaining measurements contained a large amount of variation between data points. This variation depended on the degree of influence of the US % in the prediction equation. Most rejected measurements were attributable to problems involved with infusing the large volumes of the solution into the animals within the required period of time.

Differences between compensating and non-compensating animals with respect to tissue deposition were recorded. Bone, muscle and fat tissue deposition rates were superior for the compensating animals. The muscle deposition rate (measured as change in eye muscle diameter over time), seemed to indicate that a maximum rate had been reached, due to the lack of difference between the late maturing treatments.

Changes in liveweight were as predicted from literature. Late maturing animals superior to early maturing and compensating animals superior to non-compensating. The early maturing thin animals were able to make up the sixty kilogram deficit over the early maturing fat animals within there period of time within the feedlot. Analysis of feed intake produced irregularities with respect to type of regression model, and the time of peak feed intake and net energy available for growth (NEg), being reached. Overall, the compensating animals consumed a significantly greater amount of food and NEg per kilogram of metabolic weight, but their efficiency of utilisation was significantly superior.

The only differences between maturity types are the weight gained and the length of time they spend in the feedlot. The choice of maturity type therefore, has to take into account

that the extra weight gained by the later maturing animals must offset the opportunity cost of the extra period of time they spend in the feedlot and vice versa for the earlier maturing animals. Comparisons of pre-feedlot planes of nutrition revealed that those animals on a lower plane of nutrition (which was achieved at a lower cost), are significantly more efficient in the feedlot.

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APPENDIX 1

REGRESSION OF WEIGHT OF WATER vs TIME
*** Summary of analysis ***

	d.f.	s.s.	m.s.	v.r.
Regression	7	19878.	2839.7	6.18
Residual	14	6437.	459.8	
Total	21	26314.	1253.1	
Change	-3	-1012.	337.5	0.73

Percentage variance accounted for 63.3
Standard error of observations is estimated to be 21.4

*** Estimates of regression coefficients ***

	estimate	s.e.	t(14)	t pr.
Constant	154.1	20.0	7.72	<.001
WEEKS	6.75	5.13	1.32	0.209
EF : LF	32.7	28.2	1.16	0.265
EF : ET	-61.9	28.3	-2.19	0.046
EF : LT	-24.2	28.3	-0.86	0.406
WEEKS.EF : LF	-0.46	7.25	-0.06	0.951
WEEKS.EF : ET	8.46	7.28	1.16	0.265
WEEKS.EF : LT	5.93	7.28	0.81	0.429
Constant	92.3	20.0	4.61	<.001
WEEKS	15.21	5.17	2.94	0.011
ET : EF	61.9	28.3	2.19	0.046
ET : LF	94.6	28.3	3.35	0.005
ET : LT	37.6	28.3	1.33	0.205
WEEKS.ET : EF	-8.46	7.28	-1.16	0.265
WEEKS.ET : LF	-8.91	7.28	-1.22	0.241
WEEKS.ET : LT	-2.52	7.31	-0.35	0.735
Constant	186.9	20.0	9.36	<.001
WEEKS	6.30	5.13	1.23	0.239
LF : EF	-32.7	28.2	-1.16	0.265
LF : ET	-94.6	28.3	-3.35	0.005
LF : LT	-57.0	28.3	-2.02	0.064
WEEKS.LF : EF	0.46	7.25	0.06	0.951
WEEKS.LF : ET	8.91	7.28	1.22	0.241
WEEKS.LF : LT	6.39	7.28	0.88	0.395
Constant	129.9	20.0	6.49	<.001
WEEKS	12.69	5.17	2.45	0.028
LT : EF	24.2	28.3	0.86	0.406
LT : LF	57.0	28.3	2.02	0.064
LT : ET	-37.6	28.3	-1.33	0.205
WEEKS.LT : EF	-5.93	7.28	-0.81	0.429
WEEKS.LT : LF	-6.39	7.28	-0.88	0.395
WEEKS.LT : ET	2.52	7.31	0.35	0.735

REGRESSION OF WEIGHT OF FAT vs TIME

*** Summary of analysis ***

	d.f.	s.s.	m.s.	v.r.
Regression	7	6314.	902.0	1.26
Residual	14	10030.	716.5	
Total	21	16344.	778.3	
Change	-3	-1035.	345.0	0.48

Percentage variance accounted for 7.9
Standard error of observations is estimated to be 26.8

*** Estimates of regression coefficients ***

	estimate	s.e.	t(14)	t pr.
Constant	37.1	24.9	1.49	0.158
WEEKS	9.72	6.40	1.52	0.151
EF : LF	-11.3	35.2	-0.32	0.752
EF : ET	26.6	35.3	0.75	0.464
EF : LT	5.1	35.3	0.15	0.886
WEEKS.EF : LF	4.17	9.05	0.46	0.652
WEEKS.EF : ET	-6.39	9.09	-0.70	0.493
WEEKS.EF : LT	-2.77	9.09	-0.31	0.765
Constant	63.7	25.0	2.55	0.023
WEEKS	3.33	6.45	0.52	0.614
ET : EF	-26.6	35.3	-0.75	0.464
ET : LF	-37.9	35.3	-1.07	0.301
ET : LT	-21.4	35.4	-0.61	0.554
WEEKS.ET : EF	6.39	9.09	0.70	0.493
WEEKS.ET : LF	10.56	9.09	1.16	0.265
WEEKS.ET : LT	3.62	9.13	0.40	0.698
Constant	25.8	24.9	1.03	0.318
WEEKS	13.88	6.40	2.17	0.048
LF : EF	11.3	35.2	0.32	0.752
LF : ET	37.9	35.3	1.07	0.301
LF : LT	16.5	35.3	0.47	0.648
WEEKS.LF : EF	-4.17	9.05	-0.46	0.652
WEEKS.LF : ET	-10.56	9.09	-1.16	0.265
WEEKS.LF : LT	-6.94	9.09	-0.76	0.458
Constant	42.3	25.0	1.69	0.113
WEEKS	6.94	6.45	1.08	0.300
LT : EF	-5.1	35.3	-0.15	0.886
LT : LF	-16.5	35.3	-0.47	0.648
LT : ET	21.4	35.4	0.61	0.554
WEEKS.LT : EF	2.77	9.09	0.31	0.765
WEEKS.LT : LF	6.94	9.09	0.76	0.458
WEEKS.LT : ET	-3.62	9.13	-0.40	0.698

REGRESSION OF WEIGHT OF PROTEIN vs TIME

*** Summary of analysis ***

	d.f.	s.s.	m.s.	v.r.
Regression	7	1825.6	260.80	8.06
Residual	14	453.1	32.37	
Total	21	2278.7	108.51	
Change	-3	-75.3	25.12	0.78

Percentage variance accounted for 70.2
Standard error of observations is estimated to be 5.69

*** Estimates of regression coefficients ***

	estimate	s.e.	t(14)	t pr.
Constant	46.59	5.30	8.80	<.001
WEEKS	2.25	1.36	1.65	0.121
EF : LF	9.42	7.49	1.26	0.229
EF : ET	-17.66	7.50	-2.35	0.034
EF : LT	-7.05	7.50	-0.94	0.363
WEEKS.EF : LF	-0.03	1.92	-0.02	0.987
WEEKS.EF : ET	2.34	1.93	1.21	0.245
WEEKS.EF : LT	1.69	1.93	0.87	0.397
Constant	28.94	5.31	5.45	<.001
WEEKS	4.59	1.37	3.35	0.005
ET : EF	17.66	7.50	2.35	0.034
ET : LF	27.08	7.50	3.61	0.003
ET : LT	10.60	7.51	1.41	0.180
WEEKS.ET : EF	-2.34	1.93	-1.21	0.245
WEEKS.ET : LF	-2.38	1.93	-1.23	0.239
WEEKS.ET : LT	-0.66	1.94	-0.34	0.740
Constant	56.01	5.30	10.58	<.001
WEEKS	2.21	1.36	1.63	0.126
LF : EF	-9.42	7.49	-1.26	0.229
LF : ET	-27.08	7.50	-3.61	0.003
LF : LT	-16.47	7.50	-2.20	0.045
WEEKS.LF : EF	0.03	1.92	0.02	0.987
WEEKS.LF : ET	2.38	1.93	1.23	0.239
WEEKS.LF : LT	1.72	1.93	0.89	0.388
Constant	39.54	5.31	7.44	<.001
WEEKS	3.93	1.37	2.87	0.012
LT : EF	7.05	7.50	0.94	0.363
LT : LF	16.47	7.50	2.20	0.045
LT : ET	-10.60	7.51	-1.41	0.180
WEEKS.LT : EF	-1.69	1.93	-0.87	0.397
WEEKS.LT : LF	-1.72	1.93	-0.89	0.388
WEEKS.LT : ET	0.66	1.94	0.34	0.740

APPENDIX 2

CRUDE PROTEIN (%)

INGREDIENT	MEAN	CV%	95% CONFIDENCE INTERVALS	MAXIMUM	MINIMUM
HOMINY CHOP	10.8	5.93	1.57	11.5	9.4
CHICKEN LITTER	23.0	7.04	3.95	25.5	20.5
MOLASSES	10.2	6.27	1.57	10.6	8.7
PREMIX	30.4	3.95	2.94	32.8	28.8
MIXED RATION	14.7	13.54	4.86	16.6	10.2
CALCULATED MIXED RATION	14.5	4.34	1.54	15.4	13.4

CALCIUM (%)

