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A STUDY OF THE EFFECTS OF UNDERNUTRITION  
AND RE-ALIMENTATION ON THE ROMNEY ISHE  
(In Three Parts)

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A Thesis presented at Massey Agricultural College  
in partial fulfilment of the requirements for the  
degree of Master of Agricultural Science in the  
University of New Zealand.

by

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PART I

THE EFFECT OF UNDERNUTRITION AND RE-ALIMENTATION ON THE  
BODY COMPOSITION OF THE MATURE ROMNEY EWE

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## CHAPTER I

### INTRODUCTION

The production of meat with an optimum amount of fat is becoming a problem of increasing importance, since the consuming public is showing a distinct aversion to overfat meat. The problem of overfat meat, particularly ewe mutton, has recently been stressed, (Smith-Filling and Barton, 1954; Merritt, 1954). These authors emphasize the importance of overfatness on market realizations, overfat ewes being penalized in price as a consequence of the excessive fat having little sale value. Such animals are also inefficient producers.

As a consequence of variation in seasonal pasture production on New Zealand sheep farms, ewes are frequently provided with food greatly in excess of their energy requirements. Under these conditions they tend to accumulate excess amounts of depot fat. This may result in an increased susceptibility to metabolic disturbances such as pregnancy toxæmia, (Parry, 1956; McDonald, 1959), and be of importance in the aetiology of sleepy sickness and eversion of the vagina. Fat ewes also tend to become cast and inactive, and may be deleterious to subsequent reproductive performance, (Marshall and Hammond, 1945). Wallace, (1960) comments that high condition per se may not reduce reproductive performance, and accordingly it may be poor practice to overdo starving the condition off ewes after weaning. This observation has support from the work of Leatham, (1959).

In recent years considerable interest has been directed to the importance of a high fat consumption as one of the possible predisposing factors to arteriosclerosis in man, (Yadkin, 1958; Shorland, 1958; Shorland, 1956; Schweigert, 1955; Schweigert, 1957; Eagle, 1956; Ronnie, 1956-57; and Fullerton, (1956-57). An increased level of cholesterol in the blood is recognized as one of the factors having a positive relationship to vascular degeneration. The quality of saturated fat and the ratio of saturated fat to the amount of essential fatty acids seems to play a role in cholesterol metabolism.

Other nutrients may also be involved, such as the type of carbohydrate, and certainly the total caloric intake is important. Although knowledge of the aetiology of this disease is incomplete, the psychological impact of this widely published hypothesis on consumers is known to be of considerable importance.

Studies of the kind reported here provide information on the effect of restricting the feed of sheep during facial eczema precautions, drought feeding of sheep, and at other times when limited feeding is necessary. It also provides information on a farming practice that appears to be at present the most reliable means of control over carcass acceptability.

The objectives of the experiment were as follows:

- (1) To ascertain the effects of varying periods of undernutrition on the body composition and non-carcass components in the mature ewe.
- (2) To determine the effects of re-alimentation after restricted feeding on the carcass components and non-carcass components of liveweight.
- (3) To obtain by means of blood composition studies, and the weights of certain entire organs, some insight into possible metabolic effects of this restricted feeding.
- (4) To study the effects of undernutrition and recovery on the diameter of the muscle fibres.

## CHAPTER II

### REVIEW OF LITERATURE

#### (A) SOME PHYSIOLOGICAL ASPECTS OF UNDERNUTRITION AND OBESITY

One of the most important contributions made by Cannon (1929) to the field of physiology was his development of the concept of physiological homeostasis. Lerner, (1953) extended Cannon's definition and stated that homeostasis refers to the property of an organism to adjust itself to variable conditions or to the self-regulatory mechanisms of the organism which permit it to stabilize itself in fluctuating inner and outer environments. Since little is known of the hormonal, metabolic, autonomic, environmental, and psychological regulatory mechanisms in ruminants, it is more profitable to study the relationships found in all species of animals. Marked differences however exist between ruminants and non-ruminants, (Folley and French, 1948; Pennington, 1952). For example, the ease of oxidation of acetate, acetoacetate and related molecules shown by ruminants is probably related to their greater capacity to withstand the accumulation of tissue metabolites which can result in prolonged fasting being dangerous in non-ruminants. Considerable physiological variation also exists between individuals, (Prosser, 1955).

Obesity has been classified by Houssay (1955), Mayer (1953), and Long (1957), as being due to one or a combination of the following causes:

- (1) Alimentary obesity, due to excessive food intake.
- (2) Constitutional or endogenous obesity due to heredity.
- (3) Endocrine obesity due to disturbances in the functions of the hypophysis, adrenals, thyroid, gonads, or pancreas.
- (4) Increased lipogenesis, decreased rates of mobilization of fat, or other disturbances in tissue metabolism.
- (5) Lesions of the hypothalamus, and psychosomatic factors either mediated through the autonomic nervous system or acting at the cortical level.

(1) Mobilization and deposition of adipose tissue

Adipose tissue develops from primitive fat cells, the cells of this tissue having a specific structure, distinct from the fibroblasts of the connective tissue, (Wertheimer and Shapiro, 1948). Adipose tissue is supplied by a dense capillary net, the capillaries forming loose meshes in contact with the fat cells. The functional activity of the adipose tissue is regulated by an abundant nerve supply to both the vessels and the parenchyma. Until recently it was commonly believed that the adipose tissue was an inert depot, which surrendered its fat reserves only at the time of starvation and in a passive manner. The fat reserves were thought to serve as an insulating layer or mechanical padding of the body. Since the classic work of Schoenheimer and Rittenberg (1937) it became accepted that the adipose tissue takes part in the continuous dynamic processes of the body. Such a dynamic state implies the necessity for rapid synthesis and breakdown of fat, (Shorland, 1955). Today knowledge of the marked and extremely rapid response of fatty acid synthesis, glucose utilization and fat mobilization, amply demonstrates the central function of the adipose tissue in the calorific homeostasis of the organism, (Wertheimer and Shafrir, 1960). Shorland (1955), and Hilditch (1956), have reviewed much of the current information on the formation and composition of depot fats, including the endogenous synthesis of fatty acids from acetate, the dynamic state of tissue glycerides, and conditions which influence their fatty acid composition. An extensive discussion of the effect of age, sex, fasting, hormones and diet on tissue lipids, especially liver lipids is given by Deuel, (1955). Mobilization and deposition go on continuously without regard to the nutritional state of the animal. The lowering of the fat content of the tissue during undernutrition is the result of mobilization exceeding deposition, (Wertheimer and Shapiro, 1948). The adipose tissue furnishes a readily transportable and metabolizable substrate in the form of unesterified fatty acids, (UFA), and their release implies the presence of regulated intrinsic lipolysis. This substrate is rapidly combustible, being bound to various plasma proteins, (Olson and Vester, 1960). The release of UFA could represent the main means by which fat is liberated from the depots, and the plasma UFA concentration seems to closely reflect fat utilization.

The UFA undergoes marked changes in concentration in response to alteration in nutritional or endocrine status, (Olson and Vester, 1960). Annison (1960), with sheep has shown that the concentration of UFA rises during fasting of pregnant and non-pregnant ewes, which indicates continued mobilization of depot fats. White et al. (1956) working with sheep similarly showed that there was a marked absence of fasting ketosis. Glucose or insulin reduce the UFA concentration while epinephrine or fasting increase it. In fasting the major source of UFA is from adipose tissue. The determination of the level of this labile compound in vitro gives a more sensitive index of fat mobilization than measurements of depot fat and/or the increase in liver fat, (Wertheimer and Shafrir, 1960). Fat deposition in adipose tissue may result in part from the absorption of plasma lipid components, but mainly appears to be elaborated in the storage tissue itself, by means of cellular enzyme systems. The transient deposition of glycogen-characteristic of the initial stages of lipogenesis, precedes the appearance of triglycerides in the fat storage cells.

The fat content of the adipose tissue has been shown to be dependent on its innervation and the autonomous nervous stimulation is an important factor in the regulation of fat movement into and out of the storage cells, (Shapiro and Wertheimer, 1956; Wertheimer and Shapiro, 1948). Nervous irritation seems to result in a more rapid fat catabolism, (Mallow, 1957). The hormone epinephrine has also been shown to cause a transient increase in the levels of UFA. It appears that under the sympathetic discharge of epinephrine in an emergency condition, the adipose tissue, and not the glycogen stores, most rapidly satisfies the demand for oxidizable substrate. The mechanism whereby a sympathetic discharge causes such mobilization is unknown.

The fat depots of ruminants are little altered by a change in the quality of the diet, (Thomas et al. 1934; Edwards and Holley, 1939), or by nutritional plane, (Hilditch and Pedelty, 1941). The location of adipose tissue in the body influences the fatty acids. Thus the degree of saturation generally decreases from the external depots inwards, and the change is associated with a similar gradient in body temperature, and possibly with a difference in the rate of glyceride deposition, (Callow, 1958)

Passmore et al. (1958) found the composition of the tissue lost by obese patients to be fat 73-83 per cent, protein 4-7 per cent, and water 10-23 per cent by weight, based on indirect results of nitrogen and energy balances. Ljunggren (1957), and Ljunggren et al. (1959), conclude that the tissue gained by overeating in normal and obese patients is similar in composition to that lost with undernutrition.

