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A feeding trial was conducted to establish the dietary crude protein requirement for optimum growth and feed efficiency of *Callipyge* (CLPG)lambs. Thirty-two CLPG and twenty-nine half-sibling normal lambs were stratified by weight and randomly assigned to one of four diets containing 9.9, 13.5, 16.7, and 19.9% CP. Diets were offered *ad libitum* and contained 85% concentrate and 15% alfalfa hay. Data were analyzed using the GLM procedures of SAS to compare the effects of genotype, diet, and genotype*diet on performance characteristics of the lambs. Dry matter intake, ADG, and feed efficiency were similar ($P > .05$) between genotypes and diets. All visceral organs of CLPG lambs were lighter ($P < .02$) than those of normal lambs. Higher leg scores ($P = .00$), larger loin eye areas ($P = .00$) and higher carcass protein ($P = .00$) of CLPG lambs provided evidence that CLPG lambs deposited more protein compared with normal lambs. In this study, superior feed efficiency and ADG for lambs expressing the CLPG gene were not found.

INFLUENCE OF DIETARY CRUDE PROTEIN LEVELS ON THE PERFORMANCE BY NORMAL AND *CALLIPYGE* LAMBS

by

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CHAPTER I

INTRODUCTION

In recent years, the major aim in meat animal production has been to increase the lean content of the carcass because of increasing incidence of health problems associated with excessive consumption of animal fat. Production of leaner meat may be accomplished through genetic selection and manipulation with well-formulated diets including an appropriate level and quality of protein and other nutrients.

Muscle hypertrophy or double muscling (DM) has been recognized as a genetic disorder in cattle for several years (Cully, 1807, as cited by Oliver and Cartwright, 1968). Double-muscled cattle have been reported to excel in carcass characteristics mainly as a result of increased musculature and freedom from subcutaneous and intermuscular fat (Kidwell et al., 1952). More recently, however, considerable attention has been given to lambs expressing the *Callipyge* gene. The presence of the gene results in muscle hypertrophy particularly in the rack, loin and hind legs. Jackson (1993) reported lambs expressing the *Callipyge* gene have superior feed efficiency, higher dressing percentage, and less carcass fat than normal lambs.

The muscle hypertrophy condition was first observed in a Dorset sheep by a farmer in Oklahoma and has been extensively studied in recent years. The muscle hypertrophy condition is not noticeable in newborn lambs and becomes apparent at about three to four weeks of age.

Currently recommended crude protein requirements for growing lambs by the National Research Council (1985) may fail to meet the requirements for these heavily muscled sheep. Increased synthesis of muscle by CLPG lambs may result from either a higher rate of muscle synthesis which may entail more dietary crude protein or lower degradation rate. The objective of this trial, therefore, was to establish the dietary crude protein requirements of optimum growth and feed efficiency of *Callipyge* lambs.

CHAPTER II

LITERATURE REVIEW

Protein requirements

Proteins are found in all living cells where they play a vital role in all phases of activity that constitute the life of the cell. This is more importantly so in young fast-growing animals as the major part of growth will be the retention of protein (Tamminga, 1982).

Crude protein (CP) requirements of growing lambs are recommended by the National Research Council (NRC) (1985). Hinds et al. (1964) and Hinds et al. (1965) reported the CP requirements of early weaned growing lambs was 17.7%. However, based on differences in the extent of degradation of dietary protein in the rumen and subsequent amino acid absorption in the lower intestinal tract, other researchers (Glimp et al., 1967; and Braman, 1972;) found that 17.7% CP is equal to approximately 12% dietary CP with diets containing heat-treated soybean meal. Protein requirements of growing lambs may also be expressed as a function of body weight, body weight gain and digestibility of the protein in the ration (Preston, 1966). Preston (1966) expressed these relationships as follows:

$$CP = 4.88\ W_{kg}^{0.75}\ (1 + 6.64\ G) \quad r = 0.86$$

$$DP = 2.79\ W_{kg}^{0.75}\ (1 + 6.02\ G) \quad r = 0.84$$

where CP and DP are grams of crude and digestible protein per day, respectively, W is the body weight and G is the daily rate of gain, both in kilograms.

Tissues of sheep, like those of other ruminant and non-ruminant animals, require the same exogenous source of essential amino acids (EAA) (Black et al., 1957; and Downes, 1961). Quantitative requirements for EAA have been estimated by several researchers (Hutton and Annison, 1972; Armstrong and Annison, 1973; Owens et al., 1973; and Bergen, 1980). Assessment of the quantitative requirement of EAA for ruminant animals, however, is difficult because the supply of EAA absorbed in the small intestine originates from dietary protein escaped from ruminal degradation and microbial protein synthesized in the rumen by rumen microorganisms. Amino acid utilization in various functions may also result in changes for quantitative needs of EAA. Reis and Schinckel (1963) reported increased wool growth in lambs in response to increasing the postruminal supply of sulphur-containing amino acids. In addition, growth rate has a big influence on the EAA requirements, being higher for animals gaining weight at higher rates (Fenderson and Bergen, 1975; and Towns and Bergen, 1979).

It may be expected that, when the diet contains a large proportion of ruminally degradable protein, the composition of amino acids reaching the small intestine will be quite similar to that of microbial protein. Storm and Orskov (1984) found that methionine and lysine are the most limiting AA in lambs when microbial protein served as the only protein source. These authors also stated that arginine and histidine are also potentially limiting. Lysine has been reported to be first-limiting when corn-based diets are fed to steers (Burris et al., 1976; and Titgemeyer et al., 1988). This may be because corn is a poor source of lysine. Several EAA are also considered to be colimiting for growth of ruminants, such that maximal response is achieved when whole proteins are postruminally supplied rather than when one or two critical AA are provided (Chalupa et al., 1973; Chalupa and Chandler, 1975; and Chalupa and Scott, 1976).

Protein supply

In ruminant animals, unlike other species, amino acids absorbed from small intestine are a combination of microbial protein synthesized in the rumen by rumen microorganisms, protected or undegraded dietary proteins (UDP) that escape from ruminal degradation, and endogenous secretions. Amino acid composition of dietary protein reaching the small intestine can be altered through a variety of chemical and physical treatments (Glimp et al., 1967; Chalupa, 1973; Amos et al., 1974; Fahmy et al., 1992; Zinn and Owens, 1993; and

Cosby and Stanson, 1995).

Microbial protein. The microbial population of the rumen is composed of bacteria, protozoa and fungi (Orpin and Joblin, 1989; Stewart and Bryant, 1989; and Williams and Coleman, 1989). During ruminal digestion, microbes synthesize considerable amounts of microbial crude protein (MCP). Hungate (1966) stated that microbial material synthesized in the rumen contains 65.5% CP. Digestibility and net protein utilization (NPU) are higher for ruminal protozoa than for ruminal bacteria. True digestibility, biological value and NPU are 74, 81 and 60, respectively for rumen bacteria and 90, 80 and 73, respectively for rumen protozoa (McNaught et al., 1954; and Bergen et al., 1968a). The bacteria are the dominant microbial group in the rumen and MCP synthesis and outflow depend primarily upon bacteria (Bergen and Yokoyoma, 1977).

Although protozoa are important components of protein breakdown in the rumen, they make only a minor contribution to postruminal protein supply since protozoa are selectively retained in the rumen (John and Ulyatt, 1984; and Veira, 1986). Protozoa as bacterial predators can play a key role in the turnover of bacterial protein (Leng and Nolan, 1984). Extensive intraruminal N recycling (e.g., ammonia to microbial nitrogenous compounds to ammonia) is achieved by protozoa resulting in efficient microbial protein production

(Coleman and Sandfort, 1979; and Wallace and McPherson, 1987).

The function of the fungi is not clear (Bauchop, 1979). Highly lignified fiber diets fed animals resulted in fungi accounting for 8% of the whole microbial population in the rumen (Citron et al., 1987), but when animals were fed diets rich in concentrates fungi numbers decrease (Fonty et al., 1987).

The bulk amino acid composition of microbial protein remains remarkably constant under various dietary conditions (Weller, 1957; Purser and Buechler, 1966; Schelling, Hinds and Hatfield, 1967; and Bergen et al., 1968b). In several production instances, such as rapidly growing, young ruminants and ruminants at peak lactation, microbial protein synthesized plus the escape of dietary protein may not meet the animal's protein requirement to provide optimal performance and feed efficiency (Satter et al., 1977; Huber and Kung, 1981; and Ensminger et al., 1990a).

By-pass protein. Bypass protein also called protected or escaped protein or undegraded dietary protein (UDP) is the feed protein which escapes digestion in the rumen and passes into the lower digestive tract where it is digested and absorbed. Escaped protein may be beneficial, since it will be utilized efficiently in postruminal digestion. Production of meat, milk and wool in ruminant animals is improved when feeds low in fiber and high in protein which escapes rumen fermentation and is digested post-ruminally are fed (Little and Mitchell, 1967; Orskov et al., 1970; Hedde and Knox, 1974; and Clark, 1975).

Several methods have been developed to protect the dietary protein from ruminal degradation. Heat may be applied to protein sources creating cross-linkages of free amino groups within and between proteins, these cross-linkages reduce the surface of the protein which comes in contact with enzymes resulting in blocking the site of enzymatic attack. Increasing animal performance by heat treatment of proteins has been reported by several researchers mainly as a result of decreasing ruminal degradation of proteins (Goering and Waldo 1974; and Cosby and Stanson, 1995).