INGREDIENT	MEAN	CV%	95% CONFIDENCE INTERVALS	MAXIMUM	MINIMUM
HOMINY CHOP	0.4	40.00	0.39	0.7	0.2
CHICKEN LITTER	4.2	15.00	1.54	5.5	3.5
MOLASSES	1.6	16.88	0.67	2.2	1.4
PREMIX	9.2	9.89	2.23	10.6	8.2
MIXED RATION	1.8	12.78	0.56	2.3	1.6
CALCULATED MIXED RATION	1.8	7.78	0.35	2.1	1.7

PHOSPHOROUS (%)

INGREDIENT	MEAN	CV%	95% CONFIDENCE INTERVALS	MAXIMUM	MINIMUM
HOMINY CHOP	0.5	20.00	0.24	0.7	0.4
CHICKEN LITTER	1.3	10.00	0.31	1.4	1.0
MOLASSES	0.2	15.00	0.06	0.2	0.1
PREMIX	0.4	27.50	0.27	0.5	0.2
MIXED RATION	0.7	10.00	0.17	0.8	0.6
CALCULATED MIXED RATION	0.7	12.86	0.21	0.8	0.5

FAT (%)

INGREDIENT	MEAN	CV%	95% CONFIDENCE INTERVALS	MAXIMUM	MINIMUM
HOMINY CHOP	9.3	17.42	3.97	11.0	6.5
CHICKEN LITTER	2.7	9.63	0.64	3.0	2.3
MOLASSES	0.6	61.67	0.91	1.3	0.2
PREMIX	4.6	18.04	2.02	6.3	3.8
MIXED RATION	7.1	7.04	1.22	7.7	6.3
CALCULATED MIXED RATION	6.7	15.52	2.55	7.9	4.9

ASH (%)

INGREDIENT	MEAN	CV%	95% CONFIDENCE INTERVALS	MAXIMUM	MINIMUM
HOMINY CHOP	2.7	11.11	0.72	3.0	2.2
CHICKEN LITTER	17.2	12.50	5.27	22.3	15.5
MOLASSES	17.1	4.80	2.00	18.4	15.9
PREMIX	34.0	14.47	12.05	40.7	28.2
MIXED RATION	8.3	6.75	1.37	9.4	7.6
CALCULATED MIXED RATION	9.1	4.18	0.93	9.8	8.6

MOISTURE (%)

INGREDIENT	MEAN	CV%	95% CONFIDENCE INTERVALS	MAXIMUM	MINIMUM
HOMINY CHOP	11.6	6.03	1.39	12.5	10.9
CHICKEN LITTER	26.2	11.18	7.16	30.0	20.4
MOLASSES	26.9	8.66	5.70	29.6	22.2
PREMIX	8.9	9.89	2.16	10.4	7.7
MIXED RATION	15.8	4.94	1.90	16.6	14.5
CALCULATED MIXED RATION	16.8	5.18	2.13	18.0	15.1

NEUTRAL DETERGENT FIBRE (%)

INGREDIENT	MEAN	CV%	95% CONFIDENCE INTERVALS	MAXIMUM	MINIMUM
HOMINY CHOP	28.5	13.30	9.27	34.3	20.5
CHICKEN LITTER	43.5	12.16	12.95	53.6	36.1
MOLASSES	0.0	0.0	0.0	0.0	0.0
PREMIX	16.2	21.23	8.41	20.9	12.1
MIXED RATION	27.8	7.95	5.41	32.0	24.7
CALCULATED MIXED RATION	28.2	11.38	7.87	31.8	21.5

ACID DETERGENT FIBRE (%)

INGREDIENT	MEAN	CV%	95% CONFIDENCE INTERVALS	MAXIMUM	MINIMUM
HOMINY CHOP	8.1	12.59	2.51	10.0	6.3
CHICKEN LITTER	32.8	11.37	9.13	39.3	29.3
MOLASSES	0.0	0.0	0.0	0.0	0.0
PREMIX	7.2	18.33	3.22	9.2	5.6
MIXED RATION	12.0	10.75	3.16	13.7	9.9
CALCULATED MIXED RATION	12.8	8.20	2.56	14.4	11.6

CRUDE FIBRE (%)

INGREDIENT	MEAN	CV%	95% CONFIDENCE INTERVALS	MAXIMUM	MINIMUM
HOMINY CHOP	6.7	19.10	3.13	9.1	4.8
CHICKEN LITTER	26.2	12.10	7.75	30.9	22.7
MOLASSES	0.0	0.0	0.0	0.0	0.0
PREMIX	5.6	17.68	2.43	6.9	3.9
MIXED RATION	*	*	*	*	*
CALCULATED MIXED RATION	10.3	11.26	2.84	11.8	8.8

METABOLISABLE ENERGY (MJ/kg)

INGREDIENT	MEAN	CV%	95% CONFIDENCE INTERVALS	MAXIMUM	MINIMUM
HOMINY CHOP	13.3	1.95	0.65	13.7	12.9
CHICKEN LITTER	8.0	5.63	1.11	8.7	7.5
MOLASSES	10.9	1.10	0.30	11.0	10.7
PREMIX	9.0	7.89	1.74	9.9	8.1
MIXED RATION	*	*	*	*	*
CALCULATED MIXED RATION	11.6	2.07	0.60	11.9	11.2

APPENDIX 3

Liveweight measurements over time

	EARLY MATURING FAT			LATE MATURING FAT		
WEEK	WEIGHT (kg)	CV%	n	WEIGHT (kg)	CV%	n
1	256.33	5.50	24	286.04	4.80	24
2	256.08	7.79	24	287.67	4.37	24
3	266.38	7.15	24	302.46	4.41	24
4	274.62	7.85	24	313.50	4.86	24
5	286.63	8.31	24	325.87	4.32	24
6	292.21	8.80	24	336.04	5.23	24
7	305.21	7.87	24	353.21	5.23	24
8	314.83	8.11	24	360.38	4.48	24
9	329.25	9.35	24	375.87	6.07	24
10	345.62	8.85	24	388.54	5.77	24
11	355.75	8.83	24	404.33	5.82	24
12	353.67	7.99	24	404.17	5.05	24
13	355.21	8.12	14	411.83	5.29	23
14	358.14	8.35	14	421.35	6.56	23
15	377.33	8.66	9	423.30	6.67	20
16	388.56	7.79	9	441.65	5.99	20
17	384.78	7.74	9	439.15	6.11	20
18	380.17	8.35	6	450.86	5.25	14
19	*	*	0	458.00	3.51	6
20	*	*	0	468.83	3.48	6
21	*	*	0	468.33	3.56	6

Liveweight measurements over time

	EARLY MATURING THIN			LATE MATURING THIN		
WEEK	WEIGHT (kg)	CV%	n	WEIGHT (kg)	CV%	n
1	196.48	11.02	23	216.29	10.38	24
2	209.35	10.37	23	233.92	10.81	24
3	216.74	10.30	23	245.67	11.69	24
4	229.96	10.49	23	258.12	11.61	24
5	239.17	10.62	23	268.25	11.92	24
6	250.70	9.76	23	285.08	11.61	24
7	265.17	10.42	23	296.67	11.86	24
8	281.57	11.06	23	313.04	11.98	24
9	294.61	10.08	23	325.37	12.61	24
10	304.78	10.05	23	336.12	12.14	24
11	313.65	10.40	23	345.08	11.85	24
12	326.32	9.57	22	356.88	12.13	24
13	336.81	8.39	21	366.67	11.01	24
14	343.52	9.01	21	373.54	10.79	24
15	349.37	7.70	19	381.59	11.02	22
16	359.42	7.93	19	395.36	10.63	22
17	357.79	7.38	19	393.64	10.56	22
18	364.77	7.22	13	402.50	10.84	20
19	360.17	8.03	6	403.50	10.90	8
20	360.67	6.25	6	409.25	10.85	8
21	361.00	5.24	6	410.25	11.27	8

REGRESSION OF LIVEWEIGHT vs TIME
*** Summary of analysis ***

	d.f.	s.s.	m.s.	v.r.
Regression	11	238639.	21694.44	907.95
Residual	56	1338.	23.89	
Total	67	239977.	3581.74	
Change	-3	-66.	22.00	0.92

Percentage variance accounted for 99.3
Standard error of observations is estimated to be 4.89