## (2) Endocrinology

Adequate nutrition and the factors which influence the availability and metabolism of nutritive substances markedly affect the functional integrity of the reproductive system, (Leatham, 1959). The level of UFA is subject to hormonal control. Insulin, glucagon, growth hormone, lipid mobilizing hormone of the posterior pituitary, thyroid hormone, oestrogen, androgen, glucocorticoids, and the medullary hormones of the adrenal are involved, (Olson and Vester, 1960). Keys et al. (1950) concludes that the hypophysis appears to show hypofunction with starvation, being reduced in weight, with possibly a decreased secretion of adrenotrophic and gonadotrophic hormones. Implantation of pituitary glands from normal rats resulted in the adrenal glands and ovaries of starved animals becoming normal in size. Hypophysectomy will prevent weight gain in obese animals, which regress to a lower weight, (Mayer, 1953). Long (1957) has emphasized that it is difficult to elucidate the endocrinology of obesity, because an endocrine disturbance could result from an excessive food intake. Thus the endocrine changes that may occur could be a consequence of the metabolic adaptations that increased or decreased food intake imposes on the organism.

Ovarian development, maturation, and function are known to be inhibited by severe inanition, protein deficiency, or deficiency of certain vitamins, probably lowering the secretion of gonadotrophins, (Ershoff, 1952; Hart et al. 1930), which in turn causes impaired gonadal function. Inanition may cause a state of anoestrus, (Werner, 1939), a decrease in the number of ovarian follicles, (Gilbert, 1942; Huseby and Ball, 1945; and Jackson, 1947), in the female, while in the male the testes show atrophy and loss of motile sperm and the male loses sex interest and vigour, (Asdell and Crowell, 1935; Evans and Bishop, 1922; Macomber, 1933; Mason, 1933).

In the adult human female, Stephens (1944), and Zimmer et al. (1944), showed that ovarian atrophy and disruption of cycles follows underfeeding. A low energy intake, but normal intake of protein in rats, (Scheer et al. 1947; and mice, Ball et al. 1947; Huseby et al. 1945), inhibits the development of reproductive organs and causes irregular cycles and lowered fertility. In cows, underfeeding causes impaired heat symptoms, irregular heat intervals, and decreased fertility, (Moustgaard, 1959; Joubert, 1954). Low planes of nutrition have caused delayed onset of puberty and lowered ovulation rate in sheep, (Clark, 1934; El-Sheikh et al. 1955; and in swine, Stewart, 1934). The ovarian changes induced by inanition may be reversed by refeeding, Ball et al. 1947; Schultze, 1955; Asdell, 1949; Reid et al. 1951; Steinberg, 1947). A reduction in caloric intake may negate the presence of an adequate protein level, (Leathem, 1959). No significant primary effect of androgens on UEA metabolism have been noted, (Olson and Vester, 1960). It has been generally assumed that fatness may be the cause of lowered fertility, (Maynard and Loosli, 1956; Asdell, 1949). It is still an open question whether fat animals are infertile owing to fatness, or whether they are fat because they have a low fertility, or sterile. Persistent overfeeding of ruminants does not lead to increased fertility. An increased supply of concentrate feed for a short period before breeding may however increase the ovulation rate, (Clark, 1937; Nichols, 1926; Reid, 1951). Flushing can apparently increase the lamb crop by up to 20 per cent, (Moustgaard, 1959). Flushing of well-fed ewes does not seem to cause lowered fertility, (Wallace, 1960). The weight gains in the female during pregnancy appear to be primarily due to progesterone, (Dewar, 1957). Keys and Brozob (1955) noted a marked difference in the distribution of fat between the male and female, suggesting important sex hormonal effects on fat metabolism.

Among the body functions known to be affected by the thyroid hormone, (Selye, 1948; Housay, 1955), the primary function appears to be on metabolic rate, probably acting through various enzyme systems, (Barber, 1954). Keys et al. (1950) suggest that the consistent evidence showing that the thyroid gland atrophies on starvation may be the primary cause of metabolic rate declining on inanition. Kirton and Barton (1958) with ewes, were unable to demonstrate a reduction in weight of the thyroid gland during

28 days of undernutrition. It is generally recognized however that underfeeding reduces the secretion of thyroid stimulating hormone (TSH), and thyroid function. Furthermore, various procedures known to cause an increase in adrenocortical activity through a reflex discharge of adrenocorticotrophic hormone (ACTH) from the anterior pituitary, have been shown to decrease the activity of the thyroid gland of rabbits and rats, (Brown - Grant et al., 1954, 1957; Brown - Grant, 1956). Pipes et al. (1960) showed with thyroidal  $^{131}\text{I}$  that underfeeding resulted in a decreased rate of thyroxine secretion in mice. In general, it seems that stress stimuli excite the adrenal cortex and inhibit the thyroid gland, and that this reflects a reciprocal relationship between the secretion of ACTH and TSH, (Harris, 1955). A neural mechanism seems to be involved in this ACTH release, (Sayers, 1957). Recently Gerwing et al. (1958) suggested that in the guinea-pig, monkey, and man stress may produce an increase in thyroid activity. However, Brown - Grant and Rethes (1960) found a decrease in the rate of  $^{131}\text{I}$  release from the thyroid gland of guinea-pigs in response to various stimuli, as well as by large doses of ACTH. It is well known that body weight increase is retarded in hypothyroidism. In a hypermetabolic state the UFA level is increased and fat reserves are mobilized, (Wertheimer and Shafrix, 1960). The effect of the thyroid hormone may act directly or indirectly through the adrenal medulla, or via neurohumoral epinephrines. Solomon and Dowling, (1960) in a comprehensive review suggest that the central nervous system acts as a regulator of the synthesis and/or release of TSH via the portal veins of the hypothalamus, by means of neurohumoral secretions.

Insulin appears to exert its effect on fat metabolism by influencing the metabolism of glucose, thereby stimulating the uptake of glucose by adipose tissue. In the absence, however, of glucose as in fasting, the storage of fatty acids is reduced and the release of UFA accelerated, (Olson and Vester, 1960). The effects of adrenal cortical hormones on UFA levels are inconsistent. Insulin acting synergistically with growth hormone also induces fat mobilization and nitrogen storage, (Greenbaum, 1953; Jungas and Ball, 1960; Randle, 1957). The levels of insulin appear to be

rapidly and markedly reduced in starvation, (Wertheimer and Shafriz, 1960).

Hyperadrenocorticism is frequently associated with obesity, but this disease usually involves hypertrophy of the islets of Langerhans suggesting an increased secretion of insulin, (Hausberger, 1958).

### (3) Intake regulation

Obesity has long been associated with lesions of the hypothalamus, (Meyer, 1953). Artificial lesions, or lesions due to disease, involving the ventromedial hypothalamic nuclei, in the region of the tuber cinereum have been shown to result in a variable expression of obesity, (Brobeck, 1946; Kennedy, 1952; Hetherington and Ranson, 1942; Kennedy, 1957a; Kennedy, 1957b). These workers, together with Andik *et al.* (1957, 1958), have shown that appetite is greatly increased as a result of these lesions, and an increase in weight occurs. The weight increase is largely fat, (Brobeck, 1946; Long, 1957). The mechanism whereby these hypothalamic lesions cause obesity does not operate through the hypophysis, (Hetherington and Ranson, 1942; Brobeck, 1946). Long (1957) considered that food intake is regulated by more than one area in the hypothalamus, and by neural mechanisms that either stimulate or inhibit the desire to eat. Larson (1950) reported that an injection of hypertonic saline into the hypothalamus of sheep and goats causes hyperphagia. There may be a decrease in hypothalamic control of food intake with age, (Kennedy, 1952). The above workers claim to have disproved the hypothesis of Mayer (1953), and Mayer (1955), that appetite is controlled by blood-sugar level. Conditioned reflexes, physical characteristics of food (size, shape, colour, odour), palatability, stress reactions, and psychological factors appear to be of importance in normal food intake regulation, (Lepkovsky, 1948). "Appetite centres" in the central nervous system, and nerves which conduct the impulses to and from the central nervous system have been recognized, (Lepkovsky, 1948). Brobeck (1960) suggests that food intake control is under similar forces to temperature regulation. He postulates that heat sensitive neurons are present in the medial hypothalamus, while a satiety centre is present in the medial hypothalamus, the latter

being controlled by higher nervous mechanisms. He further postulates that feeding reflexes are superimposed with these mechanisms upon integrating centres which are responsible for food intake. This is supported by the observation that short-term exposure to high environmental temperature is followed by a reduction in food intake, (Mayer, 1955). The state of the internal fat depots may also be involved in food intake regulation, (Tayler et al. 1957; Tayler, 1959).

#### (4) Metabolic rate

Blaxter and Wood (1954), have confirmed the observation of Benedict and Ritzman (1927), and Ritzman and Benedict (1938), that basal metabolism declines as the energy intake is reduced. Animals subjected to undernutrition become more economical in their use of food. Houssay (1955) maintains that basal metabolic rate declines gradually with undernutrition. Metabolic patterns and probably endocrine balances are in part genetically determined, (Fenton and Dowling, 1953). Mitchell et al. (1928) with sheep were unable to demonstrate any differences in the amounts of metabolizable energy required for maintenance between normal and undernourished animals. Cresswell (1958) found with Cheviots and Romneys a decreased metabolic rate with food restriction, but noted breed differences. The Cheviots rapidly adjusted their metabolic rates to an equilibrium value, while the Romneys showed no obvious metabolic plateau, the trend being downward but in an erratic manner. Cresswell (1958) quoted evidence which suggests that there is a seasonal fluctuation in metabolic rate, with a fall in the autumn, and a rise in summer, probably following thyroid activity. Long (1957) claimed that the metabolic rate of rats when obese, was about twice that prior to them becoming obese. Keys and Brozob (1953) emphasize that basal metabolic rate per unit mass of non-fat tissue appears to increase during the development of obesity, suggesting that newly formed tissue has a very high basal metabolic rate. Cresswell (1958) found that the Romneys metabolism rose faster on re-alimentation than that of Cheviots, presumably due to genetic differences between these breeds.