Several chemical agents including aldehydes (Ferguson, 1971; and Hatfield, 1973), tanning materials (Leroy et al., 1970; and Hatfield, 1973), and other chemicals (Miller, 1972; Miller, 1973; and Wildi and Miller, 1973) form reversible cross linkages with amino and amide groups which decrease solubility of proteins at the pH of the rumen. In the acidic abomasum, many of these linkages are destroyed and proteins become available for enzymatic attack.

Encapsulation of amino acids is also used to protect free amino acids from ruminal degradation (Broderick et al., 1970; and Grass and Unangst, 1972). In addition, lipid (fat) treatment protects protein from ruminal degradation by forming emulsions with proteins. In this system, proteins emulsified with fat are treated with formaldehyde in order to form formaldehyde linkages to proteins which cannot be digested in the rumen. This protein protected fat system may also be applied to alter the ratio of the saturated to unsaturated fatty acids in beef fat and in butterfat (Ensminger et al., 1990b). More methods other than mentioned above have been reported to protect proteins from ruminal degradation.

Endogenous protein sources. Endogenous N sources consist of microbial products, sloughed tissues, metabolic endproducts and enzymes. Endogenous contribution from the abomasum and small intestine have been estimated to be .04% of dry matter consumed (Tamminga, 1979).

Protein degradation

Protein degradation by rumen microorganism. It has been estimated that as little as 40% or as much as 80% of dietary protein is degraded in the rumen and converted into microbial protein (Smith, 1969; Lewis and Swan, 1971; Leng, 1973; Hogan, 1974; Satter and Roffler, 1974; and Smith et al., 1974). Rumen bacteria are responsible for more than 50% of the proteolytic activity in the rumen (Warner, 1956; and Annison, 1956). On diets containing roughage protozoa activity can be 10-20% of total proteolytic activity (Nugent and Mangan, 1981).

Dietary protein degradation includes proteolysis and deamination. Many of the proteins in the diet are hydrolyzed (i.e., proteolysis) into peptides and amino acids by rumen micro-organisms particularly by species of *Bacteroides*, *Butyrivibrio*, and *Selenomonas* being more potent proteolytic rumen bacteria. Some amino acids are degraded further (i.e., deamination) to branched chain fatty acids, volatile fatty acids (VFA), ammonia and carbon dioxide. Hungate (1966) stated that valine is degraded into the branched chain fatty acid, isobutyric acid and ammonia, and proline can be broken down into valeric acid and ammonia. The peptides and much of the ammonia are used for the synthesis of microbial proteins in the rumen. Ammonia can also be supplied as effectively and more economically by non-protein nitrogen (NPN) such as urea (Chalupa, 1972). The growth of rumen organisms may be slowed when the rumen ammonia concentration is low as is the case of protein deficient diets or diets containing protein components resistant to degradation (McDonald et al., 1981). The supply of energy, sulphur and nitrogen are probably the most limiting factors in the synthesis of microbial protein (Thomas and Rook, 1981).

The conversion of the dietary protein into microbial protein can be wasteful. When the diet contains readily digestible proteins in large quantities or excess supplies of NPN, deamination will be very rapid and the ammonia released will not be completely trapped by the microorganism for amino acid synthesis. The excess ammonia is absorbed from the rumen and converted to urea by the liver and may be partly returned back to the rumen via saliva or excreted in the urine and thus wasted (Hibbitt, 1988). Ammonia toxicity is also an often consequence of feeding diets containing high amounts of readily digestible protein or excess NPN (Word et al., 1969).

The optimum pH for proteolysis and deamination has been determined to be between 6 and 7 (Blackburn and Hobson, 1960; Henderick, 1962; and Lewis and Emery, 1962). Deamination stops at a pH higher than 7.2 (Chalmers, 1969), while at pH below 4.5 deamination is quite low (Lewis and Emery, 1962).

Degradability is positively correlated to solubility (McDonald and Hall, 1957). A more soluble protein, however, does not always mean a more degradable protein. Soluble albumin has been reported to be relatively slowly degraded (Annison, 1956). Solubility of feed protein depends partly upon the relative amount of soluble albumins and globulins versus less soluble prolamins and gluteins (Tamminga, 1979). Wohlt et al. (1976) reported that feed proteins containing large amounts of albumin and globulin protein have a higher protein solubility compared with feed proteins containing prolamin and glutein as major protein fractions. It has been also reported that plant proteins are generally degraded more completely compared with animal proteins (Nolan, 1993).

Protein structure in feeds and rumen conditions influence rate and extent of protein degradation. The number of disulphide cross-linkages and the nature and extent of secondary and tertiary structure are closely associated with lower degradation rates (Mahadevan et al., 1980). Protease and peptidase accessibility to the surface of proteins may be decreased in the presence of lipids and other water-insoluble substances. Microbial cell wall binding to the protein may also be reduced in the absence of metal co-factors or appropriate pH or enzymes (Nolan, 1993).

The extent of dietary protein degradation in the forestomach of ruminants is mainly affected by rumen retention time. Hungate (1966) reported ruminal retention times of sheep to be between .8 to 2.2 days. Level of feed intake and particle size of dietary ingredients are the determining factors for rumen retention time (Hungate, 1966; and Church, 1970). Decreased degradability of dietary protein has been observed at higher levels of feed intake (Hvelplund et al., 1976; and Tamminga et al., 1979). Increasing intake also results in increased particle passage from the reticulo-rumen and decreased colonal-cecal retention time in ruminants (Colucci et al., 1982; and Eliman and Orskov, 1985). Microbial digestion and microbial access to feed particles increase by a factor of two when the diameter of a spherical particle is reduced by half (Owens and Goetsch, 1993).

Protein digestion by the host. The dietary proteins which are not degraded (i.e., bypass protein) in the rumen along with microbial protein synthesized in the rumen are subject to digestion by the host's enzymes in the abomasum and small intestine. Digestion of protein begins with pepsin secreted in the gastric juice, followed by proteolytic enzymes from the pancreas and the mucosa of the small intestine (Fauconneau and Michel, 1970).

The abomasum which is analogous to the gastric stomach of non-ruminants contains fundic and pyloric regions as secretory tissues. In the fundic mucosa parietal cells secrete HCl whereas chief cells are responsible for secretion of pepsinogen the inactive zymogen form of pepsin. Pepsinogen is activated to pepsin by H^+. The secretion of the pylorus is mucous exhibiting low peptic activates (Merchen, 1993). The initial step in protein digestion is its denaturation at acid pH (2 to 3) in the abomasum as a result of contact with gastric HCl. Pepsin is an endopeptidase hydrolyzing peptide bonds within the main polypeptide structure rather than adjacent to amino or carboxyl terminal residues (i.e., exopeptidase). Pepsin splits denatured protein into large polypeptide derivates and is specific for peptide bonds formed by aromatic amino acids (phenylalanine, tyrosine and tryptophan) or dicarboxylic amino acids (eg, glutamate) (Mayes, 1996).

Basic rules in the processes of digestion and absorption of proteins and nucleic acids in the small intestine of ruminant and non-ruminant animals are the same. However, it has been reported that the rate of flow of digesta into and out of the abomasum is fairly constant, even when the ruminant is fed intermittently and the pH in the abomasum is consistently low (2 to 3) (Kay, 1969). Ruminant animals also have larger amounts of pancreatic ribonucleases compared with non-ruminant animals (Barnard, 1969). RNA digestion helps recycle nitrogen and phosphorus in the ruminant (Owens and Zinn, 1993).

Large polypeptides enter the duodenum to be hydrolyzed further by pancreatic proteolytic enzymes. Trypsin, chymotrypsin, and elastases secreted as zymogens are endopeptidases. The pancreatic zymogens are produced by the acinar cells of the pancreas and delivered to the duodenum. Trypsin specifically attacks peptide bonds of basic amino acids (histidine, lysine and arginine) whereas chymotrypsin is specific for peptide bonds containing uncharged amino acid residues, such as aromatic amino acids (phenylalanine, tyrosine and tryptophan). Elastase has broad specificity in attacking bonds next to small amino acid residues such as glycine, alanine and serine. Enteropeptidase as a protease secreted by the intestinal mucosa catalyzes cleavage of a particular peptide bonds on the carboxyl side of lysine and arginine residues. In the duodenum, enteropeptidase also triggers the activation of the pancreatic zymogens by catalyzing the cleavage of a particular peptide bond in trypsinogen, thus producing a fully activated trypsin molecule. Once activated, trypsin activates the other pancreatic zymogens as well as activating additional trypsinogen molecules. Another pancreatic protease is carboxypeptidase secreted as procarboxypeptidase (zymogen form of carboxypeptidase) by the acinar cells of the pancreas. Carboxypeptidase is an exopeptidase and attacks the carboxyl terminal peptide bond, liberating single amino acids. Aminopeptidases and dipeptidases are also secreted by the glands of Brunner and Lieberkuhn in the intestine to complete digestion of peptides to free amino acids. In addition, pancreatic nucleases (ribonucleases (RNases) and deoxyribonucleases (DNases)) break nucleic acids (RNA and DNA) down into nucleotides (eg, ATP and AMP). Nucleic acids consist of nucleotide residues which are composed of a sugar, a weak base, and at least one phosphoryl group.

Nucleoside triphosphates serve as the monomer unit precursors of the nucleic acids. A nucleoside is a nucleotide without a phosphoryl group. (Horton et. al., 1993; and Mayes, 1996).