*** Estimates of regression coefficients ***

	estimate	s.e.	t(56)	t pr.
Constant	236.04	4.02	58.71	<.001
TIME	11.26	1.03	10.95	<.001
EF : LF	26.82	5.69	4.72	<.001
EF : ET	-59.30	5.69	-10.43	<.001
EF : LT	-36.60	5.69	-6.44	<.001
TIME.EF : LF	3.14	1.45	2.16	0.035
TIME.EF : ET	3.44	1.45	2.37	0.022
TIME.EF : LT	4.83	1.45	3.32	0.002
TIMESQ	-0.1312	0.0555	-2.36	0.022
TIMESQ.EF : LF	-0.0903	0.0785	-1.15	0.255
TIMESQ.EF : ET	-0.0803	0.0785	-1.02	0.311
TIMESQ.EF : LT	-0.1263	0.0785	-1.61	0.113
Constant	262.86	4.02	65.38	<.001
TIME	14.39	1.03	14.00	<.001
LF : EF	-26.82	5.69	-4.72	<.001
LF : ET	-86.12	5.69	-15.15	<.001
LF : LT	-63.42	5.69	-11.15	<.001
TIME.LF : EF	-3.14	1.45	-2.16	0.035
TIME.LF : ET	0.30	1.45	0.21	0.836
TIME.LF : LT	1.69	1.45	1.16	0.251
TIMESQ	-0.2215	0.0555	-3.99	<.001
TIMESQ.LF : EF	0.0903	0.0785	1.15	0.255
TIMESQ.LF : ET	0.0100	0.0785	0.13	0.899
TIMESQ.LF : LT	-0.0359	0.0785	-0.46	0.649
Constant	176.74	4.02	43.96	<.001
TIME	14.69	1.03	14.29	<.001
ET : EF	59.30	5.69	10.43	<.001
ET : LF	86.12	5.69	15.15	<.001
ET : LT	22.69	5.69	3.99	<.001
TIME.ET : EF	-3.44	1.45	-2.37	0.022
TIME.ET : LF	-0.30	1.45	-0.21	0.836
TIME.ET : LT	1.39	1.45	0.95	0.344
TIMESQ	-0.2115	0.0555	-3.81	<.001
TIMESQ.ET : EF	0.0803	0.0785	1.02	0.311
TIMESQ.ET : LF	-0.0100	0.0785	-0.13	0.899
TIMESQ.ET : LT	-0.0459	0.0785	-0.59	0.561
Constant	199.44	4.02	49.61	<.001
TIME	16.08	1.03	15.64	<.001
LT : EF	36.60	5.69	6.44	<.001
LT : LF	63.42	5.69	11.15	<.001
LT : ET	-22.69	5.69	-3.99	<.001
TIME.LT : EF	-4.83	1.45	-3.32	0.002
TIME.LT : LF	-1.69	1.45	-1.16	0.251
TIME.LT : ET	-1.39	1.45	-0.95	0.344
TIMESQ	-0.2574	0.0555	-4.64	<.001
TIMESQ.LT : EF	0.1263	0.0785	1.61	0.113
TIMESQ.LT : LF	0.0359	0.0785	0.46	0.649
TIMESQ.LT : ET	0.0459	0.0785	0.59	0.561

APPENDIX 4

Average daily gains over time

	EARLY MATURING FAT		LATE MATURING FAT	
WEEK	ADG/WEIGHT	n	ADG/WEIGHT	n
1	*	24	*	24
2	0.000	24	0.001	24
3	0.006	24	0.007	24
4	0.004	24	0.005	24
5	0.006	24	0.005	24
6	0.003	24	0.004	24
7	0.006	24	0.007	24
8	0.004	24	0.003	24
9	0.006	24	0.006	24
10	0.007	24	0.005	24
11	0.004	24	0.006	24
12	-0.001	24	0.000	24
13	0.001	14	0.003	23
14	0.001	14	0.003	23
15	0.007	9	0.001	20
16	0.004	9	0.006	20
17	-0.001	9	-0.001	20

Average daily gains over time

WEEK	EARLY MATURING THIN ADG/WEIGHT	n	LATE MATURING THIN ADG/WEIGHT	n
1	*	23	*	24
2	0.009	23	0.011	24
3	0.005	23	0.007	24
4	0.008	23	0.007	24
5	0.006	23	0.005	24
6	0.007	23	0.008	24
7	0.008	23	0.006	24
8	0.008	23	0.007	24
9	0.006	23	0.005	24
10	0.005	23	0.005	24
11	0.004	23	0.004	24
12	0.006	22	0.005	24
13	0.004	21	0.004	24
14	0.003	21	0.003	24
15	0.002	19	0.003	22
16	0.004	19	0.005	22
17	-0.001	19	-0.001	22

ADG/WEIGHT (EFFICIENCY)
*** Summary of analysis ***

	d.f.	s.s.	m.s.	v.r.
Regression	11	0.0002248	0.204E-04	4.67
Residual	52	0.0002276	0.438E-05	
Total	63	0.0004524	0.718E-05	
Change	-3	-0.0000085	0.284E-05	0.65

Percentage variance accounted for 39.1
Standard error of observations is estimated to be 0.00209

*** Estimates of regression coefficients ***

	estimate	s.e.	t(52)	t pr.
Constant	0.00133	0.00225	0.59	0.559
TIME	0.000845	0.000538	1.57	0.122
EF : LF	0.00164	0.00319	0.51	0.610
EF : ET	0.00532	0.00319	1.67	0.101
EF : LT	0.00821	0.00319	2.58	0.013
TIME.EF : LF	-0.000281	0.000761	-0.37	0.713
TIME.EF : ET	-0.000564	0.000761	-0.74	0.462
TIME.EF : LT	-0.001293	0.000761	-1.70	0.095
TIMESQ	-0.0000520	0.0000277	-1.88	0.066
TIMESQ.EF : LF	0.0000111	0.0000391	0.28	0.778
TIMESQ.EF : ET	0.0000154	0.0000391	0.39	0.696
TIMESQ.EF : LT	0.0000515	0.0000391	1.32	0.194
Constant	0.00296	0.00225	1.31	0.194
TIME	0.000564	0.000538	1.05	0.299
LF : EF	-0.00164	0.00319	-0.51	0.610
LF : ET	0.00368	0.00319	1.15	0.254
LF : LT	0.00658	0.00319	2.06	0.044
TIME.LF : EF	0.000281	0.000761	0.37	0.713
TIME.LF : ET	-0.000283	0.000761	-0.37	0.712
TIME.LF : LT	-0.001012	0.000761	-1.33	0.189
TIMESQ	-0.0000409	0.0000277	-1.48	0.145
TIMESQ.LF : EF	-0.0000111	0.0000391	-0.28	0.778
TIMESQ.LF : ET	0.0000043	0.0000391	0.11	0.912
TIMESQ.LF : LT	0.0000404	0.0000391	1.03	0.306
Constant	0.00664	0.00225	2.95	0.005
TIME	0.000281	0.000538	0.52	0.603
ET : EF	-0.00532	0.00319	-1.67	0.101
ET : LF	-0.00368	0.00319	-1.15	0.254
ET : LT	0.00290	0.00319	0.91	0.368
TIME.ET : EF	0.000564	0.000761	0.74	0.462
TIME.ET : LF	0.000283	0.000761	0.37	0.712
TIME.ET : LT	-0.000729	0.000761	-0.96	0.342
TIMESQ	-0.0000366	0.0000277	-1.32	0.192
TIMESQ.ET : EF	-0.0000154	0.0000391	-0.39	0.696
TIMESQ.ET : LF	-0.0000043	0.0000391	-0.11	0.912
TIMESQ.ET : LT	0.0000361	0.0000391	0.962	0.361
Constant	0.00954	0.00225	4.23	<.001
TIME	-0.000448	0.000538	-0.83	0.409
LT : EF	-0.00821	0.00319	-2.58	0.013
LT : LF	-0.00658	0.00319	-2.06	0.044
LT : ET	-0.00290	0.00319	-0.91	0.368
TIME.LT : EF	0.001293	0.000761	1.70	0.095
TIME.LT : LF	0.001012	0.000761	1.33	0.189
TIME.LT : ET	0.000729	0.000761	0.96	0.342
TIMESQ	-0.0000005	0.0000277	-0.02	0.986
TIMESQ.LT : EF	-0.0000515	0.0000391	-1.32	0.194
TIMESQ.LT : LF	-0.0000404	0.0000391	-1.03	0.306
TIMESQ.LT : ET	-0.0000361	0.0000391	-0.92	0.361

APPENDIX 5

Feed intake and feed intake / metabolic weight over time

WEEK	EARLY MATURING FAT			LATE MATURING FAT		
	FEED INTAKE (kg)	FEED INTAKE/ $W^{0.75}$ (kg)	n	FEED INTAKE (kg)	FEED INTAKE/ $W^{0.75}$ (kg)	n
1	37.44	0.59	24	44.37	0.64	24
2	44.37	0.67	24	49.92	0.69	24
3	40.21	0.60	24	51.31	0.69	24
4	56.85	0.82	24	61.01	0.80	24
5	56.85	0.80	24	62.40	0.80	24
6	66.56	0.91	24	76.27	0.94	24
7	65.17	0.87	24	77.65	0.94	24
8	65.17	0.84	24	76.54	0.90	24
9	67.53	0.84	24	77.24	0.88	24
10	61.98	0.76	24	74.33	0.82	24
11	65.17	0.80	24	74.88	0.83	24
12	78.45	0.96	24	76.69	0.84	24
13	73.49	0.89	14	75.75	0.82	23
14	85.59	1.00	14	74.64	0.80	23
15	60.31	0.69	9	70.27	0.73	20
16	77.59	0.89	9	82.34	0.86	20
17	83.02	0.96	9	78.37	0.80	20
18	0.00	0.00	0	94.29	0.95	6
19	0.00	0.00	0	94.29	0.94	6
20	0.00	0.00	0	60.45	0.60	6

Feed intake and feed intake / metabolic weight over time

WEEK	EARLY MATURING THIN			LATE MATURING THIN		
	FEED INTAKE (kg)	FEED INTAKE/ $W^{0.75}$ (kg)	n	FEED INTAKE (kg)	FEED INTAKE/ $W^{0.75}$ (kg)	n
1	34.73	0.63	23	42.99	0.72	24
2	41.96	0.74	23	49.92	0.81	24
3	41.96	0.71	23	49.92	0.78	24
4	54.98	0.90	23	54.08	0.82	24
5	50.64	0.80	23	56.85	0.82	24
6	63.67	0.97	23	72.11	1.01	24
7	68.01	0.99	23	72.11	0.97	24
8	63.67	0.90	23	72.94	0.95	24
9	69.02	0.95	23	72.66	0.93	24
10	58.89	0.79	23	72.66	0.91	24
11	60.51	0.79	23	67.12	0.82	24
12	64.98	0.83	22	72.66	0.87	24
13	62.90	0.79	21	71.79	0.85	24
14	64.70	0.80	21	71.70	0.83	24
15	62.62	0.76	19	70.00	0.79	22
16	65.15	0.79	19	74.09	0.84	22
17	60.36	0.72	19	68.43	0.76	22
18	93.33	1.13	6	94.00	1.04	8
19	93.33	1.13	6	85.00	0.93	8
20	59.00	0.71	6	66.39	0.73	8