(5) Disease

The importance of disease in the aetiology of obesity and leanness has been emphasized by a number of workers. Overweight has been associated with degenerative diseases including those involving the cardio-vascular and renal systems, (Long, 1957), increased mortality, liver cirrhosis, (Mayer, 1953), renal damage, heart enlargement, and the accumulation of liver fat, (Long, 1957). Keys et al. (1950) also emphasize that during undernutrition the albumin fraction of the blood tends to decline resulting in a reduced resistance to infective organisms. Berg and Simms (1960a) found that a restricted food intake was more favourable to health, activity and fertility in aging rats than ad libitum feeding, thus confirming that nutrition is a factor involved in regulating the time of onset of disease. Subsequently Berg and Simms (1960b) showed that longevity was extended and the onset of disease delayed on a restricted diet, with maximum body weight not being optimal in this respect. This indicates a metabolic factor influencing susceptibility of aging tissues to disease and certain tumours.

(6) Genetic factors

Some evidence has accumulated to show that obesity may be partly inherited in farm animals. Fenton and Dowling (1953) showed that certain strains of mice could be relatively easily caused to deposit excessive fat by feeding highly purified diets containing 30 per cent. casein and from 5 to 50 per cent. fat, while other strains did not show this effect. Mayer (1953) emphasized that genetic factors are of importance in the aetiology of obesity. Dickerson (1954) comments that the increased rate of fat deposition due to the gene for "yellow" in mice has been attributed to a hormonal alteration in carbohydrate metabolism, (Weitze, 1940), which increases appetite, and reduces food requirements per unit of gain even though the extra gain is fatty tissue with a high calorific value per unit of weight, (Dickerson and Gowen, 1947). Falconer and Isaacson (1959) have reported the presence of a new recessive gene causing obesity. Hull (1960) with mice showed that selection for high body

weight at three weeks of age increased the proportion of fat. Falconer (1960) emphasized the importance of environment in selection. Mice whose growth was increased by selection on a low plane of nutrition were less fat than those which had been increased by selection on a high plane. Equality of growth was therefore reached by different physiological pathways. There seems no difference in final size between hill sheep which spend their first winter under rigorous conditions and sheep wintered under good conditions, (Purser and Roberts, 1959).

The level of physical activity, because of its high energy cost may exert a profound influence on the production of obesity, (Mayer, 1955). When the temperature is lowered, there tends to be an increased deposition of fat, and fluctuations in ambient temperature could be of considerable importance in producing obesity in domestic animals.

(B) THE EFFECT OF NUTRITION ON BODY COMPOSITION

Several studies of liveweight loss in sheep evoked by undernutrition have been reported, (Robinson, 1948; Franklin, 1952; White et al., 1956; Kirton and Barton, 1958a, 1958b), but only Robinson (1948), and Kirton and Barton (1958) have indicated the components of loss and/or gain of the animals. Consequently information reported on other species of animals is included, although the pathways through which undernutrition affects body composition and indeed the final result, may differ markedly between species.

The effect of undernutrition on the young animal may well differ from that in the aged animal. With aging there appears to be a loss of body protein, the nature of colloids change, collagen density increases, there is a reduced capacity to regenerate, and a general failure of homeostasis, (Comfort, 1954, Sobel and Marmorston, 1958). Current thought does not appear to differentiate between these two ages.

(1) Carcass

Since the effect of nutrition was investigated by Moulton et al. (1921, 1922, 1923) experiments conducted by the Hammond School (Verges, 1939a, 1939b; Wallace, 1948; Palsson and Verges, 1952; with sheep, McMeekan, 1940-41, and Pomeroy, 1941; with pigs)

and more recently Wilson, (1952, 1954<sup>a</sup>, 1954<sup>b</sup>) with poultry, and goats, (1958<sup>a</sup>, 1958<sup>b</sup>, 1960) and Osbourn and Wilson (1960) also with goats, have clarified the effects of nutrition on body composition. The most important findings pertinent to this discussion appear to be as follows:

The different tissues attain their maximum growth rate in a definite order with age, in the order, nervous tissue, bone, muscle, and fat. Fat accumulates in the following order, mesenteric fat, perirenal fat, intermuscular fat, subcutaneous fat, and intramuscular fat, so that growth rate with age follows an outward trend from the central nervous system, bone, tendon, muscle, intermuscular, and subcutaneous fat. During late foetal life to maturity, any part, organ or tissue is most affected by undernutrition at the stage when it has highest growth intensity. The different tissues and body regions are utilized for the supply of energy and protein in the reverse order of maturity, fat first, then, muscle, and bone, these tissues being first depleted in the latest maturing body regions such as the loin and pelvis. Any part, organ, or tissue which has been retarded in development by restricted nutrition, exhibits recuperative capacity if the animal is changed to a high level of nutrition, and may, if undernutrition has not been too severe or prolonged, recover completely. Paschke (1958) has indicated the possible importance of growth promoting factors with high organ specificity in recovery. Little is known of the phenomenon of regression in tissues, but many factors are undoubtedly involved, (Abercrombie, 1957). Child (1920) showed that those tissues, parts, or organs, with a higher metabolic rate had priority over tissues with a lower metabolic rate. Hammond (1944) advanced the theory of the "Priority of Partition of Nutrients According to Metabolic Rate", the metabolic rate being lowest for fat, but increasing in the order - muscle, bone, placenta and foetus, brain and central nervous system. When nutrients are limited the effect will be greatest on fat tissues and least on the nervous system. Direct determinations of oxygen consumption from the rates of blood flow and arterio-venous oxygen differences, indicate that at rest the internal organs use 60 per cent., and the total musculature 25 per cent., of the total oxygen consumed (Brozek, 1955).

The nature and lability of the tissue could be a better explanation of the effects observed.

There is one major point on which the workers of the Cambridge School disagree. McMeekan (1940-41), and Falsson and Verges (1952) agree that undernutrition affects individual organs and tissues of the body differentially, and also that within a tissue some parts are penalized more than others. Wallace (1948) disagrees that there is a differential effect within the tissue and maintains that this is due to McMeekan's method of analysis. Wallace (1948) concludes that undernutrition does not have a differential effect within a tissue, so that within a tissue the priority for nutrients in the blood stream is the same. Falsson and Verges (1952) concluded that plane of nutrition does have a differential effect on different anatomical units of the same tissue. Cochran (pers. comm.) has criticized the regression approach used by Falsson and Verges (1952) on the basis that relative increase in size and not growth rates were compared, and that the results from this type of analysis do not agree with their earlier conclusions using different methods of measurement. Wilson (1957, 1960) maintains that the large difference found by other workers on the carcass proportions of animals reared on different planes of nutrition were largely due to treatment effects on the body fat. The difference in the proportion of fat will obviously effect the proportions of the remaining tissues. He maintains that a better basis of comparison for body composition would be to take animals of equal fatless weight at slaughter. Wilson concluded from his work that there was no orderly sequence of fat deposition comparable to the growth of other organs and other tissues. He claims, as did Brody (1945), and Wallace (1948), that fat deposition is not an integral part of growth. By recalculating McMeekan's (1940-41) data for muscle weights as a percentage of the carcass less dissectible fat, Wilson (1952, 1954a, 1954b) showed a complete reversal of many of the significant results claimed by McMeekan. While this throws doubt on the validity of the methods used by McMeekan, and Falsson and Verges, he does not provide any evidence on the effect of nutrition within a tissue. Terroine et al. (1922-3), and Terroine et al. (1924) early considered that the com-

position of the fat-free body of an adult mammal was almost unalterable by under-nutrition, or by high and low-protein diets. The objection to comparison between treatments being made on the basis of equal fatless weight is that the results are not directly applicable in animal husbandry.

Falsson (1955), and Wilson and Osbourn (1960) emphasize that a low plane of nutrition followed by a high plane appears to result in a greatly increased rate of growth, and probably a slowing down of physiological age. The animals ability to recover from the effects of this retardation is influenced by the following factors; the nature, severity, and duration of undernutrition, the stage of development at the commencement of undernutrition, the rate at which an animal matures, and the pattern of re-alimentation, including the recovery of weight, form, and body composition. Wilson and Osbourn (1960) in a review of 'compensatory growth', conclude that growth usually proceeds at enhanced rates during re-alimentation. This therefore appears to enable a restricted animal to achieve its normal body size and conformation at a later stage of growth. Periods of prolonged or severe restriction, however, can cause permanent stunting, and modification of adult body form and composition. Osbourn and Wilson (1960) found that chickens restricted in food quantity showed a greater increase in liveweight after re-alimentation than chickens that had been mildly restricted. Re-alimentation tended to increase fat. The limits of the undernutrition to which an animal can be subjected and still recover its 'normal' development are at present ill-defined. Jackson (1937) found that restricted rats did not have the capacity to recuperate after undernutrition. Meyer et al. (1956) found a greater proportion of fat in rats restricted in food intake and subsequently fed ad lib. compared to ad lib. fed controls. Subsequently Meyer and Weir (1960) with sheep found that when the level of nutrition was restricted by 84 per cent., sheep recovered rapidly, while greater restriction prevented recovery. Restricted feeding with rats had a more severe effect on their fat and they also had a higher death rate than similarly treated sheep. Re-alimentation in this study increased the fat content in both species. Summers and Fisher (1960) found that when chickens

were placed on a nitrogen free diet, carcass nitrogen was lost, but carcass fat and liver fat increased. On repletion to the starting weight there was a lag in the gain of carcass nitrogen. Widdowson et al. (1960) studied the effect of under-nutrition and re-alimentation on pigs, but interpretation of their results was difficult because they used four animals only.