The existence of digesta in the gastrointestinal tract is one of the factors modifying proteolytic enzyme secretions, being higher in the presence of digesta. Vagal nerve stimulation also stimulates exocrine pancreatic secretions in ruminant and non-ruminant animals. Cholecystokinin (CCK) and secretin are considered to be the major hormones regulating pancreatic enzyme responses to meals. Secretory locations of CCK are duodenum, jejunum and ileum while secretin is secreted from duodenum and jejunum. In addition, gastrin, glucagon, somatostatin, neurotensin, dopamine and histamine are among other hormones and bioactive compounds involved in regulating proteolytic enzymes in the gastrointestinal tract (Solomon, 1987; and Kato et. al., 1991).

Ben-Ghedalia et al. (1974) reported the pH of the intestinal contents sampled at the pylorus of rams fluctuated between 2.6 to 3.0, increased gradually along the intestine. These authors also reported that since the conversion of the zymogens into active enzymes occurs above pH 5, proteolytic activities increase slowly along the intestine and reach the highest activity in the samples withdrawn at a distance of 7 m from the pylorus.

Amino acids in the L form are actively absorbed from the jejunum and ileum, and competition for absorption exists between certain groups of amino acids (Owens and Zinn, 1993). The most intensive absorption of free amino acids was in the section 7 to 15 m distant from the pylorus of rams (Ben-Ghedalia et. al., 1974). Webb et al. (1992) also indicated peptides quantitatively are a more important form of amino acid absorption in ruminants than free amino acids, and the rumen and omasum may be major sites of peptide absorption. Peptides taken up by the intestinal mucosa are subsequently cleaved by dipeptidases in the cytosol of the epithelial cells of the intestinal mucosa and are transferred into the blood as free amino acids (Merchen, 1993).

Digestion of residual N compounds also takes place in the large intestine. The large intestine has a pH between 7 to 8 and experiences only microbial digestion, no digestive enzymes from the host exist in this part of the GI tract.

Virtually all proteins denatured in the abomasum are digested in the small intestine. However, Postruminal apparent digestibility of N compounds ranges from 65 to 75% of duodenal N. (Owens and Zinn, 1993).

Effects of diet and other factors on body composition

Growth has been defined as increases in tissue mass as a combination of hyperplasia in early life and hypertrophy later in life (Owens et al., 1993). Growth is composed, mainly, of fat and protein deposition in soft tissues or minerals in the case of bone. Body composition during postnatal growth is influenced by several factors mainly diet, live weight, maturity, genotype, sex and the environment.

Diet. The importance of nutrition starts immediately after birth. Colostrum has been shown to stimulate protein synthesis in the gastro-intestinal tract and skeletal muscles (Patureau-Mirand et al., 1991; Burrin et al., 1992).

It has been illustrated in sheep that nitrogen (N) retention is linearly related to N intake when N intake is below the requirement (protein-dependent phase of protein accretion). When N intake is in excess of the requirement, N retention is shown to be related to energy intake (energy-dependent protein accretion) (Black and Griffiths, 1975). A similar protein accretion response has also been demonstrated for pigs (Campbell et al., 1984; and Campbell et al., 1985a) and broiler chickens (Gous and Morris, 1985). Excess amounts of protein in the diet beyond that needed for maximal protein accretion causes heat production resulting in reduced efficiency of energy utilization (Hartsook and Hershberger, 1971; Walker and Norton, 1971; Holmes et al., 1980; and Campbell et al., 1985b). Under these conditions lipogenesis is depressed while protein deposition is increased (Campbell, 1988). Nevertheless, protein deposition may also decrease under conditions of protein adequacy if energy intake is not met for maximal protein accretion (Black and Griffiths, 1975). Cook (1996) stated that high protein diets caused a decrease in amount of fatty acid synthase mRNA in adipose resulting in decreased fat deposition.

Refeeding after periods of nutrient restriction has resulted a greater growth rate (i.e., compensatory growth) compared with the growth rate of animals of same weight given unlimited access to feed (Elliott and O'Donovan, 1969; and Allden, 1970). Age, severity and duration of nutrient restriction, however, play important roles in compensatory growth of body tissue mass (Allden, 1968).

Live weight. Body composition at birth includes the skeleton, muscles and internal organs with fat representing less than 2% of body weight (Trenkle and Marple, 1983). Fat and protein deposition occur simultaneously during early growth. However, in later growth, beyond a certain body weight, the rate of protein accretion becomes negligible, whereas fat accretion continues thus becoming a large and constant fraction of weight gain (Zucker and Zucker, 1963; Bailey and Zobrisky, 1968; Searle et al., 1972; and Graham and Searle, 1972). Growth of skeleton, organs and musculature continue in postnatal growth until the animal reaches about 50 to 60% of its mature weight at which time skeletal and organ weight gain has slowed and fat deposition has increased to a modest rate (Trenkle and Marple, 1983).

Maturity. Increase in body weight with time (i.e., age) follows a characteristic S-shaped curve indicating a slow increase in the beginning and acceleration before a final decreased rate as the animal becomes mature (Gill and Oldham, 1993). Rates of maturity for different parts of the body vary with age. Muscles, for example, are considered as faster developing tissues than fat (Elsley et al., 1964). The proportion of fat to protein in a carcass at slaughter, therefore, depends upon the animal's stage of maturity with higher protein proportion for less mature animals.

Genotype. It has been generally agreed that genotypes which are heavier at maturity grow faster and have less proportion of fat in their empty bodies and carcasses than do animals of smaller mature size (Wood et al., 1975; Gregory et al., 1977; Moody et al., 1978; Theriez et al., 1981; Leclercq and Saadoun, 1982; and Ellis et al., 1983). Different responses to insulin and IGF-1 within different genotypes have been responsible in stimulating uptake of glucose and amino acids and decreased protein degradation (Oddy, 1993).

Breed differences largely exist in terms of growth and carcass composition. Comparison of 10 breeds and crosses of cattle indicated that daily body weight gain varied between breeds from 682 (Murray Grey) to 857 gd^{-1} (Simmental) (Southgate et al., 1982). McClelland et al. (1976) also observed that the Finn sheep compared with Finnish and Southdown cross were considerably fatter, particularly at empty body weights between 20 and 30 kg. In addition, the carcasses of Colbred sheep have been reported to contain less fat in comparison with other breeds (Wood et al., 1980).

Sex. Among cattle and sheep intact males have leaner carcasses than females (Searle and Griffiths, 1976; Webster et al., 1977; and Campbell et al. 1985b). Castrated males also have less fat than females of the same weight (Thompson et al., 1979). Barrows, however, are reported to be fatter than gilts (Trenkle and Marple, 1983). The endocrine system, in particular the male and female sex hormones, plays an important role in the control of protein synthesis and breakdown (Lobley et al., 1985).

The environment. High ambient temperature has been shown to influence growth negatively (Morrison, 1983). Black (1983) also stated that moderate cold depresses growth rate and fat synthesis, but does not have any effect on protein deposition.

Conclusion

Although crude protein requirements for lambs of small, medium and large mature weight genotypes have been recommended by the National Research Council (NRC) (1985) lambs expressing the *Callipyge* gene have recently been discovered in the sheep industry, and their crude protein requirement has not yet been established. Lambs expressing the *Callipyge* gene produce leaner and higher yielding carcasses compared with normal muscled lambs. Sheep producers and consumers indicate CLPG lambs would improve the desirability of market lambs. Thus, it is essential that the protein requirements for optimum growth and feed efficiency be established for lambs expressing the *Callipyge* gene.

CHAPTER III

MATERIALS AND METHODS

Feeding and management

A feeding trial, containing thirty-two lambs (51.6 ± 0.96 kg) expressing muscle hypertrophy (*Callipyge*/GLPG) and twenty-nine normal (49.2 ± 0.99 kg) half-sibling white faced ewe lambs, was conducted to establish the dietary crude protein requirements for optimum growth and feed efficiency of CLPG lambs. Animals, approximately 5 months of age, were supplied from the U.S. Sheep Research Station (Dubois, ID) and transported to our sheep facility shortly after weaning. Each genotype was stratified by body weight and randomly assigned to one of four diets containing 9.9, 13.5, 16.7 and 19.9% CP. Diets were ground and mixed and offered *ad libitum*. Diets were formulated to meet all nutrient requirements, except protein, for fast growing lambs as recommended by NRC (1985). Diets also contained 85% concentrate including heat-treated soybean meal (SoyPlus/West Central, Ralston, Iowa 51549) and corn and 15% second cutting alfalfa hay (Table 1). Lambs were adapted to experimental diets for a four-week period. Water was available at all times.

All lambs were individually self-fed and housed in

1.22 x 2.44 m slotted floor pens in a temperature modified sheep barn at the Livestock Center at the University of Wyoming. The feeding trial started in early September, 1995 and ended at the end of February, 1996. The two-day weights of lambs were averaged for initial weights. Lambs were then weighed every two weeks until the first lamb reached the target slaughter-weight of 61.87 ± 0.30 kg and thereafter weekly to determine ADG and feed efficiency. Feeders were checked each morning and appropriate diets were added as needed. Tared feeders containing feed were also weighed to calculate DMI on the same days lambs were weighed.

Slaughter and carcass data

Lambs reaching the target slaughter weight of 61.87 ± 0.30 kg were killed by using procedures outlined in the Humane Slaughter Act and under State of Wyoming inspection equivalent to federal inspection. Based on a schedule of the University's slaughter crew the sheep were transported, fasted overnight and killed the following morning (0600 h). At slaughter the weights of the following were collected: head, pelt, full and empty GI tract, heart, liver (without gall bladder), kidneys, small intestine (ingesta and external fat removed) and hot carcass. After the carcasses were chilled at 2-3° C for two wk chilled carcass weight, carcass length (distance between the anterior side of the 1st rib and the aitch bone), loin eye area, fat depth over the loin (12th rib), body wall thickness 12.7 cm off the midline between the 12th and 13th ribs, leg scores, and metacarpal length and weight were collected. Dressing percentage, empty body weight, shrink-percentage, and protein (Kirton and Barton, 1962) and fat percentage (Riley and Field, 1969) of carcasses were calculated.