APPENDIX 6

REGRESSION OF FEED INTAKE vs TIME (QUADRATIC MODEL)

*** Summary of analysis ***

	d.f.	s.s.	m.s.	v.r.
Regression	11	8200.	745.41	28.70
Residual	56	1454.	25.97	
Total	67	9654.	144.09	
Change	-3	-67.	22.18	0.85

Percentage variance accounted for 82.0
Standard error of observations is estimated to be 5.10

*** Estimates of regression coefficients ***

	estimate	s.e.	t(56)	t pr.
Constant	34.20	4.19	8.16	<.001
WEEKS	5.03	1.07	4.70	<.001
EF : LF	4.97	5.93	0.84	0.406
EF : ET	-3.59	5.93	-0.61	0.547
EF : LT	2.88	5.93	0.49	0.629
WEEKS.EF : LF	1.30	1.52	0.86	0.394
WEEKS.EF : ET	1.08	1.52	0.71	0.479
WEEKS.EF : LT	1.06	1.52	0.70	0.488
WEEKSSQ	-0.1489	0.0579	-2.57	0.013
WEEKSSQ.EF : LF	-0.1037	0.0819	-1.27	0.210
WEEKSSQ.EF : ET	-0.1127	0.0819	-1.38	0.174
WEEKSSQ.EF : LT	-0.1035	0.0819	-1.26	0.211
Constant	39.16	4.19	9.34	<.001
WEEKS	6.34	1.07	5.91	<.001
LF : EF	-4.97	5.93	-0.84	0.406
LF : ET	-8.55	5.93	-1.44	0.155
LF : LT	-2.09	5.93	-0.35	0.726
WEEKS.LF : EF	-1.30	1.52	-0.86	0.394
WEEKS.LF : ET	-0.22	1.52	-0.15	0.883
WEEKS.LF : LT	-0.24	1.52	-0.16	0.873
WEEKSSQ	-0.2526	0.0579	-4.36	<.001
WEEKSSQ.LF : EF	0.1037	0.0819	1.27	0.210
WEEKSSQ.LF : ET	-0.0090	0.0819	-0.11	0.913
WEEKSSQ.LF : LT	0.0002	0.0819	0.00	0.998
Constant	30.61	4.19	7.30	<.001
WEEKS	6.11	1.07	5.70	<.001
ET : EF	3.59	5.93	0.61	0.547
ET : LF	8.55	5.93	1.44	0.155
ET : LT	6.47	5.93	1.09	0.280
WEEKS.ET : EF	-1.08	1.52	-0.71	0.479
WEEKS.ET : LF	0.22	1.52	0.15	0.883
WEEKS.ET : LT	-0.02	1.52	-0.01	0.989
WEEKSSQ	-0.2616	0.0579	-4.52	<.001
WEEKSSQ.ET : EF	0.1127	0.0819	1.38	0.174
WEEKSSQ.ET : LF	0.0090	0.0819	0.11	0.913
WEEKSSQ.ET : LT	0.0092	0.0819	0.11	0.911
Constant	37.08	4.19	8.85	<.001
WEEKS	6.09	1.07	5.68	<.001
LT : EF	-2.88	5.93	-0.49	0.629
LT : LF	2.09	5.93	0.35	0.726
LT : ET	-6.47	5.93	-1.09	0.280
WEEKS.LT : EF	-1.06	1.52	-0.70	0.488
WEEKS.LT : LF	0.24	1.52	0.16	0.873
WEEKS.LT : ET	0.02	1.52	0.01	0.989
WEEKSSQ	-0.2523	0.0579	-4.36	<.001
WEEKSSQ.LT : EF	0.1035	0.0819	1.26	0.211
WEEKSSQ.LT : LF	-0.0002	0.0819	0.00	0.998
WEEKSSQ.LT : ET	-0.0092	0.0819	-0.11	0.911

REGRESSION OF FEED INTAKE / MEATABOLIC WEIGHT ($W^{0.75}$) vs TIME (QUADRATIC MODEL)

*** Summary of analysis ***

	d.f.	s.s.	m.s.	v.r.
Regression	11	0.01779	0.0016168	5.58
Residual	56	0.01622	0.0002896	
Total	67	0.03400	0.0005075	
Change	-3	-0.00040	0.0001342	0.46

Percentage variance accounted for 42.9
Standard error of observations is estimated to be 0.0170

*** Estimates of regression coefficients ***

	estimate	s.e.	t(56)	t pr.
Constant	0.1473	0.0140	10.53	<.001
WEEKS	0.00947	0.00358	2.65	0.011
EF : LF	0.0025	0.0198	0.13	0.899
EF : ET	0.0253	0.0198	1.28	0.206
EF : LT	0.0340	0.0198	1.72	0.092
WEEKS.EF : LF	0.00102	0.00506	0.20	0.842
WEEKS.EF : ET	0.00154	0.00506	0.30	0.762
WEEKS.EF : LT	-0.00052	0.00506	-0.10	0.919
WEEKSSQ	-0.000390	0.000193	-2.02	0.048
WEEKSSQ.EF : LF	-0.000186	0.000273	-0.68	0.498
WEEKSSQ.EF : ET	-0.000319	0.000273	-1.17	0.248
WEEKSSQ.EF : LT	-0.000195	0.000273	-0.71	0.479
Constant	0.1498	0.0140	10.70	<.001
WEEKS	0.01049	0.00358	2.93	0.005
LF : EF	-0.0025	0.0198	-0.13	0.899
LF : ET	0.0228	0.0198	1.15	0.254
LF : LT	0.0314	0.0198	1.59	0.118
WEEKS.LF : EF	-0.00102	0.00506	-0.20	0.842
WEEKS.LF : ET	0.00053	0.00506	0.10	0.918
WEEKS.LF : LT	-0.00153	0.00506	-0.30	0.763
WEEKSSQ	-0.000576	0.000193	-2.98	0.004
WEEKSSQ.LF : EF	0.000186	0.000273	0.68	0.498
WEEKSSQ.LF : ET	-0.000133	0.000273	-0.49	0.629
WEEKSSQ.LF : LT	-0.000009	0.000273	-0.03	0.975
Constant	0.1727	0.0140	12.34	<.001
WEEKS	0.01101	0.00358	3.08	0.003
ET : EF	-0.0253	0.0198	-1.28	0.206
ET : LF	-0.0228	0.0198	-1.15	0.254
ET : LT	0.0086	0.0198	0.44	0.665
WEEKS.ET : EF	-0.00154	0.00506	-0.30	0.762
WEEKS.ET : LF	-0.00053	0.00506	-0.10	0.918
WEEKS.ET : LT	-0.00206	0.00506	-0.41	0.686
WEEKSSQ	-0.000709	0.000193	-3.67	<.001
WEEKSSQ.ET : EF	0.000319	0.000273	1.17	0.248
WEEKSSQ.ET : LF	0.000133	0.000273	0.49	0.629
WEEKSSQ.ET : LT	0.000124	0.000273	0.45	0.652
Constant	0.1813	0.0140	12.95	<.001
WEEKS	0.00895	0.00358	2.50	0.015
LT : EF	-0.0340	0.0198	-1.72	0.092
LT : LF	-0.0314	0.0198	-1.59	0.118
LT : ET	-0.0086	0.0198	-0.44	0.665
WEEKS.LT : EF	0.00052	0.00506	0.10	0.919
WEEKS.LT : LF	0.00153	0.00506	0.30	0.763
WEEKS.LT : ET	0.00206	0.00506	0.41	0.686
WEEKSSQ	-0.000585	0.000193	-3.03	0.004
WEEKSSQ.LT : EF	0.000195	0.000273	0.71	0.479
WEEKSSQ.LT : LF	0.000009	0.000273	0.03	0.975
WEEKSSQ.LT : ET	-0.000124	0.000273	-0.45	0.652

APPENDIX 7

REGRESSION OF FEED INTAKE vs TIME (BROKEN STICK MODELS)

*** Summary of analysis ***

	d.f.	s.s.	m.s.	v.r.
Regression	12	8724.7	727.06	43.04
Residual	55	929.1	16.89	
Total	67	9653.8	144.09	
Change	-12	-8724.7	727.06	43.04

Percentage variance accounted for 88.3
Standard error of observations is estimated to be 4.11