Mitchell et al. (1928) compared the composition of sheep which had been well-fed and were then emaciated. They found that bone fat was the last of the fat depots to be depleted. Pomeroy (1941) with pigs, claimed that subcutaneous, intermuscular, perirenal, and omental fats decrease with undernutrition. He maintained that the effect of a submaintenance diet on fat depots was greater than on muscle, or bone, the later developing body regions being most affected. Subcutaneous fat, a later-developing fat, is penalized more than the intermuscular fat. Pomeroy (1941) showed that the subcutaneous and intermuscular fats of the late-developing joints are more reduced by undernutrition than the corresponding fats in the early-developing joints. Robinson (1948) with mature ewes on either super- or sub-maintenance diets, found with the submaintenance animals that fat loss was at first rapid and then declined, while with the supermaintenance ewes fat increase was slow at first, and then increased rapidly. His data indicate that the subcutaneous:intermuscular fat tended to increase as also did the perirenal, mesenteric, and omental fats. He supports previous work in that the effect on the three main tissues was in reverse order to their rate of development. Widdowson and McCance (1956) showed with rats that undernutrition markedly reduced fat, the effect being greater in females than males. Palsson (1955) maintained that males seem to be more affected by restricted nutrition than females. Gern and Brozek (1956) using a soft tissue teleoroentgenograph technique with humans, concluded that those parts of the body with greatest initial fat thickness, or those individuals with greatest amounts of fat, showed greatest losses of fat when on a low calorie diet. Keys and Brozek (1953) conclude that when body weight is lost simply as a result of reduced calorie intake it is commonly believed that this loss represents a decrease of fat, provided appreciable amounts of 'depot fat' remain

in the body. In the early stages of gaining weight from the emaciated state the solid tissue formed may have almost half of its weight in the form of non-fat cellular matter. Later the greater proportion of the weight gain appears to be pure fat. It is not known whether new cells are actually formed. Riney (1955) with deer, showed that fat reserves were depleted in undernutrition, mobilization of fat reserves being in reverse order to that of deposition. Kirton and Barton (1958) with mature ewes on a submaintenance diet, and with various levels of thyroxine given over a 28-day period, were unable to show statistically significant treatment effects on either dissected, or chemical fat of the carcass, the leg, or the 9-10-11 rib cut. This appeared to be due to the high variability of fat content, and to the relatively small numbers of animals in each treatment group. No effect was noted by them on perirenal fat.

The effect of nutrition on muscular tissue will be discussed in Part III.

Pomeroy (1944) with pigs found that prolonged undernutrition decreased the weight of bone, although this effect was less than that observed with fat and muscular tissue. Younger animals have a greater proportion of their bone in a more labile form than mature animals. Consequently the level of nutrition could be expected to have a greater effect on younger animals. Trowbridge et al. (1918) concluded that the skeletal fat would be withdrawn only under conditions of extreme low level of nutrition. Robinson (1948) found that a submaintenance ration did not decrease the total weight of bone in mature ewes. Keys et al. (1950) maintained that bones show a loss of weight on starvation. Kirton and Barton (1958) were unable to demonstrate a loss of weight in the leg bones with 28-days of undernutrition. Pratt and McCance (1960) with cockerels found that undernutrition reduced weight, disturbed ossification, and mitotic division ceased. Mendes and Waterlow (1958) reported that rats fed a poor-quality, low protein diet continued to build collagen even though body weight did not increase. Harkness et al. (1958) with mice found that collagen was lost from the body of mice with a decrease in weight.

(2) Internal organs

There appears to be justification for a greater emphasis to be placed on a more critical study of changes induced in the internal organs. Collectively these organs comprise a high proportion of the body weight and changes would aid in the physiologic interpretation of nutritional experiments.

Jackson (1932) noted that the liver was reduced in rats on underfeeding. Keys et al. (1950) concluded that undernutrition caused a large reduction in the weight of the liver, which appears to be one of the most susceptible organs to a reduction in food intake. This is probably due to the organ's high metabolic activity, (Dukes, 1951) and the high proportion of labile material present. This observation is supported by Pomeroy (1941) with pigs. Robinson (1948) found that the greatest part of the 26 per cent. decrease in liver weight of the control ewe occurred initially during submaintenance. A rapid increase in weight was noted with ewes on a supermaintenance diet. Widdowson and McCance (1956) with rats report a decrease in liver weight on inanition. A reduction of 24 per cent. in liver weight was noted by Kirton and Barton (1958) with undernutrition. Ferguson (1954), and Fenton and Dowling (1953), with sheep and mice respectively emphasized that fatty infiltration during starvation could mask the weight changes of the liver. This accumulation of fat in the liver may be the result of excessive amounts of fat in the diet, production of endogenous fat, and starvation, (Shorland, 1955). Anterior pituitary extracts accelerate fat deposition in the liver. The additional fat in the liver appears to come from depot fat. There is a marked decrease in the size and microscopic appearance of the liver with fasting, (Kosterlitz and Campbell, 1945) and the organ readily contributes to the protein pool. Litwack et al. (1952), with Benton et al. (1955) emphasized that the percentage of liver fat increased by feeding low levels of protein and that the extent of fat accumulation was influenced by the type and quality of the protein source.

In their comprehensive review Keys et al. (1950) emphasize that the kidneys undergo atrophy during inanition. Pomeroy (1941) also reports that 20-30 per cent. of the weight of the kidneys was lost on a submaintenance diet. Robinson (1948) has

shown that the kidneys increased and decreased in weight, with super- and sub-maintenance diets respectively.

Large reductions in the weight of the heart in starved animals has been noted by Keys et al. (1950). Starvation seems to cause a marked softness, paleness, and flabbiness, the gross appearance being that of anaemia and oedema. Apart from these gross morphological changes there appears to be little information on the effects of inanition on the heart or blood vessels. Pomeroy (1944) observed that the weight of the heart fell abruptly to 79.4 per cent. of the controls during the first 23 days. He considered this decrease to be due to a loss of fat and muscular atrophy. Kirton and Barton (1958) observed that a short period of undernutrition significantly lowered the heart weight.

Keys et al. (1950) conclude that undernutrition results in a loss in weight of the spleen, although it was highly variable. Several workers, (Jackson, 1913; Stewart, 1919; Kudo, 1924; and Jackson, 1915), have reported that the spleen is markedly reduced by inanition. Robinson (1948) with sheep was unable to demonstrate any marked effect of undernutrition on this organ. This observation was supported by Kirton and Barton, (1958). They suggested that the weight of the spleen in the sheep is more resistant to undernutrition, or alternatively that the degree of inanition was less severe than imposed by Keys et al. (1950). Robinson (1948) however noted an increase in weight with his supermaintenance ewes.

Widdowson and McCance (1956) reported an increase in the weight of the adrenal glands in rats during undernutrition, while Robinson (1948) observed considerable adrenal atrophy. Hypertrophy was also noted in the adrenal gland by Keys et al. (1950) on starvation, but the results were inconsistent.

### (3) Gastrointestinal tract and its contents

The smooth muscles of the gastrointestinal tract appear to respond to starvation in a similar way to striated muscles, (Keys et al. 1950). Weight losses of 57 per cent of the gastrointestinal tract were found with starved rats, (Jackson, 1915). Pomeroy (1944) showed that pigs on a submaintenance diet show a loss in weight of the

stomach and intestines, with the small intestine appearing to decrease both in length and by a thinning of the intestinal wall. Robinson (1948) tends to support these observations. Kirton and Barton (1958) reported that thyroxine treatment lowered the weight of the empty gastric tract, while a low plane of nutrition reduced both the empty gastric, and intestinal tracts.

Wilson and Osbourn (1960) concluded that re-alimentation results in a prolongation of the period of growth to mature weight, and an increase in the rate of gain during the re-alimentation period, especially during the early stages of re-alimentation. There is considerable divergence of opinion as to whether the increased rates of gain of re-alimentated animals are true increases in body tissue, or due to increased gut contents.

Even when ruminants are fed alike the weight of "fill" may differ markedly between individuals, (Ritzman and Benedict, 1938). The digestive "fill" is influenced by the character and volume of the feed (Hopper, 1944), and the period of fasting. The effect of fasting on the physical changes in the contents of the alimentary tract appear to be largely a substitution of dry matter by water, (Ritzman and Benedict, 1938). Nevens (1928) also found that large amounts of free liquid were present in the rumens of dairy cattle fasted for 4 to 6 days. Benedict and Ritzman (1923), and Winchester and Morris (1956), maintain, however, that the water intake is a function of dry-matter ingested. Phillips (1960) with steers showed that the water drunk to hay eaten is significantly changed when the availability of either food or water is restricted. Restricting the hay eaten increased the water consumed. Quin (1943) also noted an increased wateriness of rumen contents in sheep on starvation. McMeekan (1940-41) showed that the rate of increase in body tissue of the low-high group of pigs was no different from that of the high-high group during recovery. He concluded that the higher rate of gain in weight observed in the low-high group was due to increases in gut content. Taylor et al. (1957) with cattle, have shown that the carcass gains for re-alimentated cattle, restricted during the winter period, were 40 per cent. greater than the carcass gains of cattle growing at a constant rate during winter and the following spring. The lower intake of cattle previously grown on a high plane of

nutrition may be due to the higher amount of internal fat physically limiting food intake, (Tyler et al. 1959). Increased gut contents may exaggerate the rate of gain shown by re-alimentated animals.