The equation to predict protein percentage of carcasses described by Kirton and Barton (1962) was:

$$Y = 21.18 - 0.194\ X$$

where;

Y = carcass protein (%)

X = carcass fat (%)

Standard error estimate = 0.44%

In this equation carcass fat (%) was calculated using the Riley and Field (1969) equation:

$$\text{Percent fat}^{*} = 14.34 + 16.0\ A + 20.3\ B$$

where;

A = body-wall thickness in inches

B = rib-eye fat thickness in inches

Standard error estimate = 2.95

* Percent fat (ether extract) in the boneless carcass, not including kidney and pelvic fat.

Laboratory Analysis

Feed samples were ground to pass through a 2 mm screen and analyzed for crude protein using a Tecator Keltec 2020 digestion block and Tecator Keltec 1026 distilling unit (Perstorp Analytical Inc. Silver Springs, Maryland 20904), DM and OM (AOAC, 1984). All samples were analyzed in duplicate.

Statistical Analysis

Performance trial data and carcass and slaughter data were analyzed using the GLM procedures of SAS (1985) to compare the effects of genotype, diet and genotype*diet on performance characteristics of the lambs. Initial body weight was used as a covariate in the statistical model because of differences in body weights of CLPG and normal lambs (51.6 ± 0.96 kg and 49.2 ± 0.99 kg, respectively). In both performance trial data and carcass and slaughter data, when significant treatment effects were found, linear, quadratic and cubic effects of diet were evaluated using contrast analysis. If more than one contrast was significant ($p < .10$), the contrast of the highest order is reported.

Table 1. Chemical and ingredient composition of the diets

Item (%)	Diet-1	Diet-2	Diet-3	Diet-4
CP (DMB)	9.9	13.5	16.7	19.9
DM	87.6	88.0	88.6	88.8
OM (DMB)	92.7	93.2	92.5	92.1
Alfalfa	15.0	15.0	15.0	15.0
Corn	82.0	72.5	63.0	53.0
Soy-Plus	0	9.5	19.0	29.0
Limestone	1.5	2.1	2.2	1.4
TMS[a]	1.5	0.9	0.8	0.6

[a] Trace Mineralized Salt (Sheep Trace mineral salt, Moorman Manufacturing Co, Quincy, Ill. 62301).

DMB: Dry matter basis.

CHAPTER IV

RESULTS

Feeding trial data.

There were statistically no differences between initial body weights of CLPG and normal lambs (P = .08) (Table 2). Initial body weight tended to be heavier for sheep receiving the higher dietary CP level (P > .06) (Figure 1). Since each lamb was killed at a constant target slaughter weight (61.87 ± 0.30 kg) final weights did not differ between genotypes regardless of protein levels in the diet (P > .20). All sheep reached the target slaughter weight of 61.87 ± 0.30 kg in a similar time period (i.e., days on feed) regardless of genotype or diet (P > .37). Weight change was not significantly different between the two genotypes across treatments (P > .35).

Dry matter intake was similar for both CLPG and normal lambs (P = .78). Lambs fed the diet containing 19.9% CP consumed the largest amount of food (1.71 ± 0,07 kg/d) compared with other lambs fed 9.9%, 13.5%, and 16.7% CP (Table 3). Linear increases in DMI were observed (P = .01) as dietary protein level increased (Figure 2). Although the numerical value of ADG was higher for CLPG lambs (0.22 vs. 0.21 kg/d) the presence of CLPG gene had no effect on

Table 2. Feeding trial data of CLPG and normal lambs.

	LSMEANS			
Observation	CLPG	Normal	SE_{pooled}	P
Initial wt., kg	51.67	49.24	1.37	0.08
Final wt., kg	62.15	61.59	0.43	0.20
Days on feed, d	55.97	56.01	3.96	0.99
Weight change, kg	11.79	11.39	0.41	0.35
DMI, kg/d	1.50	1.48	0.07	0.78
ADG, kg/d	0.22	0.21	0.01	0.77
Feed efficiency	0.12	0.14	0.02	0.47

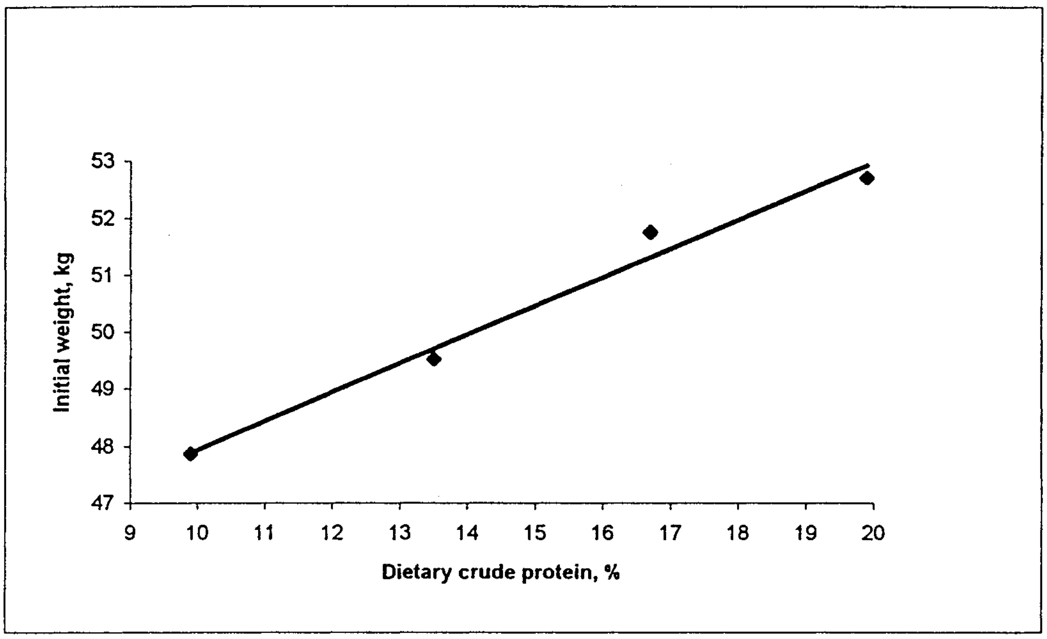

Figure 1. Initial weights varied across different protein levels in the diet (p = .06).

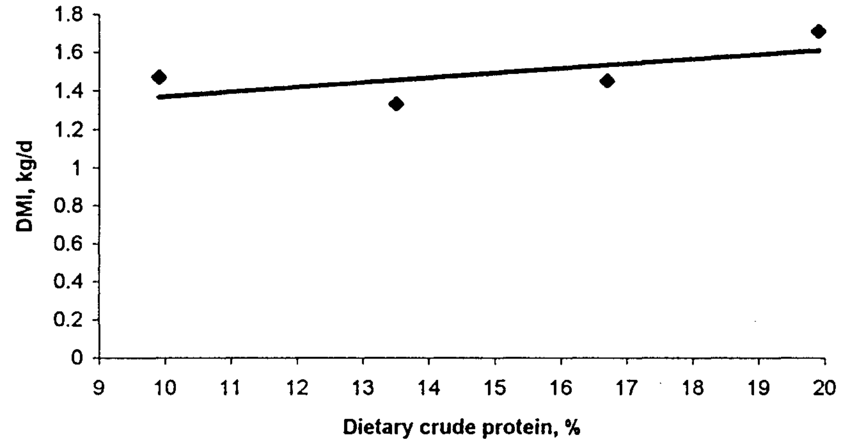

Figure 2. Increasing DMI as CP increased in the diet (linear, p = .01).

ADG (P = .77) regardless of dietary protein levels (P = .98). Feed efficiency responses of both CLPG and normal lambs were similar (P = .47) although feed efficiency was numerically greater for normal lambs compared with CLPG lambs (0.14 vs. 0.12). A diet effect was not found on feed efficiency (P = .59). No genotype*diet interactions were detected for initial wt., final wt., weight change, DMI, ADG, and feed efficiency (P > .15). Table 3 shows diet effects.

Slaughter data.

Shrunk weight did not differ within genotype (p = .10) regardless of diet (P = .41). *Callipyge* lambs had significantly heavier hot carcass weights (P = .00) compared with normal lambs (Table 4; Table 5 also represents diet effect on shrunk wt., hot carcass wt., head and pelt weights). Hot carcass weights tended to increase (P = .26) as a response to increasing protein levels in the diet (Figure 3). Weight of the head was lighter for CLPG lambs compared with normal lambs (P = .01). Decreasing head weights were observed as level of protein increased in the diet (Figure 4), but this was not significant (P = .25). *Callipyge* lambs also had lighter pelts compared with normal lambs (P = .01) regardless of diet (P = .77).

Visceral organ weights of CLPG and normal lambs are presented in Table 6. Diet effects on visceral organ

Table 3. Dietary CP level effect on performance data[a].

	LSMEANS					
	Dietary CP level					
Observation	9.9%	13.5%	16.7%	19.9%	SE_{pooled}	P
Int. wt., kg[b]	47.87	49.51	51.75	52.70	2.76	0.06
Final wt., kg	61.70	62.24	61.99	61.55	0.85	0.68
Days on feed	59.84	55.06	58.92	50.52	7.95	0.37
Wt. change, kg	11.63	12.17	11.55	11.02	0.85	0.36
DMI, kg/d[c]	1.47	1.33	1.45	1.71	0.14	0.01
ADG, kg/d	0.21	0.22	0.21	0.22	0.02	0.98
Gain/Feed	0.17	0.13	0.12	0.11	0.05	0.59

[a] Genotype*Diet means can be found in Appendix Table 1.
[b] Linear effect of increasing protein level (P = .04).
[c] Linear effect of increasing protein level (P = .02).