*** Estimates of regression coefficients ***

	estimate	s.e.	t(55)	t pr.
Constant	27.32	3.36	8.12	<.001
Z1	6.34	1.07	5.92	<.001
EF : LF	14.58	4.22	3.46	0.001
EF : ET	6.00	4.22	1.42	0.160
EF : LT	14.14	4.22	3.35	0.001
Z1.EF : LF	-2.24	1.39	-1.61	0.114
Z1.EF : ET	-2.38	1.39	-1.71	0.093
Z1.EF : LT	-3.23	1.39	-2.32	0.024
Z2	1.063	0.282	3.78	<.001
Z2.EF : LF	-0.838	0.364	-2.30	0.025
Z2.EF : ET	-1.184	0.364	-3.25	0.002
Z2.EF : LT	-1.201	0.364	-3.30	0.002
Z3	31.62	2.98	10.60	<.001
Constant	41.90	3.36	12.46	<.001
Z1	4.10	1.07	3.83	<.001
LF : EF	-14.58	4.22	-3.46	0.001
LF : ET	-8.58	4.22	-2.04	0.047
LF : LT	-0.44	4.22	-0.10	0.917
Z1.LF : EF	2.24	1.39	1.61	0.114
Z1.LF : ET	-0.14	1.39	-0.10	0.919
Z1.LF : LT	-0.99	1.39	-0.71	0.480
Z2	0.225	0.282	0.80	0.427
Z2.LF : EF	0.838	0.364	2.30	0.025
Z2.LF : ET	-0.346	0.364	-0.95	0.347
Z2.LF : LT	-0.363	0.364	-1.00	0.324
Z3	31.62	2.98	10.60	<.001
Constant	33.32	3.36	9.91	<.001
Z1	3.96	1.07	3.70	<.001
ET : EF	-6.00	4.22	-1.42	0.160
ET : LF	8.58	4.22	2.04	0.047
ET : LT	8.14	4.22	1.93	0.059
Z1.ET : EF	2.38	1.39	1.71	0.093
Z1.ET : LF	0.14	1.39	0.10	0.919
Z1.ET : LT	-0.85	1.39	-0.61	0.545
Z2	-0.120	0.282	-0.43	0.671
Z2.ET : EF	1.184	0.364	3.25	0.002
Z2.ET : LF	0.346	0.364	0.95	0.347
Z2.ET : LT	-0.017	0.364	-0.05	0.963
Z3	31.62	2.98	10.60	<.001
Constant	41.46	3.36	12.33	<.001
Z1	3.11	1.07	2.91	0.005
LT : EF	-14.14	4.22	-3.35	0.001
LT : LF	0.44	4.22	0.10	0.917
LT : ET	-8.14	4.22	-1.93	0.059
Z1.LT : EF	3.23	1.39	2.32	0.024
Z1.LT : LF	0.99	1.39	0.71	0.480
Z1.LT : ET	0.85	1.39	0.61	0.545
Z2	-0.137	0.282	-0.49	0.627
Z2.LT : EF	1.201	0.364	3.30	0.002
Z2.LT : LF	0.363	0.364	1.00	0.324
Z2.LT : ET	0.017	0.364	0.05	0.963
Z3	31.62	2.98	10.60	<.001

REGRESSION OF FEED INTAKE / METABOLIC WEIGHT ($W^{0.75}$) vs TIME (BROKEN STICK MODELS)

*** Summary of analysis ***

	d.f.	s.s.	m.s.	v.r.
Regression	12	0.4537	0.037811	12.08
Residual	55	0.1721	0.003129	
Total	67	0.6258	0.009341	
Change	-12	-0.4537	0.037811	12.08

Percentage variance accounted for 66.5
Standard error of observations is estimated to be 0.0559

*** Estimates of regression coefficients ***

	estimate	s.e.	t(55)	t pr.
Constant	0.4750	0.0458	10.38	<.001
Z1	0.0705	0.0146	4.84	<.001
EF : LF	0.1243	0.0574	2.17	0.035
EF : ET	0.1763	0.0574	3.07	0.003
EF : LT	0.2412	0.0574	4.20	<.001
Z1.EF : LF	-0.0297	0.0189	-1.57	0.122
Z1.EF : ET	-0.0321	0.0189	-1.70	0.096
Z1.EF : LT	-0.0474	0.0189	-2.51	0.015
Z2	0.00035	0.00383	0.09	0.927
Z2.EF : LF	-0.01309	0.00496	-2.64	0.011
Z2.EF : ET	-0.01850	0.00496	-3.73	<.001
Z2.EF : LT	-0.02058	0.00496	-4.15	<.001
Z3	0.3929	0.0406	9.68	<.001
Constant	0.5993	0.0458	13.09	<.001
Z1	0.0408	0.0146	2.80	0.007
LF : EF	-0.1243	0.0574	-2.17	0.035
LF : ET	0.0520	0.0574	0.91	0.369
LF : LT	0.1169	0.0574	2.04	0.046
Z1.LF : EF	0.0297	0.0189	1.57	0.122
Z1.LF : ET	-0.0024	0.0189	-0.13	0.900
Z1.LF : LT	-0.0177	0.0189	-0.94	0.354
Z2	-0.01274	0.00383	-3.32	0.002
Z2.LF : EF	0.01309	0.00496	2.64	0.011
Z2.LF : ET	-0.00542	0.00496	-1.09	0.279
Z2.LF : LT	-0.00749	0.00496	-1.51	0.136
Z3	0.3929	0.0406	9.68	<.001
Constant	0.6513	0.0458	14.23	<.001
Z1	0.0384	0.0146	2.63	0.011
ET : EF	-0.1763	0.0574	-3.07	0.003
ET : LF	-0.0520	0.0574	-0.91	0.369
ET : LT	0.0649	0.0574	1.13	0.263
Z1.ET : EF	0.0321	0.0189	1.70	0.096
Z1.ET : LF	0.0024	0.0189	0.13	0.900
Z1.ET : LT	-0.0153	0.0189	-0.81	0.422
Z2	-0.01815	0.00383	-4.74	<.001
Z2.ET : EF	0.01850	0.00496	3.73	<.001
Z2.ET : LF	0.00542	0.00496	1.09	0.279
Z2.ET : LT	-0.00207	0.00496	-0.42	0.677
Z3	0.3929	0.0406	9.68	<.001
Constant	0.7162	0.0458	15.65	<.001
Z1	0.0231	0.0146	1.58	0.119
LT : EF	-0.2412	0.0574	-4.20	<.001
LT : LF	-0.1169	0.0574	-2.04	0.046
LT : ET	-0.0649	0.0574	-1.13	0.263
Z1.LT : EF	0.0474	0.0189	2.51	0.015
Z1.LT : LF	0.0177	0.0189	0.94	0.354
Z1.LT : ET	0.0153	0.0189	0.81	0.422
Z2	-0.02023	0.00383	-5.28	<.001
Z2.LT : EF	0.02058	0.00496	4.15	<.001
Z2.LT : LF	0.00749	0.00496	1.51	0.136
Z2.LT : ET	0.00207	0.00496	0.42	0.677
Z3	0.3929	0.0406	9.68	<.001

APPENDIX 8

REGRESSION OF LOG OF CUMMULATIVE FEED INTAKE vs LOG OF LIVEWEIGHT

*** Summary of analysis ***

	d.f.	s.s.	m.s.	v.r.
Regression	7	51.075	7.29638	175.46
Residual	56	2.329	0.04158	
Total	63	53.403	0.84767	
Change	-3	-0.953	0.31769	7.64

Percentage variance accounted for 95.1
Standard error of observations is estimated to be 0.204

*** Estimates of regression coefficients ***

	estimate	s.e.	t(56)	t pr.
Constant	-37.18	2.45	-15.19	<.001
LNW1_4	7.411	0.421	17.59	<.001
EF : LF	0.70	3.46	0.20	0.840
EF : ET	12.16	3.01	4.05	<.001
EF : LT	10.35	3.10	3.34	0.002
LNW1_4.EF : LF	-0.265	0.589	-0.45	0.655
LNW1_4.EF : ET	-1.998	0.521	-3.84	<.001
LNW1_4.EF : LT	-1.759	0.534	-3.29	0.002
Constant	-36.48	2.45	-14.91	<.001
LNW1_4	7.147	0.412	17.36	<.001
LF : EF	-0.70	3.46	-0.20	0.840
LF : ET	11.46	3.00	3.81	<.001
LF : LT	9.65	3.10	3.11	0.003
LNW1_4.LF : EF	0.265	0.589	0.45	0.655
LNW1_4.LF : ET	-1.733	0.513	-3.38	0.001
LNW1_4.LF : LT	-1.494	0.527	-2.84	0.006
Constant	-25.02	1.74	-14.36	<.001
LNW1_4	5.413	0.306	17.71	<.001
ET : EF	-12.16	3.01	-4.05	<.001
ET : LF	-11.46	3.00	-3.81	<.001
ET : LT	-1.81	2.58	-0.70	0.487
LNW1_4.ET : EF	1.998	0.521	3.84	<.001
LNW1_4.ET : LF	1.733	0.513	3.38	0.001
LNW1_4.ET : LT	0.239	0.449	0.53	0.596
Constant	-26.83	1.91	-14.08	<.001
LNW1_4	5.652	0.329	17.21	<.001
LT : EF	-10.35	3.10	-3.34	0.002
LT : LF	-9.65	3.10	-3.11	0.003
LT : ET	1.81	2.58	0.70	0.487
LNW1_4.LT : EF	1.759	0.534	3.29	0.002
LNW1_4.LT : LF	1.494	0.527	2.84	0.006
LNW1_4.LT : ET	-0.239	0.449	-0.53	0.596

APPENDIX 9

Net energy avaiable for gain over time

WEEK	EARLY MATURING FAT			LATE MATURING FAT		
	NEg[1] INTAKE (MJ)	NEg[1] INTAKE/ $W^{0.75}$ (MJ)	n	NEg[1] INTAKE (MJ)	NEg[1] INTAKE/ $W^{0.75}$ (MJ)	n
1	212.57	3.32	24	265.53	3.80	24
2	274.35	4.16	24	312.37	4.31	24
3	231.25	3.43	24	321.15	4.31	24
4	384.96	5.53	24	408.74	5.33	24
5	382.66	5.41	24	417.92	5.33	24
6	469.93	6.44	24	543.42	6.67	24
7	452.83	6.06	24	553.85	6.70	24
8	447.08	5.78	24	537.27	6.29	24
9	463.08	5.78	24	539.05	6.16	24
10	406.24	4.96	24	505.29	5.60	24
11	437.47	5.36	24	510.64	5.67	24
12	563.41	6.89	24	525.00	5.74	24
13	515.02	6.26	14	512.52	5.51	23
14	622.94	7.28	14	501.15	5.37	23
15	377.67	4.32	9	456.99	4.74	20
16	543.88	6.26	9	568.71	5.93	20
17	597.34	6.94	9	526.50	5.38	20

[1] = NEg available for production (growth).