#### (4) Food utilization

Blaxter and Graham (1955) have shown that the net energy content of a food declines as the energy intake decreases. Blaxter et al. (1955) demonstrated that increasing the level of feeding resulted in an increase in the passage of food, and a fall in its digestibility. Blaxter (1944, 1950), emphasizes that foods of high nutritive value are habitually consumed in larger amounts than those of low value. Grinding hay results in marked effects on ruminant microorganisms, increasing fatty acid production, body weight gain, digestibility, and the efficiency of food utilization, (Shaw et al. 1960). The power to ferment glucose is suppressed during undernutrition in sheep (Quin, 1943). The physical form of the food can modify the rate of passage of food through the digestive tract, (Balch, 1959-60; Blaxter et al. 1956). Raymond et al. (1954) found that digestibility slightly improved with the age of animals. Graham et al. (1959), and Armstrong et al. (1959) have stressed that environmental conditions, particularly of closely clipped sheep, can markedly effect the utilization of food energy. Some workers claim an increased efficiency of growth if periods of undernutrition and re-alimentation are used. Reid (1955) suggested that this may be due to a greater amount of muscular tissue being formed. Wilson (1960) found, however that re-alimentated goats often deposit more fat in the body than the controls fed on a high-plane throughout. Wilson and Osbourn (1960) concluded in their review, that an animal on a restricted diet followed by re-alimentation is no less efficient than a continuously grown animal, providing it does not lose weight, and is placed on an ad libitum diet during re-alimentation.

## CHAPTER III

### MATERIALS AND METHODS

This chapter will be presented in the following parts:

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## CHAPTER III

### MATERIALS AND METHODS

#### (1) Selection of animals and pre-experimental management

In the selection of animals it was necessary to assume that animals of the same liveweight would have the same dressing-out percentage, and therefore carcass weight. Although not strictly true over the range of fatness likely to be present in a sample of ewes, this assumption is necessary as it was planned to apply treatments to groups of ewes with the same initial carcass weight. With randomization this condition should be approximated closely.

Fifty Romney-crossbred ewes (mainly 4- and 5-year-old) were selected on 22 December 1959 from the Massey College flock. The initial range in liveweight of these ewes was 140 lb. to 182 lb., giving an average liveweight of 162.2 lb. These animals were grazed on pasture until the commencement of the experimental period on 2 January 1960. The mean liveweight of the sheep at this time was 160.8 lb. The loss in weight was thought to be due to feeding conditions, and to the fact that they had eaten little prior to the second weighing. The range in liveweight at the second weighing was from 133 lb. to 185 lb.

These animals were randomized on a restricted weight basis into six treatment groups (C; LPS; LPS:HP; LPL; LPL:HPS; LPL:HPL), giving 8 animals per group. The number of animals per treatment group was limited by the feeding facilities available and the meat laboratory procedure. At the commencement of the trial there was a  $\pm 2$  lb. difference in liveweight between the group means. Two spare animals (nos. 9 and 259) were chosen on the basis of their liveweights being nearer to the mean of all animals.

All sheep were drenched with phenothiazine before the pre-trial feeding period since it is generally recognized that parasitic infestation may affect the composition of the blood.

The pre-trial feeding period was designed to enable the animals to become accustomed to the environment and feeding routine, and to standardize the composition

of the food available so that the digestive tract contents would be comparable between the various groups of ewes.

(2) The design of the experiment

Table 1 shows the experimental design, slaughter dates, and duration of feeding for each group. The experiment commenced on the 4 January 1960, after a pre-trial feeding period of two days. The appropriate abbreviations for each group are also indicated in the table and these will be used throughout the text.

TABLE 1. Treatment groups. (Eight sheep per group)

<u>Group</u>	<u>Level of Nutrition</u>	<u>Date Slaughtered</u>	<u>Duration (days)</u>
C	control beginning	5.1.60	-
LPS	low plane of nutrition for 21 days	26.1.60	21
LPS:HP	low plane of nutrition for 21 days then re-alimentated	3.1.60	29
LPL	low plane of nutrition	16.2.60	42
LPL:HRS } LPL:HPL }	low plane of nutrition for 42 days then re-alimentated	23.3.60	78

Fifty ewes were allotted to the six treatment groups (C; LPS; LPS:HP; LPL; LPL:HRS; LPL:HPL), with two spare animals (nos. 9 and 259). The design of the experiment was to allot eight ewes to the control group C, and subject all the remaining animals to a low plane of nutrition for a period of 21 days. At this stage the sheep in group LPS were slaughtered, and the animals in the LPS:HP group were to be re-alimentated to a liveweight comparable to the control animals. Previous work, (Kirton and Barton, 1958a) had suggested that a low plane of nutrition for 21 days or more would affect body composition. Re-alimentation was studied to provide information on the effects of a high plane of nutrition following a period of submaintenance feeding. Groups LPL, LPL:HRS, LPL:HPL, were subjected to a further period of 21 days on a low plane of nutrition. Group LPL animals were then to be killed, and groups LPL:HRS, and LPL:HPL re-alimentated to a comparable liveweight with groups LPS and C ewes respectively.

Due to reasons to be discussed in Chapter IV this experimental design was subsequently modified.

Translated into New Zealand farming practice the period of 21 days on a low plane of nutrition would be similar to that experienced by large numbers of ewes after weaning, during facial eczema conditions, or during drought periods. A longer period of undernutrition would be comparable to an extended period of these conditions and enable a more critical assessment of the effect of a prolonged low plane of nutrition on body composition. Re-alimentation after 21 days of low plane feeding would tend to simulate the rising plane of nutrition of ewes under pasture conditions where increased pasture growth in the autumn enables a higher plane of nutrition to be provided.

(3) Management during the experimental period

The animals were confined indoors in a shed with a grating floor throughout the experiment. Within this shed, four pens were available, the allocation of animals to the four pens being made on a random basis. Feed was made available in five 2' x 6" feeding troughs per pen, while water was always available ad lib.

The objectives of feeding the animals indoors were as follows:

- (a) To control the feed intake.
- (b) To improve the accuracy of liveweight data as the fleeces were always dry.
- (c) To obtain a closer appraisal of the animals reactions than would be possible under grazing conditions.
- (d) To supply a ration with bulky characteristics thereby counteracting to some extent involution of the stomach and its possible effect on lowered appetite.
- (e) To reduce the incidence of footrot and internal parasitism, both known to affect the blood composition.

It was not intended to study the effect of a low intake of lucerne chaff per se, but to study the effect of a change in liveweight induced by a low food intake on the ewe.

The liveweights were taken every other day throughout the experiment. Animals were fed twice daily, and during these feeding periods each sheep was closely examined for signs of abnormality. In the early stages of the experiment the liveweight of the animals was employed as the main criterion of response. Liveweights obtained in other experiments have not always been satisfactory. Several authors (Lush et al. 1928; Reid, 1956; Whiteman et al. 1954; Schalk and Amadon, 1928; Bean, 1948; Patterson, 1947; Hughes and Harker, 1950; Koch et al. 1958; Baker and Giulbert, 1942; Haridins and Titus, 1939; Mitchell, 1944; Hutchinson, 1947; French and Ledger, 1957) have discussed the errors involved, and the inadequacy of using liveweight changes as a criterion of response. The previous nutritional history will also influence the response obtained, (French and Ledger, 1957; Wilson and Osbourn, 1960). It appears, however, that the procedure of weighing the animals before the morning feed, with water available ad lib., is likely to reduce the errors involved in using liveweight as a criterion of response to a minimum.

#### (4) Level of nutrition

It was considered desirable to vary the quantity rather than the composition of the ration. Since the experimental period was short and the number of animals small the treatments imposed were extreme. During periods of high plane feeding the aim was to secure a high rate of liveweight increase, while during the low plane of nutrition the energy intake was low. The bulk of the ration had to be maintained at a reasonable level so that rumination could proceed normally. Few suitable concentrate feeds were available. For these reasons chaffed, good quality, lucerne hay was chosen. The sheep found this feed palatable and ate it readily.

Water was always available ad lib. It was hoped that this would tend to prevent involution of the gastrointestinal tract during low plane feeding, and enable a full intake of day lucerne chaff with high plane feeding. The daily ration was given at two feeding times, viz. between 5 and 7.30 a.m., and between 5 and 6.30 p.m.

From the 2 January to 5 January the animals were allocated at random into four pens and fed an ad lib. diet. Group C were given lucerne on the morning of the

4 January only. At 4 p.m. that day they were transported to the College Abattoir, and were slaughtered the following afternoon. The sheep in all groups were treated in the same manner prior to slaughter.

On the 5 January all remaining animals were re-allocated to the four pens and fed twice daily, 1 lb. of lucerne chaff per sheep. This feeding level was gradually reduced to 0.45 lb./sheep/day by the 14 January. From the 15 January to 24 January all sheep were fed at a level of 0.30 lb. lucerne chaff per day. On the 26 January group LPS were slaughtered. The animals in the LPS:HP group were allocated to one pen, and the remaining groups (LPL; LPL:HPS; LPL:HPL), were allocated at random to the three remaining pens.

The ewes in the LPL, LPL:HPS, LPL:HPL groups were fed 0.30 lb. lucerne chaff per day over the period 25 January to 15 February, except on three days when the ration was increased to 0.40 lb. per day in an attempt to prevent further deaths.

To compensate for the distinct bias evident in favour of the fast and bold feeding sheep, no's. 15, 144, 149, 180, 229, 424, 438 and 538 were separated into one pen. These sheep had lost less than four lb. in weight during the period 18 January to 26 January. They were fed at the same level as the other sheep. Over this period it was also necessary to hand feed intermittently the following sheep; no's. 261, 257, 538, 558, 255, 84/52, 154, 4, 499, 100, 630, 288, 84/47, 180, 149, and 15. If a sheep was not eating readily the lucerne available, the following procedure was adopted; hand feeding of lucerne, chamoellier leaves, grass and clover, and if this was not successful, the animals were turned out on pasture for short periods. Despite this procedure the following sheep no's. 261, 538, 499, 255, 4, and 257 died.

Animals in the LPL group were slaughtered on the 16 January.