Table 4. Some of the slaughter characteristics of CLPG and normal lambs.

	LSMEANS			
Observation	CLPG	Normal	SE_{pooled}	P
Shrunk wt., kg	58.16	57.49	0.40	0.10
Hot carcass wt., kg	33.33	30.16	0.44	0.00
Head wt., g/kg EBW	0.04	0.05	0.00	0.01
Pelt wt., g/kg EBW	0.13	0.14	0.00	0.01

Table 5. Diet effect on some of the slaughter characteristics[a].

	LSMEANS					
	Dietary CP level					
Observation	9.9%	13.5%	16.7%	19.9%	SE_{pooled}	P
Shrunk wt., kg	58.26	58.13	57.39	57.52	0.82	0.41
Hot carc. wt., kg[b]	31.24	31.27	32.18	32.29	0.91	0.26
Head wt,g/kg EBW[c]	0.051	0.050	0.049	0.047	0.00	0.37
Pelt wt,g/kg EBW	0.13	0.14	0.13	0.13	0.00	0.36

[a] Genotype*Diet means can be found in Appendix Table 2.
[b] Linear effect of increasing protein level (P = .06).
[c] Linear effect of increasing protein level (P = .05).

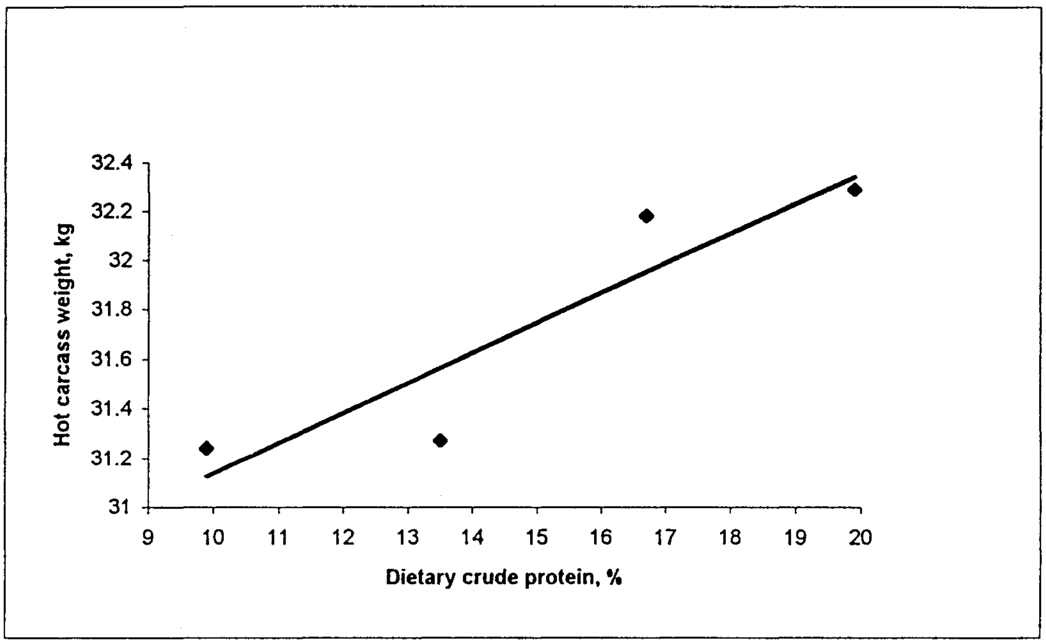

Figure 3. Hot carcass weights intented to increase as a response to increasing dietary CP level in the diet (p = .26).

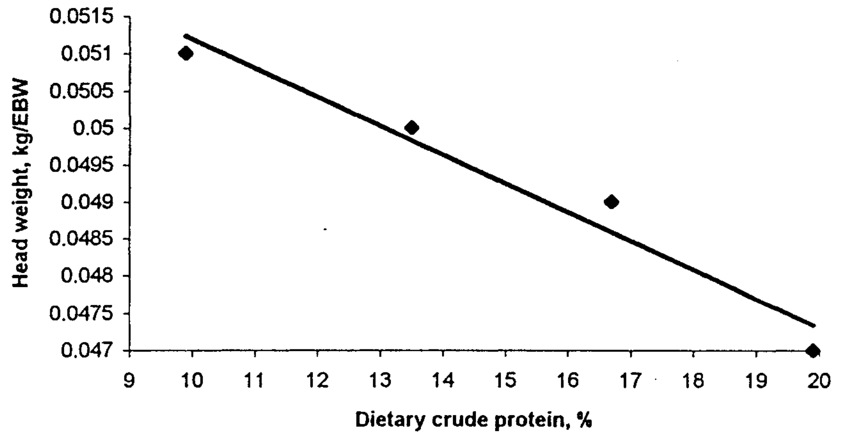

Figure 4. Decreasing weight of head as dietary crude protein level increased in the diet (p = .25).

Table 6. Visceral organs of CLPG and normal lambs.

	LSMEANS			
Observation	CLPG	Normal	SE_{pooled}	P
Heart wt., g/kg EBW	4.32	4.61	0.09	0.01
Kidneys wt., g/kg EBW	2.37	2.67	0.22	0.00
Liver wt., g/kg EBW	13.62	15.93	0.50	0.00
Small int. wt., g/kg EBW	10.86	12.44	0.45	0.00
Full GIT wt., g/kg EBW	168.38	190.63	0.42	0.00
Empty GIT wt., g/kg EBW	77.93	90.85	0.14	0.00

weight are also presented in Table 7. Lambs expressing the CLPG gene had lighter heart (P = .01) and kidneys (p = .00) without respect to dietary protein level (P > .22). *Callipyge* lambs had substantially lighter liver mass compared with normal lambs (P = .00). Sheep fed the diet containing 19.9% CP had the heaviest liver (15.71 ± 0.54 g/kg EBW) compared with other sheep consuming 9.9%, 13.5%, and 16.7% CP. Contrast analysis indicated that increasing protein level resulted in a linear increase in liver weight (P = .05) (Figure 5). Small intestine weight was significantly lighter for CLPG lambs (P = .00) compare with normal lambs. In the present study, small intestine weight was affected by genotype*diet (P = .01). This interaction showed that the small intestine weight of CLPG did not change as a proportion of empty body weight (g/kg EBW) as dietary CP level increased (Figure 6). Weights of full and empty gastro intestinal tracts (GIT) were lighter for CLPG lambs (P = .00). Full GIT mass decreases linearly (P = .00) as dietary protein level increased (Figure 7).

Normal and CLPG lambs shrunk similarly (i.e., shrink percentage) (P = .92) (Table 8) regardless of diet (P = .61). *Callipyge* lambs had significantly heavier empty body weights (P = .01) compared with normal lambs, resulting from lighter visceral organs of CLPG lambs. Dressing percentage of CLPG was substantially higher (P = .00) compared with

Table 7. Diet effects on visceral organs[a].

	LSMEANS					
	Dietary CP level					
Observation[b]	9.9%	13.5%	16.7%	19.9%	SE_{pooled}	P
Heart	4.49	4.55	4.45	4.36	0.19	0.65
Kidneys	2.40	2.57	2.50	2.61	0.14	0.22
Liver[c]	13.80	15.22	14.37	15.71	1.04	0.05
Small int.	11.71	12.55	11.59	10.75	0.92	0.36
Full GIT[d]	195.28	202.43	158.44	161.87	0.96	0.00
Empty GIT	88.93	83.44	82.63	82.57	0.51	0.25

[a] Genotype*Diet means can be found in Appendix Table 3.

[b] Weight of visceral organs are expressed in terms of g/kg empty body weight

[c] Linear effect of increasing protein level (P = .04).

[d] Linear effect of increasing protein level (P = .00).

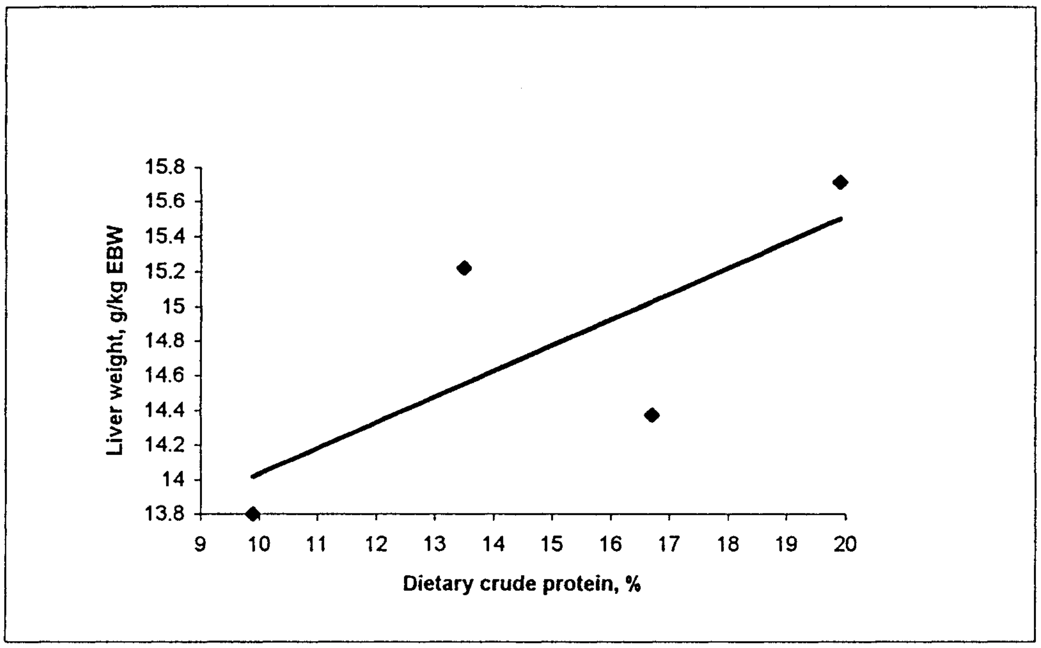

Figure 5. Changes in the liver weight as CP increases in the diet (p = .05).