Net energy available for gain over time

WEEK	EARLY MATURING THIN			LATE MATURING THIN		
	NEg[1] INTAKE (kg)	NEg[1] INTAKE/ $W^{0.75}$ (kg)	n	NEg[1] INTAKE (kg)	NEg[1] INTAKE/ $W^{0.75}$ (kg)	n
1	206.96	3.76	23	274.96	4.60	24
2	272.67	4.83	23	336.01	5.42	24
3	266.90	4.52	23	330.71	5.14	24
4	387.08	6.36	23	366.14	5.52	24
5	340.77	5.41	23	385.59	5.56	24
6	458.83	6.98	23	526.29	7.36	24
7	493.38	7.18	23	519.66	6.98	24
8	446.65	6.28	23	522.65	6.82	24
9	493.57	6.77	23	515.75	6.57	24
10	393.40	5.28	23	512.20	6.40	24
11	403.75	5.26	23	454.71	5.54	24
12	442.19	5.62	22	503.81	6.01	24
13	419.72	5.26	21	492.87	5.80	24
14	434.62	5.38	21	488.91	5.66	24
15	410.85	4.98	19	467.46	5.27	22
16	435.59	5.30	19	507.06	5.74	22
17	387.26	4.64	19	449.79	5.00	22

[1] = NEg available for production (growth).

APPENDIX 10
REGRESSION OF NET ENERGY AVAILABLE FOR GROWTH (NEg) vs TIME (QUADRATIC MODEL)

*** Summary of analysis ***

	d.f.	s.s.	m.s.	v.r.
Regression	11	488033.	44367.	18.33
Residual	56	135573.	2421.	
Total	67	623606.	9308.	
Change	-3	-5949.	1983.	0.82

Percentage variance accounted for 74.0
Standard error of observations is estimated to be 49.2

*** Estimates of regression coefficients ***

	estimate	s.e.	t(56)	t pr.
Constant	188.9	40.5	4.67	<.001
WEEKS	42.4	10.3	4.10	<.001
EF : LF	33.5	57.2	0.58	0.561
EF : ET	-11.5	57.2	-0.20	0.841
EF : LT	37.9	57.2	0.66	0.511
WEEKS.EF : LF	11.9	14.6	0.81	0.420
WEEKS.EF : ET	9.1	14.6	0.62	0.535
WEEKS.EF : LT	9.0	14.6	0.61	0.542
WEEKSSQ	-1.298	0.559	-2.32	0.024
WEEKSSQ.EF : LF	-0.988	0.790	-1.25	0.216
WEEKSSQ.EF : ET	-1.060	0.790	-1.34	0.185
WEEKSSQ.EF : LT	-0.979	0.790	-1.24	0.221
Constant	222.3	40.5	5.49	<.001
WEEKS	54.3	10.3	5.25	<.001
LF : EF	-33.5	57.2	-0.58	0.561
LF : ET	-45.0	57.2	-0.79	0.435
LF : LT	4.4	57.2	0.08	0.939
WEEKS.LF : EF	-11.9	14.6	-0.81	0.420
WEEKS.LF : ET	-2.7	14.6	-0.19	0.852
WEEKS.LF : LT	-2.9	14.6	-0.20	0.844
WEEKSSQ	-2.286	0.559	-4.09	<.001
WEEKSSQ.LF : EF	0.988	0.790	1.25	0.216
WEEKSSQ.LF : ET	-0.072	0.790	-0.09	0.928
WEEKSSQ.LF : LT	0.009	0.790	0.01	0.990
Constant	177.4	40.5	4.38	<.001
WEEKS	51.6	10.3	4.98	<.001
ET : EF	11.5	57.2	0.20	0.841
ET : LF	45.0	57.2	0.79	0.435
ET : LT	49.4	57.2	0.86	0.392
WEEKS.ET : EF	-9.1	14.6	-0.62	0.535
WEEKS.ET : LF	2.7	14.6	0.19	0.852
WEEKS.ET : LT	-0.2	14.6	-0.01	0.991
WEEKSSQ	-2.358	0.559	-4.22	<.001
WEEKSSQ.ET : EF	1.060	0.790	1.34	0.185
WEEKSSQ.ET : LF	0.072	0.790	0.09	0.928
WEEKSSQ.ET : LT	0.082	0.790	0.10	0.918
Constant	226.7	40.5	5.60	<.001
WEEKS	51.4	10.3	4.97	<.001
LT : EF	-37.9	57.2	-0.66	0.511
LT : LF	-4.4	57.2	-0.08	0.939
LT : ET	-49.4	57.2	-0.86	0.392
WEEKS.LT : EF	-9.0	14.6	-0.61	0.542
WEEKS.LT : LF	2.9	14.6	0.20	0.844
WEEKS.LT : ET	0.2	14.6	0.01	0.991
WEEKSSQ	-2.276	0.559	-4.07	<.001
WEEKSSQ.LT : EF	0.979	0.790	1.24	0.221
WEEKSSQ.LT : LF	-0.009	0.790	-0.01	0.990
WEEKSSQ.LT : ET	-0.082	0.790	-0.10	0.918

REGRESSION OF NET ENERGY AVAILABLE FOR GROWTH (NEg) / METABOLIC WEIGHT ($W^{0.75}$) vs TIME (QUADRATIC MODEL)

*** Summary of analysis ***

	d.f.	s.s.	m.s.	v.r.
Regression	11	31.56	2.8695	6.34
Residual	56	25.34	0.4524	
Total	67	56.90	0.8493	
Change	-3	-0.77	0.2557	0.57

Percentage variance accounted for 46.7
Standard error of observations is estimated to be 0.673

*** Estimates of regression coefficients ***

	estimate	s.e.	t(56)	t pr.
Constant	3.249	0.553	5.87	<.001
WEEKS	0.451	0.141	3.19	0.002
EF : LF	0.264	0.782	0.34	0.737
EF : ET	0.559	0.782	0.71	0.478
EF : LT	1.079	0.782	1.38	0.173
WEEKS.EF : LF	0.065	0.200	0.32	0.748
WEEKS.EF : ET	0.095	0.200	0.47	0.637
WEEKS.EF : LT	0.025	0.200	0.13	0.900
WEEKSSQ	-0.01688	0.00764	-2.21	0.031
WEEKSSQ.EF : LF	-0.0088	0.0108	-0.81	0.419
WEEKSSQ.EF : ET	-0.0137	0.0108	-1.26	0.211
WEEKSSQ.EF : LT	-0.0095	0.0108	-0.88	0.382
Constant	3.512	0.553	6.35	<.001
WEEKS	0.516	0.141	3.65	<.001
LF : EF	-0.264	0.782	-0.34	0.737
LF : ET	0.295	0.782	0.38	0.707
LF : LT	0.816	0.782	1.04	0.302
WEEKS.LF : EF	-0.065	0.200	-0.32	0.748
WEEKS.LF : ET	0.030	0.200	0.15	0.881
WEEKS.LF : LT	-0.039	0.200	-0.20	0.845
WEEKSSQ	-0.02568	0.00764	-3.36	0.001
WEEKSSQ.LF : EF	0.0088	0.0108	0.81	0.419
WEEKSSQ.LF : ET	-0.0049	0.0108	-0.45	0.655
WEEKSSQ.LF : LT	-0.0007	0.0108	-0.07	0.947
Constant	3.808	0.553	6.88	<.001
WEEKS	0.546	0.141	3.86	<.001
ET : EF	-0.559	0.782	-0.71	0.478
ET : LF	-0.295	0.782	-0.38	0.707
ET : LT	0.520	0.782	0.66	0.509
WEEKS.ET : EF	-0.095	0.200	-0.47	0.637
WEEKS.ET : LF	-0.030	0.200	-0.15	0.881
WEEKS.ET : LT	-0.070	0.200	-0.35	0.729
WEEKSSQ	-0.03054	0.00764	-4.00	<.001
WEEKSSQ.ET : EF	0.0137	0.0108	1.26	0.211
WEEKSSQ.ET : LF	0.0049	0.0108	0.45	0.655
WEEKSSQ.ET : LT	0.0041	0.0108	0.38	0.703
Constant	4.328	0.553	7.82	<.001
WEEKS	0.477	0.141	3.37	0.001
LT : EF	-1.079	0.782	-1.38	0.173
LT : LF	-0.816	0.782	-1.04	0.302
LT : ET	-0.520	0.782	-0.66	0.509
WEEKS.LT : EF	-0.025	0.200	-0.13	0.900
WEEKS.LT : LF	0.039	0.200	0.20	0.845
WEEKS.LT : ET	0.070	0.200	0.35	0.729
WEEKSSQ	-0.02640	0.00764	-3.46	0.001
WEEKSSQ.LT : EF	0.0095	0.0108	0.88	0.382
WEEKSSQ.LT : LF	0.0007	0.0108	0.07	0.947
WEEKSSQ.LT : ET	-0.0041	0.0108	-0.38	0.703

APPENDIX 11
REGRESSION OF NET ENERGY AVAILABLE FOR GROWTH (NEg) vs TIME (BROKEN STICK MODELS)

*** Summary of analysis ***

	d.f.	s.s.	m.s.	v.r.
Regression	12	535914.	44660.	28.01
Residual	55	87692.	1594.	
Total	67	623606.	9308.	
Change	-12	-535914.	44660.	28.01

Percentage variance accounted for 82.9
Standard error of observations is estimated to be 39.9