Following the slaughter of the ewes in the LPL group, those ewes in the LPL:HPS, and LPL:HPL groups were fed ad lib. These sheep ate about 2 lb. lucerne plus 1 lb. grass and clover, or 2 lb. lucerne plus 1 lb. chamoellier leaves per head per day. On six days, periods from 15 minutes to 12 hours were permitted on pasture. Despite this treatment it was necessary to force feed sheep no's. 149, 424, 434, 100 and 154.

during re-alimentation. Although sheep no's 558 and 76 died during this period, all other sheep remaining in these two groups appeared to have regained their appetite by 26 February.

From the 25 January to 30 January the ewes in the LES:HP group were given an ad lib. lucerne diet; the average consumption being approximately 2 lb. per sheep per day. From the 31 January to 9 February these sheep were fed about 2.5 lb. of lucerne chaff plus 2 lb. chouscellier leaves per sheep per day. On the 10 January this group was subdivided into two lots. One lot comprised of no's. 434, 542, 253 and 150, which had the following liveweight changes, 0, -10, -3, and +5 lb. from 24 January to 7 February respectively. The second lot comprised the remaining sheep in this group. The object of this was to reduce the variation in liveweight response by differential feeding. The second lot of ewes were restricted to a level of about 3 lb. lucerne chaff per sheep daily until slaughter. The first lot of ewes were given ad lib. lucerne, grass and clover, chouscellier leaves, and on four days pasture grazing. Sheep number 542 died on 23 February. On the three days prior to slaughter on the 3 March the second lot were given 4 lb. lucerne per sheep daily, and the first lot 3 lb. lucerne plus 1 lb. grass and clover per sheep daily.

#### (5) Slaughter techniques and records

Sheep to be slaughtered were starved of food and water for 23-29 hours prior to the commencement of killing, to enable the partial emptying of the alimentary tract. The animals were weighed immediately prior to slaughter, killed by cutting the throat, and the carcass dressed according to normal commercial practice, with the exception that the kidneys and thyroid glands were removed.

The following information was recorded for each ewe:

1. Liveweight
2. Eled wt.  
Blood wt. (by difference)
3. Feet wt. (four feet together)
4. Skin wt.

5. Head wt. (with tongue)
6. Hot carcass wt. (minus kidneys)
7. Wt. left fore cannon bone
8. Wt. stomach and oesophagus (full)
9. Wt. stomach and oesophagus (empty)  
Wt. stomach contents (by difference)
10. Wt. small and large intestines (full)
11. Wt. small and large intestines (empty)  
Wt. intestinal contents (by difference)
12. Wt. heart
13. Wt. lungs and trachea
14. Wt. spleen
15. Wt. liver
16. Wt. omental (caul) fat
17. Wt. mesenteric (gut) fat
18. Wt. kidneys
19. Wt. genital tract plus urinary bladder
20. Wt. pancreas
21. Wt. rest (miscellaneous pieces of skin)

The heart was removed by cutting the aorta, cleaned of blood, weighed, and sealed in bottles. The thyroid glands together with associated tissue were removed. Disease symptoms present in the internal organs were also noted. Six persons were required in the collection of these data. After slaughtering and dressing were completed, the carcasses were hung in a chiller on standard gambrels.

#### (6) Post slaughter techniques

On the morning following slaughter the carcasses were removed from the chiller, weighed to determine the cold carcass weight, and the following external measurements taken:

F	=	leg length	G	=	width of gigots
W.th	=	width of thorax	W.F.	=	width of forequarter.

These measurements were taken with steel dividers and scaled off on a wooden millimetre scale. The carcasses were then transported to a local plant for freezing and storage.

Each heart (less tendinous tissue) was cut into small pieces, macerated, and two samples each of 50 g. were taken for chemical analysis. Care was necessary to prevent the material from becoming too fine.

The thyroid glands were dissected out and weighed.

(a) Treatment of the whole carcass and its right side

The procedure followed in the present study differed in several respects from previous investigations and therefore will be described in detail. Normally three carcasses were brought from the cold store on Monday, and three on Wednesday. The dissection and chemical work each week was completed by Saturday evenings. Most of this work was done by two persons.

The frozen carcass, after weighing, was divided down the middle of the vertebrae with a meat bandsaw, and each side weighed. This procedure assumes the absence of any departure from bilateral <sup>or</sup> symmetry. Careful splitting of the carcass and the standardisation of procedure should result in errors being reduced to a minimum, (Butler *et al.* 1956; Lasley and Kline, 1957; Hankins, 1953). Following this the left side was allowed to thaw. The right side was then cut into slices approximately  $\frac{1}{4}$ " thick using the bandsaw. The slices were ground, first with the coarse plate, and twice with the fine plate, care being taken to thoroughly mix the ground material. During the third grinding the tissue was sampled at regular intervals to give a total sample of 4-6 lb. This sample was then reground twice. Six samples, each of 50 g., one from each corner and two in the centre of the tray were then weighed out, care being taken to draw material from the bottom of the tray.

(b) Left side

After thawing the leg, loin, and 9-10-11 rib-cut joints were removed from the left side of the carcass, following the method described by Falsson (1939), and each

joint weighed.

Measurements A, B, C, D, E, and X, were made on the half-loin, as described by Falsson, (1939).

Details of the dissection of these joints were as follows:

From the 9-10-11 rib-cut the following tissues were dissected and weighed: skin muscle, subcutaneous fat, intermuscular fat, muscle, and the bones.

Tendon and waste was also weighed as a composite. Muscle, subcutaneous fat, and intermuscular fat were chemically analysed as separate tissues. The vertebrae and ribs were discarded. The time taken by two persons for the dissection of the 9-10-11 rib-cut was approximately 25 minutes.

The leg was dissected into mammary gland, subcutaneous fat, intermuscular fat, muscle, the separate bones of the leg, tendon and waste, and each component weighed. The mammary tissue was added to the subcutaneous fat for chemical analysis. Subcutaneous fat, muscle, and intermuscular fat of the leg were separately sampled for chemical analysis.

The half-loin was dissected into kidney fat, subcutaneous fat, intermuscular fat, longissimus dorsi muscle, psaos muscles, skin muscle, and each component weighed. The tendon and waste, and the vertebrae after weighing were discarded. The number of vertebrae were recorded. The various muscles of the loin, together with the remaining parts of the longissimus dorsi after sections were removed, and bulked to give the general muscle of the loin. Chemical analyses were carried out on perirenal fat, subcutaneous fat, intermuscular fat, muscle (general) and the longissimus dorsi sample.

All muscle samples were ground thrice and two samples, each of 50 g. were taken. Each dissected fat depot was ground once only before sampling, since further grinding produces a sticky mass, which is difficult to sample.

(c) Chemical analysis

A detailed description of the chemical procedure has been adequately described elsewhere, (Barton and Kirton, 1956; Kirton, 1957; Kirton and Barton, 1958a) and

will not be repeated here. Kirton (1957) presents a numerical example of this procedure. The grinder was dismantled, and cleaned after each sample. As a further precaution the first material to be ground was discarded. To enable correction factors for chemical fat, and ash to be applied, the "crude residue" for each dissected tissue was bulked from each group of animals, so that the following records were available: heart, bandsaw side (six samples per side), muscle of the leg, loin, and rib-cut, longissimus dorsi, intermuscular fat of the leg, loin, and rib-cut, subcutaneous fat of the loin, leg, and rib-cut, and the perirenal fat. Within each group of animals two Soxhlet, and two ash correction factors were obtained. The weight of "crude residue" minus the fat correction weight, and minus ash weight, gives an estimate of the amount of protein in each 50 g. sample. Ulyatt (1960) found that protein estimated by difference and by the standard Kjeldahl method was not significantly different. It appears that for this type of work the "difference method" of estimating protein is sufficiently accurate.

The chemical procedure used appears to be very satisfactory for this type of work for the following reasons:

1. The tissue is thoroughly disintegrated and mixed before sampling to ensure homogeneity and complete extraction.
2. The procedure is simple, and accurate, and the estimations of water, fat, protein, and ash content are determined rapidly.
3. Chemical components of the carcass can be estimated in approximately 3-man hours compared with the estimated 72-84 hours (Palsson, 1939), required to completely dissect a carcass.
4. Estimates of water, fat, protein, and ash are obtained on the same sample.
5. Since the water and fat content of adipose tissue tend to be related (Brozek and Keys, 1953) it is essential that fat estimation be related to dry weight.
6. Filtration, as proposed by Bloor (1924) is avoided.
7. Non-lipid impurities are not removed from the extracts before the lipids are estimated, but this is considered to be unimportant in the present investigation. If this were necessary however, the non-lipid impurities extracted with the lipids:

during decantation, and subsequent extractions could be separated and added to the "crude residue" for ash and protein determinations.

8. As far as possible, evaporation of water is avoided during grinding and weighing of the tissues.
9. Minute quantities of fats, insoluble in ether may be present in the tissues. These solubility deviations, as with most other methods, have a slight influence on the accuracy of ether extraction, (Kelly, 1953).

The oven drying method of determining water content appears to be satisfactory provided that the time and temperature are standardized, (Willits, 1951).

More complete extraction of lipids could possibly be obtained by using a 50:50 mixture of ethyl alcohol and diethyl ether.

#### (7) Statistical methods

The analysis of variance technique as described by Snedecor (1959), was used to test the significance of the treatment effects. When significant treatment effects were present, the difference between the group means were tested using Duncan's Multiple Range Test, (Duncan, 1957).

The IPL:HPS and LPL:HPL groups were excluded, first, because much of the data from these groups were not strictly comparable to that which had been collected from the remaining groups and, secondly, because four sheep in the LPL:HPS group and two sheep in the LPL:HPL group died and another was killed in extremis. Two sheep in the LPL group also died but these were replaced by the two spare animals.