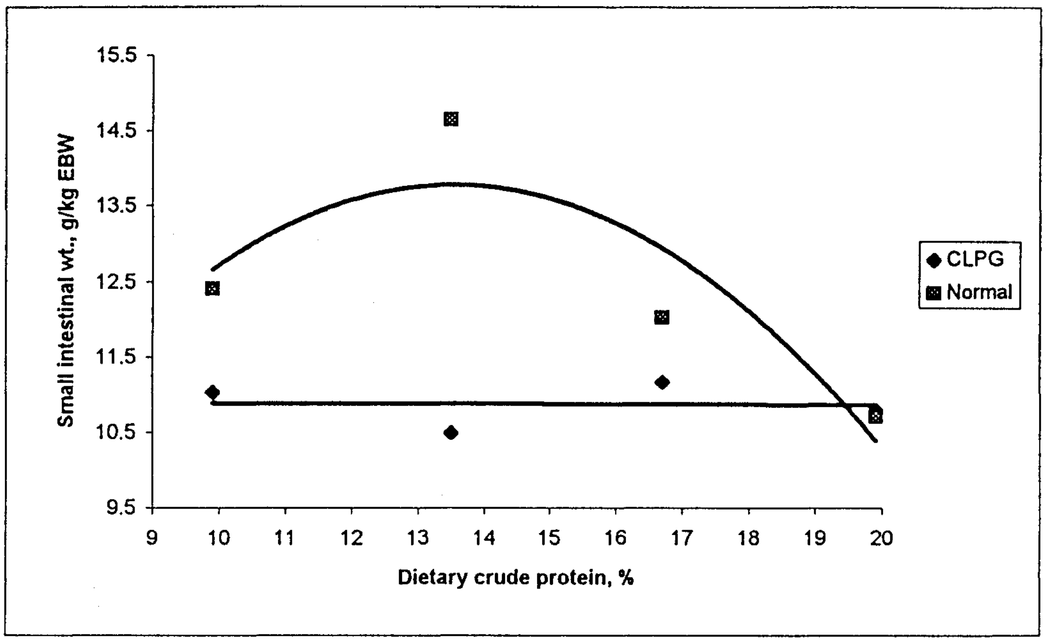

Figure 6. The effect of Genotype*Diet on small intestinal weight, g/kg EBW (p = .01).

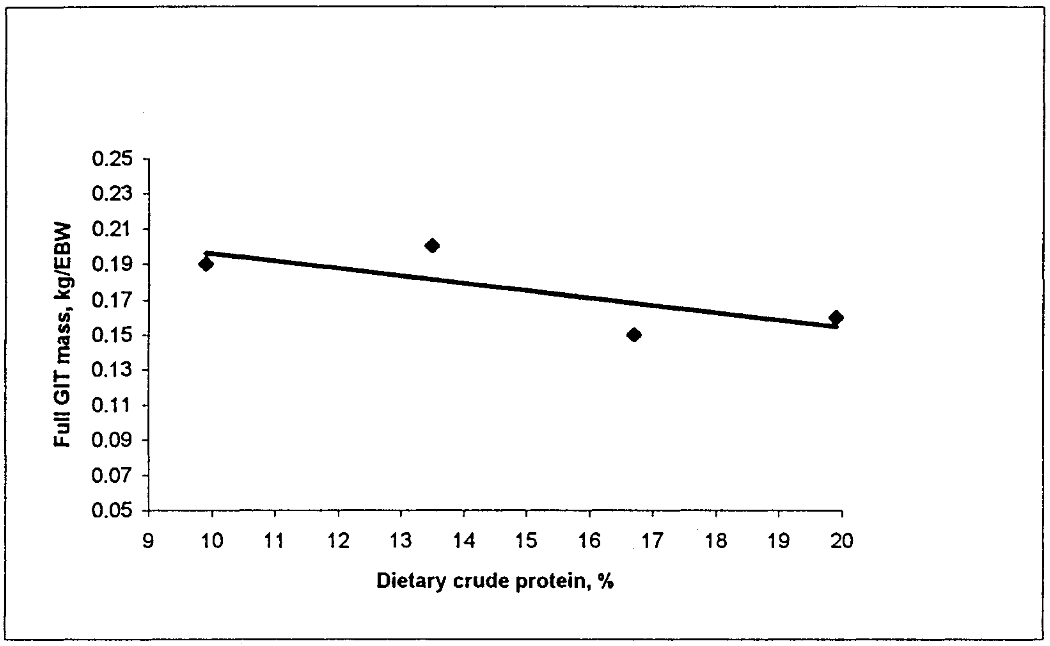

Figure 7. Decreasing full GIT mass as CP level increased in the diet (linear, p = .00).

Table 8. Calculated slaughter characteristics of CLPG and normal lambs.

Observation	CLPG	Normal	SE_{pooled}	P
	LSMEANS			
Shrink per., %	6.5	6.4	0.56	0.92
Empty body wt, kg	53.08	52.09	0.39	0.01
Dressing per., %	57.30	52.47	0.70	0.00

normal lambs. Increasing dietary CP caused linear increases in dressing percentage (P = .03) (Figure 8). Except for small intestine mass there were no interactions observed in any data collected at slaughter (P > .06). Calculated shrink %, empty body weight and dressing percentage can be found in Table 9.

Carcass data.

Carcass characteristics of CLPG and normal lambs are represented in Table 10. Table 11 also shows carcass characteristics influenced by dietary CP level. There were no genotype*diet interactions detected (P > .11) for any carcass data. Chilled carcass weight of CLPG lambs was substantially heavier (P = .00) compared with normal lambs. Lambs expressing the CLPG gene had significantly shorter carcasses (P = .05) compared with normal lambs without respect to dietary protein level (P = .26). There were no differences obtained for either metacarpal length (P = .23) of metacarpal weight (g/kg EBW) (P = .57) between the two genotypes. Body-wall thickness was less for CLPG lambs compared with normal lambs, but the difference was not significant (P = .37). The thinnest body-wall was measured in sheep consuming 9.9% CP (2.69 ± 0.11 cm). Body-wall thickness tended to increase linearly with increasing protein level in the diets (P = .06) (Figure 9). Without respect to diets *Callipyge* lambs had significantly less fat

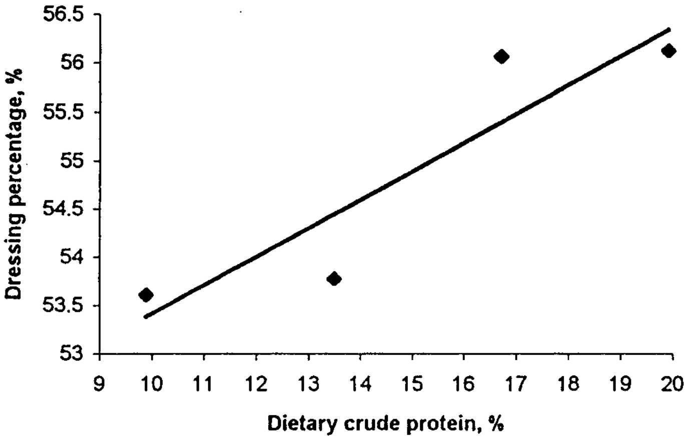

Figure 8. Dressing percentage increased as protein level increased in the diet (linear, p = .03).

Table 9. Dietary CP level effect on calculated slaughter items[a].

	LSMEANS					
	Dietary CP level					
Observation	9.9%	13.5%	16.7%	19.9%	SE_{pooled}	P
Shrink per., %	6.19	6.56	7.11	6.09	0.26	0.61
Empty BW, kg	52.44	51.90	52.81	53.18	0.79	0.54
Dress. per., %[b]	53.60	53.77	56.06	56.12	1.43	0.03

[a] Genotype*Diet means can be found in Appendix Table 4.

[b] Linear effect of increasing protein level (P = .00).

Table 10. Carcass characteristics of CLPG and normal lambs.

	LSMEANS			
Observation	CLPG	Normal	SE_{pooled}	P
Chilled carc. wt., kg	31.77	28.65	0.44	0.00
Carcass length, cm	69.48	70.60	0.56	0.05
Metac. length, cm	11.51	11.36	0.11	0.23
Metac. wt, g/kg EBW	0.79	0.80	0.14	0.57
Body-wall, cm	2.90	3.00	0.10	0.37
Fat depth, cm	0.44	0.76	0.56	0.00
Leg scores[a]	14.00	11.51	0.24	0.00
Loin eye, cm^2	25.93	17.52	0.85	0.00
Carcass prot., %	14.15	13.52	0.21	0.00
Carcass fat, %	36.23	39.44	1.13	0.00

[a] Leg scores given numerical values for data analysis starts 10 which is the lowest leg score and 15 represents the highest leg score.