*** Estimates of regression coefficients ***

	estimate	s.e.	t(55)	t pr.
Constant	120.3	32.7	3.68	<.001
Z1	56.4	10.4	5.42	<.001
EF : LF	125.5	41.0	3.06	0.003
EF : ET	78.4	41.0	1.91	0.061
EF : LT	145.1	41.0	3.54	<.001
Z1.EF : LF	-22.0	13.5	-1.63	0.109
Z1.EF : ET	-23.3	13.5	-1.72	0.091
Z1.EF : LT	-31.9	13.5	-2.36	0.022
Z2	7.40	2.74	2.71	0.009
Z2.EF : LF	-8.55	3.54	-2.42	0.019
Z2.EF : ET	-12.12	3.54	-3.43	0.001
Z2.EF : LT	-12.44	3.54	-3.52	<.001
Z3	289.1	29.0	9.97	<.001
Constant	245.8	32.7	7.52	<.001
Z1	34.4	10.4	3.30	0.002
LF : EF	-125.5	41.0	-3.06	0.003
LF : ET	-47.1	41.0	-1.15	0.255
LF : LT	19.6	41.0	0.48	0.635
Z1.LF : EF	22.0	13.5	1.63	0.109
Z1.LF : ET	-1.2	13.5	-0.09	0.929
Z1.LF : LT	-9.8	13.5	-0.73	0.471
Z2	-1.15	2.74	-0.42	0.677
Z2.LF : EF	8.55	3.54	2.42	0.019
Z2.LF : ET	-3.57	3.54	-1.01	0.317
Z2.LF : LT	-3.89	3.54	-1.10	0.277
Z3	289.1	29.0	9.97	<.001
Constant	198.7	32.7	6.08	<.001
Z1	33.2	10.4	3.19	0.002
ET : EF	-78.4	41.0	-1.91	0.061
ET : LF	47.1	41.0	1.15	0.255
ET : LT	66.7	41.0	1.63	0.109
Z1.ET : EF	23.3	13.5	1.72	0.091
Z1.ET : LF	1.2	13.5	0.09	0.929
Z1.ET : LT	-8.6	13.5	-0.64	0.527
Z2	-4.72	2.74	-1.73	0.090
Z2.ET : EF	12.12	3.54	3.43	0.001
Z2.ET : LF	3.57	3.54	1.01	0.317
Z2.ET : LT	-0.32	3.54	-0.09	0.929
Z3	289.1	29.0	9.97	<.001
Constant	265.4	32.7	8.12	<.001
Z1	24.6	10.4	2.36	0.022
LT : EF	-145.1	41.0	-3.54	<.001
LT : LF	-19.6	41.0	-0.48	0.635
LT : ET	-66.7	41.0	-1.63	0.109
Z1.LT : EF	31.9	13.5	2.36	0.022
Z1.LT : LF	9.8	13.5	0.73	0.471
Z1.LT : ET	8.6	13.5	0.64	0.527
Z2	-5.04	2.74	-1.84	0.071
Z2.LT : EF	12.44	3.54	3.52	<.001
Z2.LT : LF	3.89	3.54	1.10	0.277
Z2.LT : ET	0.32	3.54	0.09	0.929
Z3	289.1	29.0	9.97	<.001

REGRESSION OF NET ENERGY AVAILABLE FOR GROWTH (NEg) / METABOLIC WEIGHT ($W^{0.75}$) vs TIME (BROKEN STICK MODELS)

*** Summary of analysis ***

	d.f.	s.s.	m.s.	v.r.
Regression	12	41.25	3.4377	12.08
Residual	55	15.65	0.2845	
Total	67	56.90	0.8493	
Change	-12	-41.25	3.4377	12.08

Percentage variance accounted for 66.5
Standard error of observations is estimated to be 0.533

*** Estimates of regression coefficients ***

	estimate	s.e.	t(55)	t pr.
Constant	2.273	0.436	5.21	<.001
Z1	0.673	0.139	4.84	<.001
EF : LF	1.185	0.547	2.17	0.035
EF : ET	1.681	0.547	3.07	0.003
EF : LT	2.300	0.547	4.20	<.001
Z1.EF : LF	-0.284	0.181	-1.57	0.122
Z1.EF : ET	-0.306	0.181	-1.70	0.096
Z1.EF : LT	-0.452	0.181	-2.51	0.015
Z2	0.0033	0.0365	0.09	0.927
Z2.EF : LF	-0.1248	0.0473	-2.64	0.011
Z2.EF : ET	-0.1764	0.0473	-3.73	<.001
Z2.EF : LT	-0.1962	0.0473	-4.15	<.001
Z3	3.747	0.387	9.68	<.001
Constant	3.459	0.436	7.92	<.001
Z1	0.389	0.139	2.80	0.007
LF : EF	-1.185	0.547	-2.17	0.035
LF : ET	0.496	0.547	0.91	0.369
LF : LT	1.115	0.547	2.04	0.046
Z1.LF : EF	0.284	0.181	1.57	0.122
Z1.LF : ET	-0.023	0.181	-0.13	0.900
Z1.LF : LT	-0.169	0.181	-0.94	0.354
Z2	-0.1214	0.0365	-3.32	0.002
Z2.LF : EF	0.1248	0.0473	2.64	0.011
Z2.LF : ET	-0.0517	0.0473	-1.09	0.279
Z2.LF : LT	-0.0714	0.0473	-1.51	0.136
Z3	3.747	0.387	9.68	<.001
Constant	3.955	0.436	9.06	<.001
Z1	0.366	0.139	2.63	0.011
ET : EF	-1.681	0.547	-3.07	0.003
ET : LF	-0.496	0.547	-0.91	0.369
ET : LT	0.619	0.547	1.13	0.263
Z1.ET : EF	0.306	0.181	1.70	0.096
Z1.ET : LF	0.023	0.181	0.13	0.900
Z1.ET : LT	-0.146	0.181	-0.81	0.422
Z2	-0.1731	0.0365	-4.74	<.001
Z2.ET : EF	0.1764	0.0473	3.73	<.001
Z2.ET : LF	0.0517	0.0473	1.09	0.279
Z2.ET : LT	-0.0198	0.0473	-0.42	0.677
Z3	3.747	0.387	9.68	<.001
Constant	4.573	0.436	10.48	<.001
Z1	0.220	0.139	1.58	0.119
LT : EF	-2.300	0.547	-4.20	<.001
LT : LF	-1.115	0.547	-2.04	0.046
LT : ET	-0.619	0.547	-1.13	0.263
Z1.LT : EF	0.452	0.181	2.51	0.015
Z1.LT : LF	0.169	0.181	0.94	0.354
Z1.LT : ET	0.146	0.181	0.81	0.422
Z2	-0.1929	0.0365	-5.28	<.001
Z2.LT : EF	0.1962	0.0473	4.15	<.001
Z2.LT : LF	0.0714	0.0473	1.51	0.136
Z2.LT : ET	0.0198	0.0473	0.42	0.677
Z3	3.747	0.387	9.68	<.001

APPENDIX 12

REGRESSION OF LOG OF CUMMULATIVE NET ENERGY AVAILABLE FOR GROWTH (NEg) vs LOG OF LIVEWEIGHT

*** Summary of analysis ***

	d.f.	s.s.	m.s.	v.r.
Regression	7	55.517	7.93106	169.96
Residual	56	2.613	0.04666	
Total	63	58.131	0.92271	
Change	-3	-1.160	0.38675	8.29

Percentage variance accounted for 94.9
Standard error of observations is estimated to be 0.216

*** Estimates of regression coefficients ***

	estimate	s.e.	t(56)	t pr.
Constant	-37.80	2.59	-14.57	<.001
LNW1_4	7.838	0.446	17.56	<.001
EF : LF	-1.33	3.67	0.36	0.717
EF : ET	13.43	3.18	4.22	<.001
EF : LT	12.02	3.29	3.66	<.001
LNW1_4.EF : LF	-0.378	0.624	-0.61	0.547
LNW1_4.EF : ET	-2.207	0.552	-4.00	<.001
LNW1_4.EF : LT	-2.036	0.566	-3.60	<.001
Constant	-36.46	2.59	-14.07	<.001
LNW1_4	7.460	0.436	17.11	<.001
LF : EF	-1.33	3.67	-0.36	0.717
LF : ET	12.10	3.18	3.80	<.001
LF : LT	10.68	3.29	3.25	0.002
LNW1_4.LF : EF	0.378	0.624	0.61	0.547
LNW1_4.LF : ET	-1.829	0.543	-3.37	0.001
LNW1_4.LF : LT	-1.658	0.558	-2.97	0.004
Constant	-24.37	1.85	-13.20	<.001
LNW1_4	5.631	0.324	17.39	<.001
ET : EF	-13.43	3.18	-4.22	<.001
ET : LF	-12.10	3.18	-3.80	<.001
ET : LT	-1.41	2.74	-0.52	0.608
LNW1_4.ET : EF	2.207	0.552	4.00	<.001
LNW1_4.ET : LF	1.829	0.543	3.37	0.001
LNW1_4.ET : LT	0.171	0.475	0.36	0.720
Constant	-25.78	2.02	-12.77	<.001
LNW1_4	5.802	0.348	16.67	<.001
LT : EF	-12.02	3.29	-3.66	<.001
LT : LF	-10.68	3.29	-3.25	0.002
LT : ET	1.41	2.74	0.52	0.608
LNW1_4.LT : EF	2.036	0.566	3.60	<.001
LNW1_4.LT : LF	1.658	0.558	2.97	0.004
LNW1_4.LT : ET	-0.171	0.475	-0.36	0.720