The analysis of variance is based on the assumption that the residual effects are not correlated, are normally distributed, have the same variance and a mean of zero. Percentages have a multinomial distribution and therefore theoretically this invalidates the analysis of variance technique. Experience has shown however, that where the probabilities involved in a multinomial distribution are near neither zero or 100 per cent., the analysis of variance and tests of significance are not greatly distorted, (Cochran and Cox, 1950). Therefore in the present experiment percentage data were analysed by analysis of variance.

## CHAPTER IV

### RESULTS

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CHAPTER IV

RESULTS

(1) Treatment effects on liveweight and animal health

(a) Liveweight

The group means of the loss and gain in liveweight of the animals that survived to slaughter are presented in Table 2.

TABLE 2. Mean liveweight loss and gain of the treatment groups (lb.)

<u>Group</u>	<u>No.</u>	<u>Mean Weight Loss (Range)</u>	<u>Time (Days)</u>	<u>Mean % Weight Loss</u>	<u>Mean Gain in Weight (Range)</u>	<u>Time (Days)</u>	<u>Mean % Weight Gain</u>
LPS	8	15.1 (9-21)	20	10.0	-	-	-
LPS:HP	7	12.8 (9-16)	20	8.6	10.0 (2 to 21)	38	+7.3%
LPL	9	27.7 (21-33)	42	18.9	-	-	-
LPL:HPS) LPL:HPL)	9	28.2 (20-39)	42	18.9	-2.5 (-21 to +17)	34	-2.0%

The change in liveweight has been estimated on the basis of the liveweight prior to the pre-slaughter starvation period, and therefore does not correspond in all cases to the length of the treatment period.

The mean liveweight of all experimental animals for the six groups are given in Fig. 1. The group means for those animals surviving to slaughter date are presented in Fig. 2. The two spare animals (no's. 9 and 259), together with ewe no.15 were included in the LPL group at the time of slaughter. Ewe no.15 was included in this killing group as it was in extremis. Its liveweight loss was comparable to the other ewes in the LPL group.

The analysis of variance of the pre-slaughter liveweight of each group is given in Appendix I. Both a short (LPS) and a prolonged (LPL) period of undernutrition significantly reduced liveweight. The animals in the LPS:HP group appeared to gain in weight following a 21-day period of undernutrition. The LPS:HP group was not

FIG. I.  
GROUP MEAN LIVWEIGHT (LB)  
(ALL ANIMALS)