Table 11. Carcass characteristics influenced by dietary CP level in the diet[a]

	LSMEANS					
	Dietary CP level					
Observation	9.9%	13.5%	16.7%	19.9%	SE_{pooled}	P
Chilled carc. wt, kg	29.92	29.59	30.90	30.43	0.97	0.00
Carc. length, cm	70.99	69.96	69.89	69.30	1.15	0.26
Metac. length, cm	11.46	11.39	11.45	11.45	0.24	0.98
Metac. wt.[b]	0.80	0.81	0.78	0.78	0.24	0.54
Body-wall, cm[c]	2.69	2.93	3.05	3.15	0.22	0.06
Fat depth, cm	0.58	0.67	0.53	0.64	0.13	0.44
Leg scores[d]	12.78	12.37	12.82	13.05	0.50	0.32
Loin eye, cm^2 [e]	20.52	19.99	22.92	23.47	1.75	0.02
Carcass prot., %	14.20	13.77	13.84	13.54	0.44	0.26
Carcass fat, %	35.96	38.18	37.82	39.37	2.33	0.26

[a] Genotype*Diet means are represented in Appendix Table 5.

[b] Metacarpal weight is expressed in terms of g/kg EBW.

[c] Linear effect of increasing protein level (P = .00).

[d] Leg scores given numerical values for data analysis starts 10 which is the lowest leg score and 15 represents the highest leg score.

[e] Linear effect of increasing protein level (P = .02).

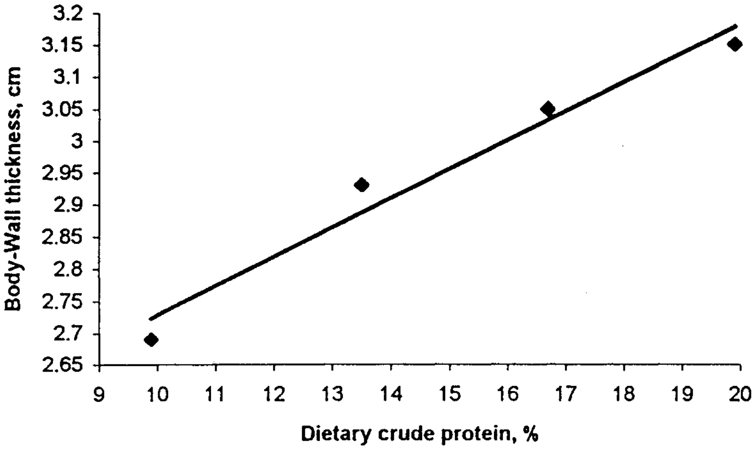

Figure 9. Body-Wall intended to increase as a response to increasing dietary protein levels in the diet (linear, p = .06).

over the loin eye (i.e., fat depth) (P = .00) compared with normal lambs. Leg scores were found to be significantly higher for CLPG lambs compared with normal lambs (P = . 00). The presence of the CLPG gene resulted in substantially larger loin eye area (P = .00). The largest average loin eye area was measured in CLPG consuming the highest dietary CP, 19.9% (28.28 ± 1.25 cm^2). Loin eye area linearly increased (P = .02) as dietary protein levels increased (Figure 10). Although there were no diet effects on carcass protein (P = .26) or fat percentage (P = .26), calculated carcass protein and fat percentages indicated that lambs expressing the CLPG gene had significantly lower fat (P = .00) and higher protein percentages (P = .00) in their carcasses compared with normal lambs.

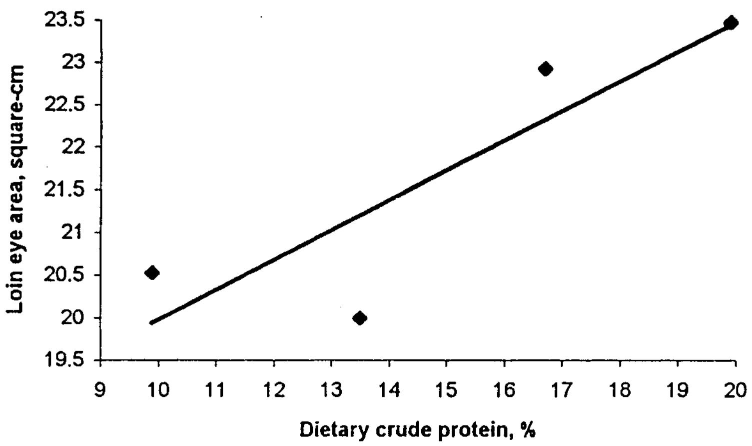

Figure 10. The effect of dietary crude protein on loin eye area of CLPG and normal lambs (linear, p = .02).

CHAPTER V

DISCUSSION

Feeding trial.

Initial BW and final BW of both CLPG and normal lambs were not different. Without respect to CP levels in the diet days on feed were similar between the two genotypes indicating that growth rate of CLPG and normal lambs followed a similar pattern. Brown et al.(1995), however, found the growth response was more rapid for CLPG lambs than normal lambs. The presence of the CLPG gene had no effect on either DMI, ADG or feed efficiency regardless of level of protein in the diet. Similar DMI (Bernal et. al., 1995; and Brown et. al., 1995) and ADG (Snowder et. al., 1994a; and Brown et. al., 1995) between CLPG and normal lambs were also observed by other researchers. In this research, it is likely the expected superior feed efficiency (Jackson, 1993; and Brown et. al., 1995) and ADG of CLPG lambs was not found due mainly to heavy initial body weights of both CLPG and normal lambs used in the trial (CLPG = 51.67 ± 0.96 kg, normal = 49.24 ± 0.99). Available data from different animal species indicated that beyond a certain body weight the rate of protein accretion becomes negligible, whereas fat gain continues thus becoming a large fraction of body weight gain

(Zukcer and Zucker, 1963; Bailey and Zobrisky, 1968; Searle et. al., 1972; and Graham and Searle, 1972). In addition, in the present study diets containing 16.7% and 19.9% CP included 60% by-pass protein (Soy-Plus) and maybe supplied excess CP for normal and perhaps CLPG lambs. Because the initial body weights of lambs from both genotypes were rather heavy, it is unlikely differences in performance due to diet or genotype could be expected.

Voluntary feed intake is usually depressed when animals are given protein deficient diets (Black, 1974). In our trial a linear increase on DMI was observed as protein levels increased in the diets. It has been reported that increasing dietary CP resulted in increases in DMI (Orskov et al., 1971; and Willms et. al., 1991). Veira et al. (1980) also reported a quadratic relationship between CP level in the diet and DMI. It has been shown in rats that under nutritional circumstances where plasma free amino acids levels are highly elevated DMI is depressed (Peng and Harper, 1970). This excessive accumulation of free amino acids in plasma and tissue pools results in the reduction of hepatic amino acid degrading enzymes (Harper, 1968). Under these conditions, except for branched-chain amino acids, plasma free amino acids return to normal levels and feed intake returns to normal, this phenomenon is called "aminostatic control" of feed intake (Peng and Harper, 1970).

Increasing CP levels from 11.0% to 19.4% with the diets made from barley and fish meal resulted in increasing in average daily gain (Orskov et al., 1971). In the same study, feed efficiency was best on the highest protein concentration. Zinn and Owens (1993), however, showed that the addition of 2% supplemental ruminal escape protein provided the greatest rate and efficiency of gain compared with addition of 4% or 6% ruminal escape protein to the basal diet of cattle containing 12.2% CP.

Slaughter data.

Lambs expressing the CLPG gene had significantly lighter organ weights when expressed in terms of empty body weight (g/kg EBW) compared with normal lambs. These results are not consistent with the results of a similar study conducted by Jackson et al. (1994) who found no differences in visceral organ weights with the exception of the large intestine between CLPG and normal lambs. Several researchers pointed out restriction of feed intake resulted in a decrease in the relative proportion of visceral tissues to EBW (Marston, 1948; Meyer and Clawson, 1964; Ledger and Sayers, 1977; Ferrel et. al., 1986; and Burrin et. al., 1990). Increased visceral organ sizes were detected when the status of diet changed from restriction to *ad libitum* as a response to increasing metabolic activity of visceral organs

(Burrin et. al., 1990). In the present study increasing liver weights were observed as dietary protein increased. The ingestion of high protein diets in growing animals increases the mass of visceral organs including liver, kidneys and gastrointestinal tract as a result of high amounts of nitrogenous compounds processed by these organs (Anugwa et. al., 1989; Pond et. al., 1989; and Pond et. al., 1990). Pond et al. (1989) showed that a high protein diet (34.1% CP) offered to growing pigs resulted in increasing liver and kidney weights compared with pigs fed the control diet (14.6% CP).

In our experiment the only interaction between diet and organ size was for small intestine mass. For normal lambs, small intestine weight (g/kg EBW) was increased gradually as protein level increased from 9.9% to 13.5%, followed by a dramatic decrease as protein level increased further (17.6% and 19.9%). Small intestinal mass of CLPG lambs, on the other hand, did not change as protein level was increased in the diet. Based on the findings of Anugwa et al. (1989) and Pond et al. (1989) indicating increases in organ mass as a response to elevated protein level in the diet, no response in small intestinal mass to increasing dietary crude protein by *Callipyge* lambs is not well explained. Species differences also exist for small intestinal responses to diet in terms of digestive physiology. It has been shown by

Pond et al. (1989) that small intestinal weight is increased in pigs but decreased in rats when fed high protein diets.

Empty body weights of CLPG lambs were heavier compared with normal lambs resulting simply from lighter visceral organs of CLPG lambs. Higher dressing percentage of CLPG lambs found in the present study is consistent with other researchers' findings (Jackson, 1993; Jackson et. al., 1994; Snowder et. al., 1994b; and Brown et. al., 1995).