APPENDIX 13

Height over time

	EARLY MATURING FAT			LATE MATURING FAT		
MEASUREMENT	HEIGHT (cm)	CV%	n	HEIGHT (cm)	CV%	n
1	109.17	2.89	24	117.21	2.21	24
2	110.83	2.93	24	118.58	2.84	24
3	112.08	2.56	24	119.67	2.53	24
4	112.04	2.87	24	120.29	2.76	24
5	113.62	2.83	24	121.42	2.87	24
6	115.29	2.46	14	122.74	3.07	23
7	117.33	2.95	9	124.90	2.61	20
8	119.00	2.32	6	127.21	2.47	14

Height over time

	EARLY MATURING THIN			LATE MATURING THIN		
MEASUREMENT	HEIGHT (cm)	CV%	n	HEIGHT (cm)	CV%	n
1	107.96	3.07	23	110.38	2.87	24
2	109.39	3.30	23	112.33	3.20	24
3	111.61	2.89	23	114.42	3.31	24
4	112.35	3.20	23	115.21	3.29	24
5	114.00	2.75	22	118.00	3.27	24
6	115.62	3.06	21	119.04	3.93	24
7	116.32	2.66	19	119.95	3.64	22
8	118.08	2.64	13	122.00	3.31	20

REGRESSION OF HEIGHT vs TIME
*** Summary of analysis ***

	d.f.	s.s.	m.s.	v.r.
Regression	11	698.916	63.5378	347.51
Residual	20	3.657	0.1828	
Total	31	702.573	22.6636	
Change	-3	-3.281	1.0936	5.98

Percentage variance accounted for 99.2
Standard error of observations is estimated to be 0.428

	estimate	s.e.	t(20)	t pr.
Constant	108.973	0.597	182.67	<.001
TIME	0.542	0.304	1.78	0.090
EF : LF	8.200	0.844	9.72	<.001
EF : ET	-2.681	0.844	-3.18	0.005
EF : LT	-0.520	0.844	-0.62	0.544
TIME.EF : LF	-0.210	0.430	-0.49	0.631
TIME.EF : ET	1.170	0.430	2.72	0.013
TIME.EF : LT	1.481	0.430	3.44	0.003
TIMESQ	0.0886	0.0330	2.69	0.014
TIMESQ.EF : LF	0.0226	0.0467	0.48	0.634
TIMESQ.EF : ET	-0.1213	0.0467	-2.60	0.017
TIMESQ.EF : LT	-0.1333	0.0467	-2.86	0.010
Constant = 2	117.172	0.597	196.41	<.001
TIME	0.332	0.304	1.09	0.288
LF : EF	-8.200	0.844	-9.72	<.001
LF : ET	-10.880	0.844	-12.90	<.001
LF : LT	-8.720	0.844	-10.34	<.001
TIME.LF : EF	0.210	0.430	0.49	0.631
TIME.LF : ET	1.380	0.430	3.21	0.004
TIME.LF : LT	1.691	0.430	3.93	<.001
TIMESQ	0.1112	0.0330	3.37	0.003
TIMESQ.LF : EF	-0.0226	0.0467	-0.48	0.634
TIMESQ.LF : ET	-0.1438	0.0467	-3.08	0.006
TIMESQ.LF : LT	-0.1559	0.0467	-3.34	0.003
Constant = 3	106.292	0.597	178.18	<.001
TIME	1.712	0.304	5.63	<.001
ET : EF	2.681	0.844	3.18	0.005
ET : LF	10.880	0.844	12.90	<.001
ET : LT	2.160	0.844	2.56	0.019
TIME.ET : EF	-1.170	0.430	-2.72	0.013
TIME.ET : LF	-1.380	0.430	-3.21	0.004
TIME.ET : LT	0.311	0.430	0.72	0.478
TIMESQ	-0.0326	0.0330	-0.99	0.334
TIMESQ.ET : EF	0.1213	0.0467	2.60	0.017
TIMESQ.ET : LF	0.1438	0.0467	3.08	0.006
TIMESQ.ET : LT	-0.0120	0.0467	-0.26	0.799
Constant = 4	108.453	0.597	181.80	<.001
TIME	2.023	0.304	6.65	<.001
LT : EF	0.520	0.844	0.62	0.544
LT : LF	8.720	0.844	10.34	<.001
LT : ET	-2.160	0.844	-2.56	0.019
TIME.LT : EF	-1.481	0.430	-3.44	0.003
TIME.LT : LF	-1.691	0.430	-3.93	<.001
TIME.LT : ET	-0.311	0.430	0.72	0.478
TIMESQ	-0.0447	0.0330	-1.35	0.191
TIMESQ.LT : EF	0.1333	0.0467	2.86	0.010
TIMESQ.LT : LF	0.1559	0.0467	3.34	0.003
TIMESQ.LT : ET	0.0120	0.0467	0.26	0.799

APPENDIX 14

Eye-muscle diameter over time

MEASUREMENT	EARLY MATURING FAT			LATE MATURING FAT		
	EYE MUSCLE DIAMETER (cm)	CV%	n	EYE MUSCLE DIAMETER (cm)	CV%	n
1	1.84	9.78	12	1.78	8.99	12
2	1.94	7.22	12	1.95	5.64	12
3	2.02	7.43	12	2.03	7.39	12
4	2.10	7.14	12	2.11	6.16	12
5	2.18	8.72	12	2.12	7.55	12
6	2.23	5.38	12	2.36	5.51	12
7	2.33	9.87	4	2.42	4.96	11
8	2.33	9.87	4	2.46	4.47	11

Eye-muscle diameter over time

MEASUREMENT	EARLY MATURING THIN			LATE MATURING THIN		
	EYE MUSCLE DIAMETER (cm)	CV%	n	EYE MUSCLE DIAMETER (cm)	CV%	n
1	1.62	9.88	12	1.65	7.27	12
2	1.70	8.24	12	1.83	8.74	12
3	1.85	9.19	12	1.93	8.81	12
4	2.04	5.88	12	2.08	9.62	12
5	2.00	8.50	12	2.12	10.80	12
6	2.21	9.95	12	2.16	8.33	12
7	2.27	8.37	10	2.28	6.14	12
8	2.30	7.39	10	2.30	6.96	12

REGRESSION OF EYE MUSCLE DIAMETER vs TIME
*** Summary of analysis ***

	d.f.	s.s.	m.s.	v.r.
Regression	11	1.53883	0.139894	81.24
Residual	20	0.03444	0.001722	
Total	31	1.57327	0.050751	
Change	-3	-0.00461	0.001535	0.89

Percentage variance accounted for 96.6
Standard error of observations is estimated to be 0.0415

*** Estimates of regression coefficients ***

	estimate	s.e.	t(20)	t pr.
Constant	1.7393	0.0579	30.04	<.001
TIME	0.1071	0.0295	3.63	0.002
EF : LF	-0.0414	0.0819	-0.51	0.619
EF : ET	-0.2862	0.0819	-3.50	0.002
EF : LT	-0.2325	0.0819	-2.84	0.010
TIME.EF : LF	0.0035	0.0417	0.08	0.934
TIME.EF : ET	0.0453	0.0417	1.09	0.290
TIME.EF : LT	0.0618	0.0417	1.48	0.154
TIMESQ	-0.00382	0.00320	-1.19	0.247
TIMESQ.EF : LF	0.00221	0.00453	0.49	0.630
TIMESQ.EF : ET	-0.00170	0.00453	-0.37	0.712
TIMESQ.EF : LT	-0.00496	0.00453	-1.09	0.287
Constant	1.6979	0.0579	29.33	<.001
TIME	0.1106	0.0295	3.75	0.001
LF : EF	0.0414	0.0819	0.51	0.619
LF : ET	-0.2448	0.0819	-2.99	0.007
LF : LT	-0.1911	0.0819	-2.33	0.030
TIME.LF : EF	-0.0035	0.0417	-0.08	0.934
TIME.LF : ET	0.0418	0.0417	1.00	0.328
TIME.LF : LT	0.0583	0.0417	1.40	0.178
TIMESQ	-0.00160	0.00320	-0.50	0.622
TIMESQ.LF : EF	-0.00221	0.00453	-0.49	0.630
TIMESQ.LF : ET	-0.00391	0.00453	-0.86	0.398
TIMESQ.LF : LT	-0.00717	0.00453	-1.58	0.129
Constant	1.4531	0.0579	25.10	<.001
TIME	0.1524	0.0295	5.16	<.001
ET : EF	0.2862	0.0819	3.50	0.002
ET : LF	0.2448	0.0819	2.99	0.007
ET : LT	0.0537	0.0819	0.66	0.519
TIME.ET : EF	-0.0453	0.0417	-1.09	0.290
TIME.ET : LF	-0.0418	0.0417	-1.00	0.328
TIME.ET : LT	0.0165	0.0417	0.39	0.697
TIMESQ	-0.00551	0.00320	-1.72	0.101
TIMESQ.ET : EF	0.00170	0.00453	0.37	0.712
TIMESQ.ET : LF	0.00391	0.00453	0.86	0.398
TIMESQ.ET : LT	-0.00326	0.00453	-0.72	0.480
Constant	1.5068	0.0579	26.03	<.001
TIME	0.1689	0.0295	5.72	<.001
LT : EF	0.2325	0.0819	2.84	0.010
LT : LF	0.1911	0.0819	2.33	0.030
LT : ET	-0.0537	0.0819	-0.66	0.519
TIME.LT : EF	-0.0618	0.0417	-1.48	0.154
TIME.LT : LF	-0.0583	0.0417	-1.40	0.178
TIME.LT : ET	-0.0165	0.0417	-0.39	0.697
TIMESQ	-0.00877	0.00320	-2.74	0.013
TIMESQ.LT : EF	0.00496	0.00453	1.09	0.287
TIMESQ.LT : LF	0.00717	0.00453	1.58	0.129
TIMESQ.LT : ET	0.00326	0.00453	0.72	0.480