Carcass characteristics.

The presence of the CLPG gene resulted in shorter carcass length compared with normal lambs, this finding is similar to the results of Meyer et al. (1995) and Brown et al. (1995). Although CLPG lambs had shorter carcasses neither metacarpal length (cm) nor metacarpal weight (g/kg EBW) differed between the two genotypes regardless of dietary crude protein level. Brown et al. (1995), however, found lighter right metacarpal weight of CLPG lambs expressed as g compared with normal lambs.

Thinner body-wall thickness, less fat over the loin, and lower carcass fat percentage of CLPG lambs provided evidence that CLPG lambs deposited less fat compared with normal lambs. In addition, CLPG lambs deposited more protein as they exhibited higher leg scores, larger loin eye area, and higher percentage of carcass protein compared with normal lambs.

Significant linear increases in loin eye area were observed as protein level was increased in the diet. Available data from rats, lambs and pigs (Hartsook and Hershberger, 1971; Walker and Norton, 1971; Holmes et. al., 1980; Campbell et al., 1985b; and Chen et. al., 1995) indicated that, when protein is supplied in excess of the amount needed for maximum protein accretion, protein deposition is increased while lipogenesis is reduced. Under these conditions excess protein causes heat production resulting in reduced efficiency of energy utilization thus depressing lipogenesis and increasing protein deposition (Campbell, 1988). However, protein deposition may also decrease under conditions of protein adequacy if energy intake is not met for maximal protein accretion (Black and Griffiths, 1975). The amount of fatty acid synthase mRNA in adipose tissues is decreased when a high protein diet is fed to animals, resulting in a higher carcass protein to fat ratio (Cook, 1996).

Relative distribution of protein accretion in muscle and visceral organs appears to favor muscle tissues in CLPG lambs. Muscles representing the largest protein store in the body dominate the protein gain during growth (Lobley, 1993). The fractional synthesis rate of gastro-intestinal tract, however, is three times higher than that of skeletal muscle of the same animal (Attaix and Arnal, 1987; and Attaix et.

al., 1988). The large muscle mass of CLPG lambs may also result from a low protein degradation rate of muscles. Oddy (1993) stated that genetically faster growing lambs have lower rates of protein breakdown in muscles and in whole body. This author also indicated that genetic differences in terms of growth are closely related to differences in responsiveness of muscles to hormones particularly insulin-like growth factor-1 (IGF-1).

CHAPTER VI

IMPLICATIONS

Chen (1995) stated that pigs producing more muscle and less fat have different dietary protein (amino acid) requirements from pigs producing less muscle and more fat to achieve their genetic potential for lean growth. The recently discovered *Callipyge* gene in domestic sheep has resulted in more muscle mass (muscle hypertrophy) and less fat content of carcasses compared with normal lamb carcasses. Dietary crude protein requirements of these heavily muscled lambs, therefore, are likely to be different from the National Research Council recommendations for normal lambs. *Callipyge* lambs obviously have certain advantages such as a lower carcass fat:protein, higher dressing percentage, and higher retail yields. In the present study, superior feed efficiency and ADG for lambs expressing the CLPG gene were not found, perhaps as a result of heavy initial body weights of lambs used in the trial. On the other hand, 19.9% CP diet provided the largest loin eye area in CLPG lambs, suggesting that protein requirements of CLPG lambs may be higher than NRC recommendations, but economic considerations should be taken into account as protein is a costly ingredient in ruminant diets. Further research should be done to establish the precise dietary crude protein requirements of CLPG lambs.

Botkin et al. (1988) identified consumption rate, availability, expense, and excessive fat of lamb meat are some of the major problems that exist in the lamb industry. Some of these problems may be solved by using lambs expressing the CLPG gene since their carcasses are higher yielding and contain leaner meat.

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Appendix Table 1. Genotype*Diet means on performance data

	LSMEANS			
	CLPG			
Observation	9.9% CP	13.5% CP	16.7% CP	19.9% CP
Initial wt., kg	49.03	50.84	50.86	53.96
Final wt., kg	61.87	62.45	62.24	62.08
Days on feed, d	59.59	59.00	53.96	51.35
Weight change, kg	11.69	12.37	11.69	11.42
DMI, kg/d	1.55	1.19	1.50	1.76
ADG, kg/d	0.21	0.20	0.24	0.22
Gain/Feed	0.12	0.14	0.12	0.11

Appendix Table 1 (Cont). Genotype*Diet means on performance data

		LSMEANS			
		Normal			
9.9% CP	13.5% CP	16.7% CP	19.9% CP	SE_{pooled}	P
46.71	48.18	50.65	51.43	5.51	0.99
61.54	62.06	61.74	61.03	1.72	0.93
59.38	51.12	63.87	49.69	15.82	0.45
11.58	11.96	11.40	11.66	1.70	0.94
1.39	1.48	1.41	1.65	0.29	0.15
0.20	0.23	0.19	0.22	0.056	0.53
0.22	0.13	0.11	0.11	0.11	0.50

Appendix Table 2. Genotype*Diet means for some of slaughter characteristics

	LSMEANS			
	CLPG			
Observation	9.9% CP	13.5% CP	16.7% CP	19.9% CP
Shrink wt., kg	58.69	58.02	57.70	58.23
Hot carcass wt., kg	32.65	33.53	33.32	33.82
Head wt., g/kg EBW	0.049	0.048	0.047	0.045
Pelt wt., g/kg EBW	0.13	0.13	0.13	0.12

Appendix Table 2 (Cont). Genotype*Diet means for some of slaughter characteristics

LSMEANS					
Normal					
9.9% CP	13.5% CP	16.7% CP	19.9% CP	SE_{pooled}	P
57.83	58.24	57.08	56.82	1.63	0.57
29.83	29.00	31.03	30.76	1.80	0.34
0.052	0.051	0.050	0.049	0.00	0.99
0.14	0.14	0.14	0.15	0.00	0.61

Appendix Table 3. Genotype*Diet means for visceral organs

	LSMEANS			
	CLPG			
Observation	9.9% CP	13.5% CP	16.7% CP	19.9% CP
Heart wt., g/kg EBW	4.30	4.40	4.41	4.18
Kidneys wt., g/kg EBW	2.32	2.27	2.42	2.48
Liver wt., g/kg EBW	13.07	13.37	13.90	14.16
Small int.wt., g/kg EBW	11.03	10.46	11.16	10.79
Full GIT wt., g/kg EBW	178.02	184.15	155.81	155.54
Empty GIT wt., g/kg EBW	79.92	80.75	75.48	75.57

Appendix Table 3 (Cont). Genotype*Diet means for visceral organs

LSMEANS					
Normal					
9.9% CP	13.5% CP	16.7% CP	19.9% CP	SE_{pooled}	P
4.69	4.71	4.50	4.53	0.41	0.76
2.48	2.87	2.58	2.74	0.29	0.15
14.54	17.08	14.83	17.26	2.06	0.20
12.40	14.64	12.02	10.70	1.85	0.01
212.53	220.70	161.07	168.20	1.92	0.06
97.95	86.12	89.77	89.56	1.02	0.35

Appendix Table 4. Genotype*Diet means for calculated slaughter characteristics

	LSMEANS			
	CLPG			
Observation	9.9% CP	13.5% CP	16.7% CP	19.9% CP
Shrink per., %	5.71	7.56	7.20	5.58
Empty body wt., kg	53.24	52.67	52.63	53.78
Dressing per., %	55.62	57.74	57.75	58.08

Appendix Table 4 (Cont). Genotype*Diet means for calculated slaughter characteristics

<u>LSMEANS</u>

<u>Normal</u>

9.9% CP	13.5% CP	16.7% CP	19.9% CP	SE_{pooled}	P
6.66	5.56	7.03	6.59	0.22	0.25
51.65	51.13	52.99	52.58	1.59	0.30
51.58	49.79	54.37	54.15	3.02	0.10

Appendix Table 5. Genotype*Diet means on carcass characteristics.

	LSMEANS			
	CLPG			
Observation	9.9% CP	13.5% CP	16.7% CP	19.9% CP
Chilled carc. wt., kg	31.31	31.41	32.08	32.29
Carc. length, cm	70.36	69.93	69.38	68.23
Metac. length, cm	11.54	11.47	11.52	11.37
Metac. wt., g/kg EBW	0.81	0.78	0.79	0.77
Body-wall, cm	2.48	3.10	3.00	3.03
Fat depth, cm	0.36	0.59	0.37	0.46
Leg scores	14.54	13.32	14.07	14.07
Loin eye, cm^2	25.25	24.26	25.95	28.28
Carc. prot., %	14.79	13.67	14.25	13.97
Carc. fat, %	32.89	38.68	36.22	37.14

Appendix Table 5 (Cont). Genotype*Diet means on carcass characteristics

LSMEANS Normal 9.9% CP	13.5% CP	16.7% CP	19.9% CP	SE_{pooled}	P
28.52	27.77	29.73	28.58	1.94	0.70
71.63	70.00	70.39	70.37	2.28	0.64
11.37	11.32	11.38	11.39	0.47	0.98
0.79	0.83	0.78	0.79	0.05	0.31
2.90	2.75	3.10	3.27	0.44	0.12
0.80	0.74	0.69	0.83	0.25	0.46
11.02	11.43	11.56	12.02	0.99	0.11
15.78	15.73	19.89	18.67	3.48	0.43
13.60	13.86	13.53	13.10	0.88	0.17
39.04	37.69	39.43	41.60	4.42	0.17