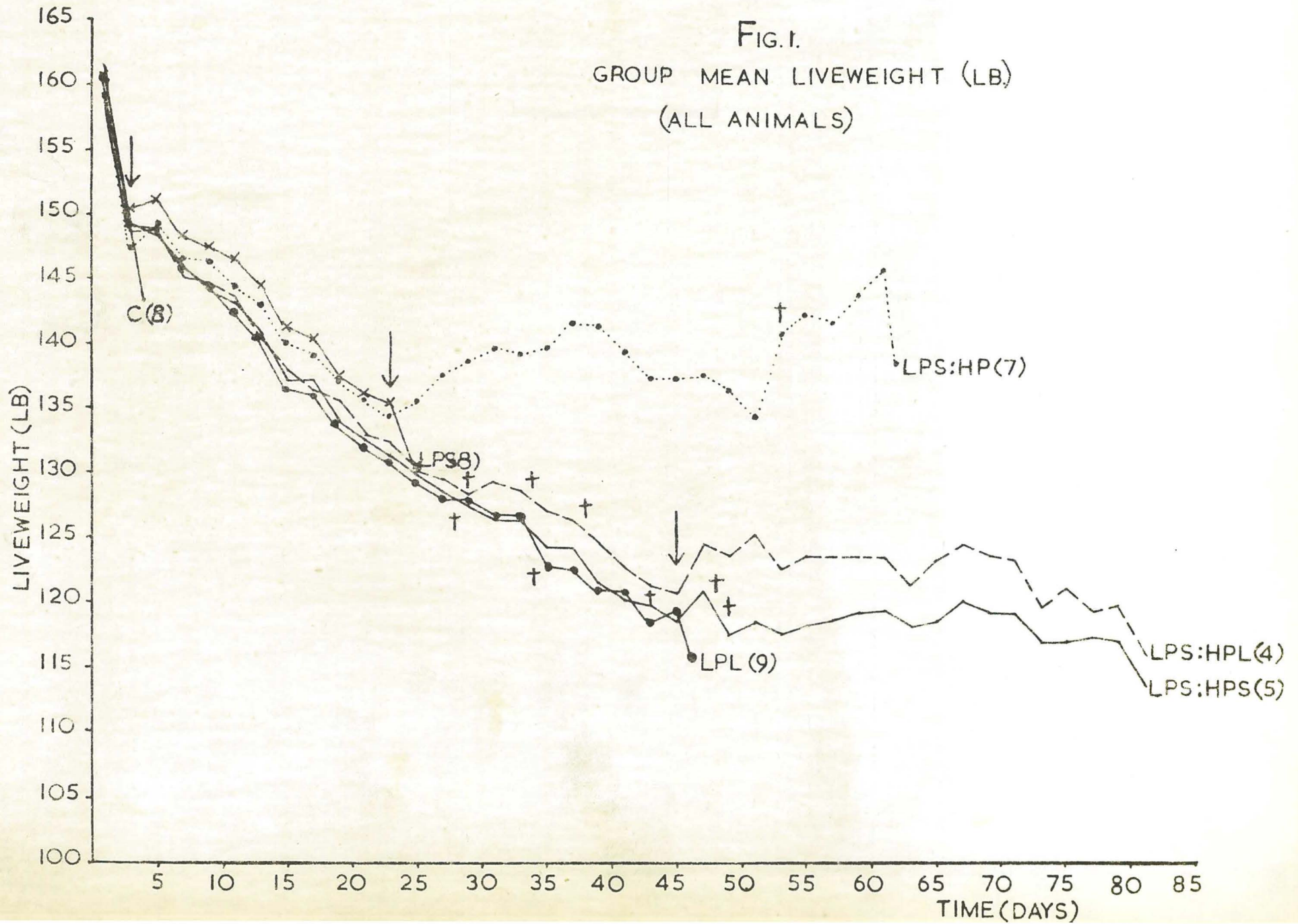
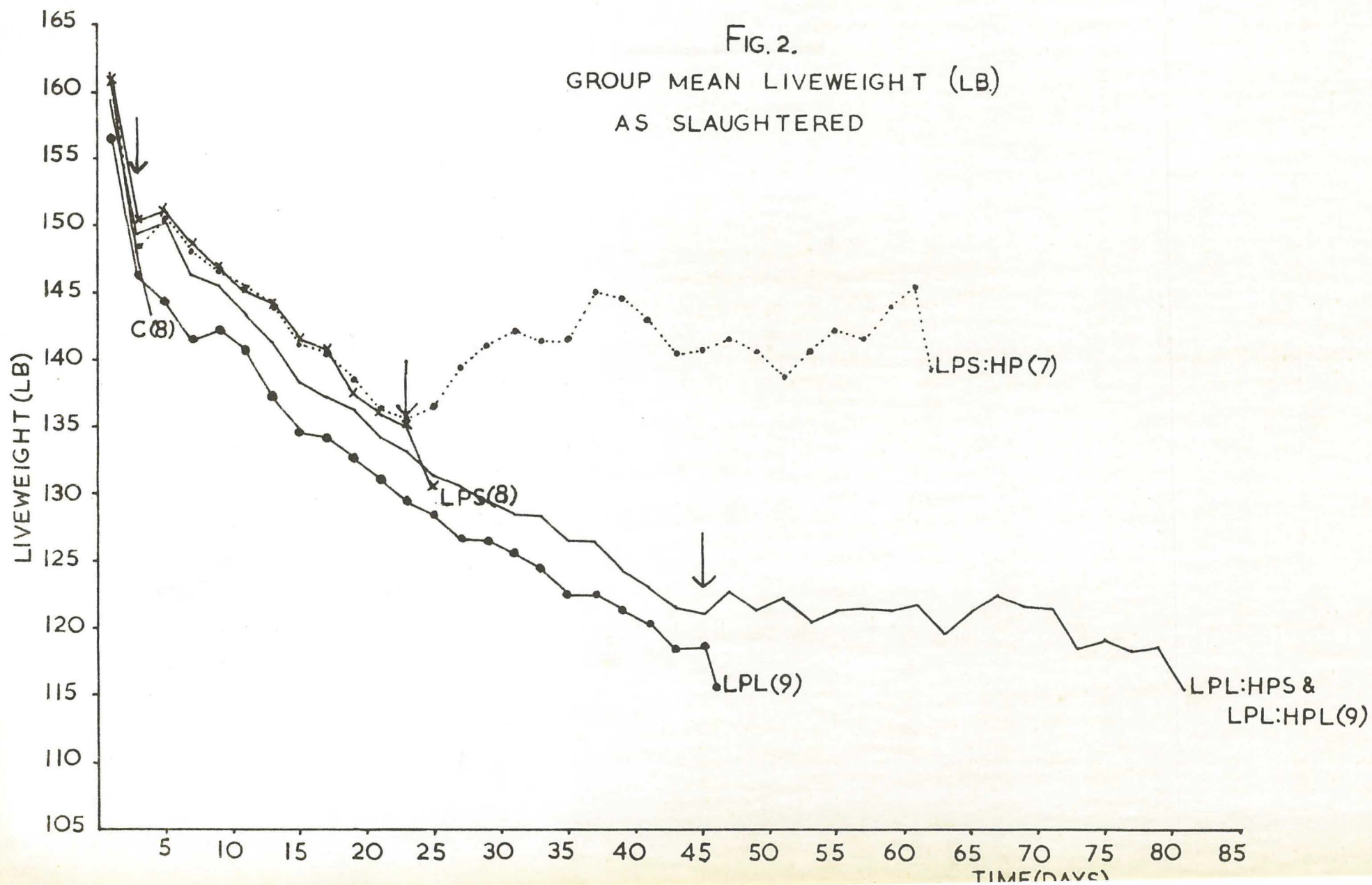
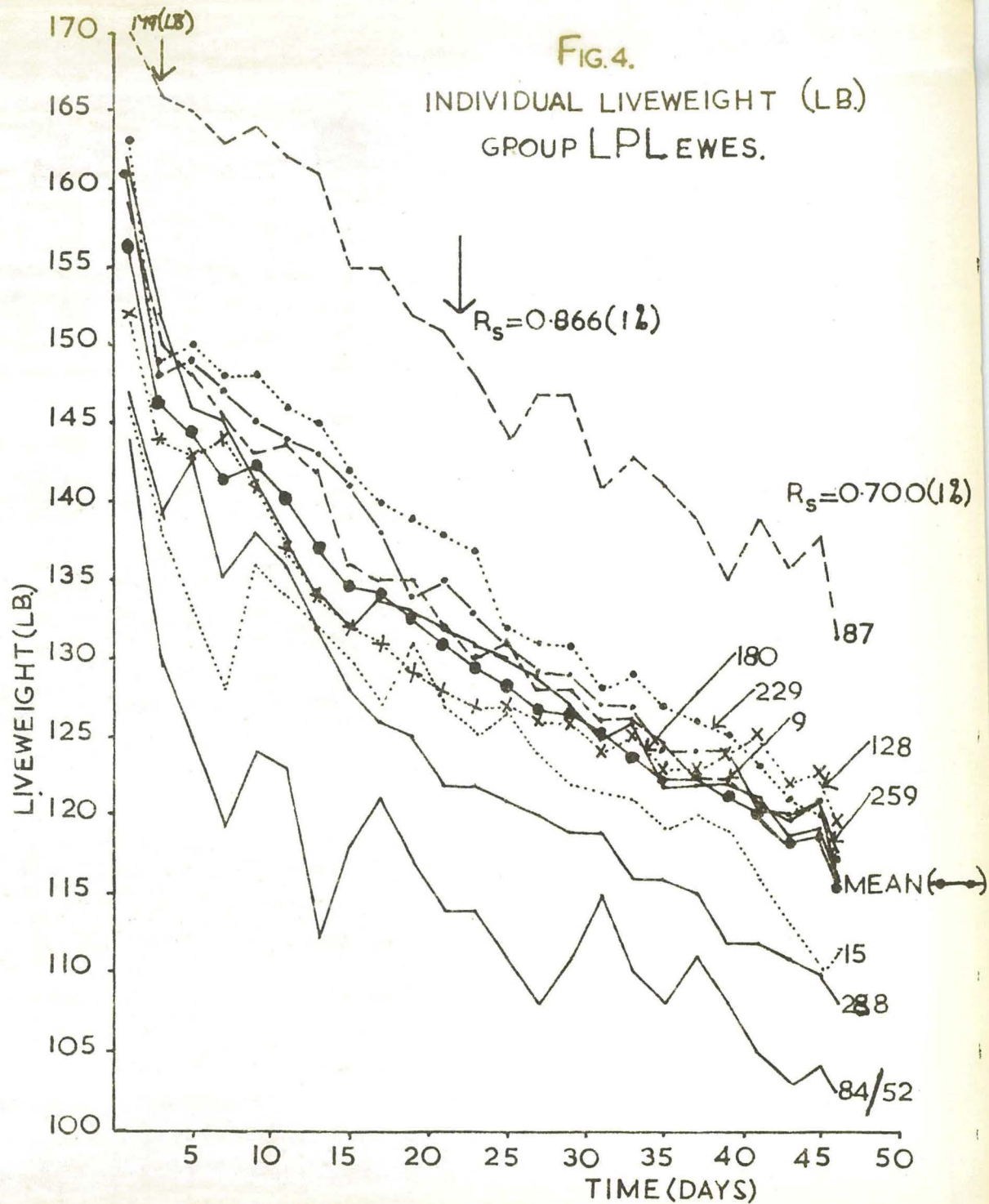
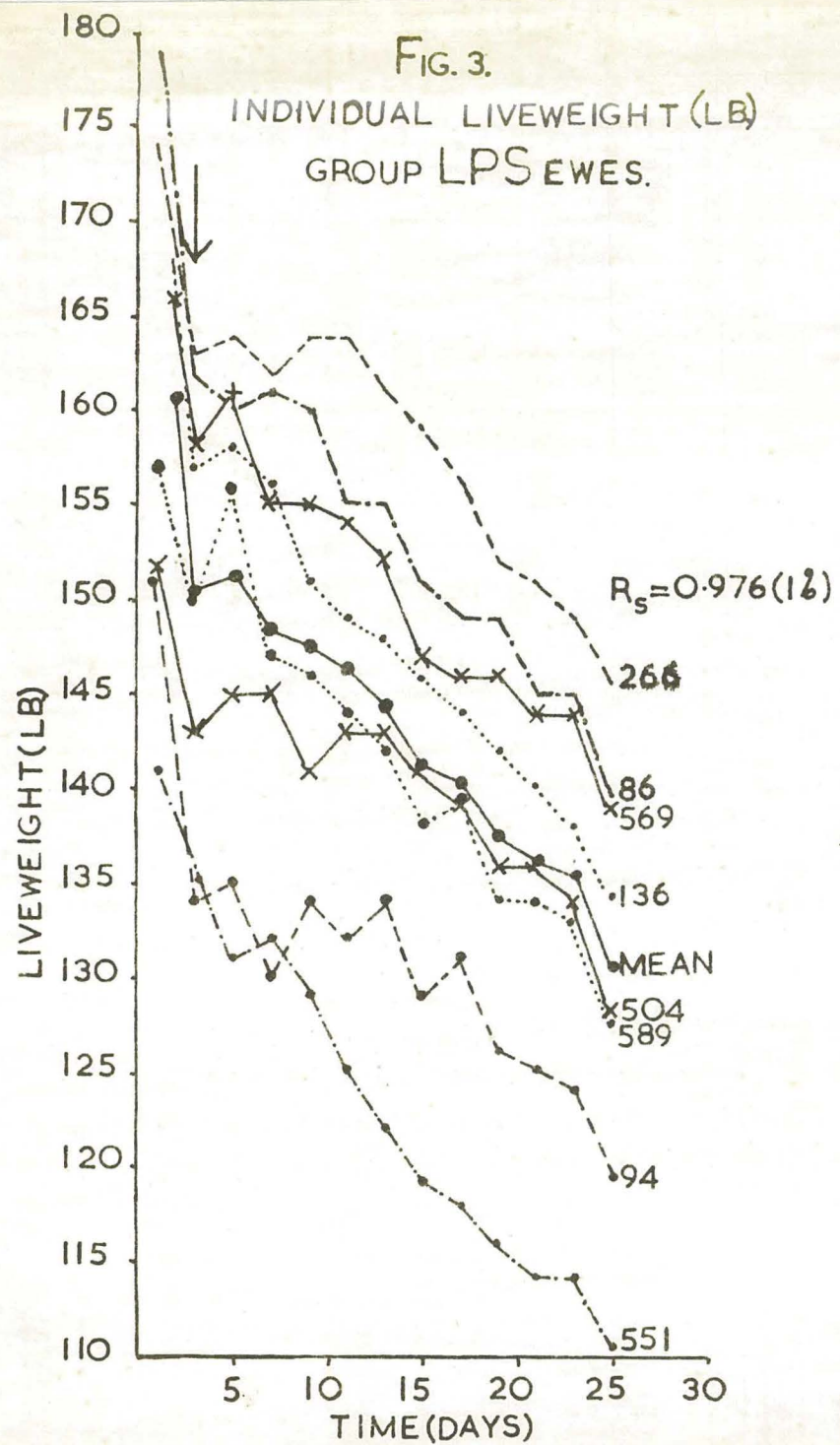


FIG. 2.  
GROUP MEAN LIVELWEIGHT (LB.)  
AS SLAUGHTERED





significantly different in liveweight from the ewes in the LES group. Furthermore the liveweights of the animals in groups C and LES:HP did not differ significantly. The remaining ewes in groups LFL:HPS and LFL:HPL did not differ in liveweight from the LFL group. It should also be emphasized that all the ewes had lost an average of 11.7 lb. during the three days prior to the start of the experiment, this loss being 6.9% of the pre-experimental weight.

The liveweights of each ewe surviving to slaughter are presented in Figs. 3,4,5, and 6. A considerable amount of variation in response to treatments is evident, particularly in groups LES:HP, LFL:HPS, and LFL:HPL on re-alimentation, despite attempt to reduce this variation by controlled feeding. Six sheep in groups LFL:HPS and LFL:HPL actually lost weight on re-feeding after 42 days on a low plane of nutrition, while only three ewes in these groups actually gained in weight. This resulted in a mean weight loss on re-alimentation for the 34-day period. Because of this, and the fact that seven ewes had died from groups LFL:HPS and LFL:HPL the animals in these two groups were excluded from further analysis.

Rank correlations given on the graphs show that there was no substantial alteration in the ranking order of the ewes at the start of the experimental period and at successive stages during the investigation. This indicates that the ewes remained in their liveweight ranking order throughout the investigation irrespective of their original liveweight.

(b) Animal health

The liveweight losses of the ewes that died during the experiment are given in Table 3. The individual liveweights of these ewes are presented in Fig. 7. These ewes in most cases showed a greater reduction in liveweight than comparable healthy animals on the same plane of nutrition. No deaths occurred before 29 days on a low plane of nutrition.

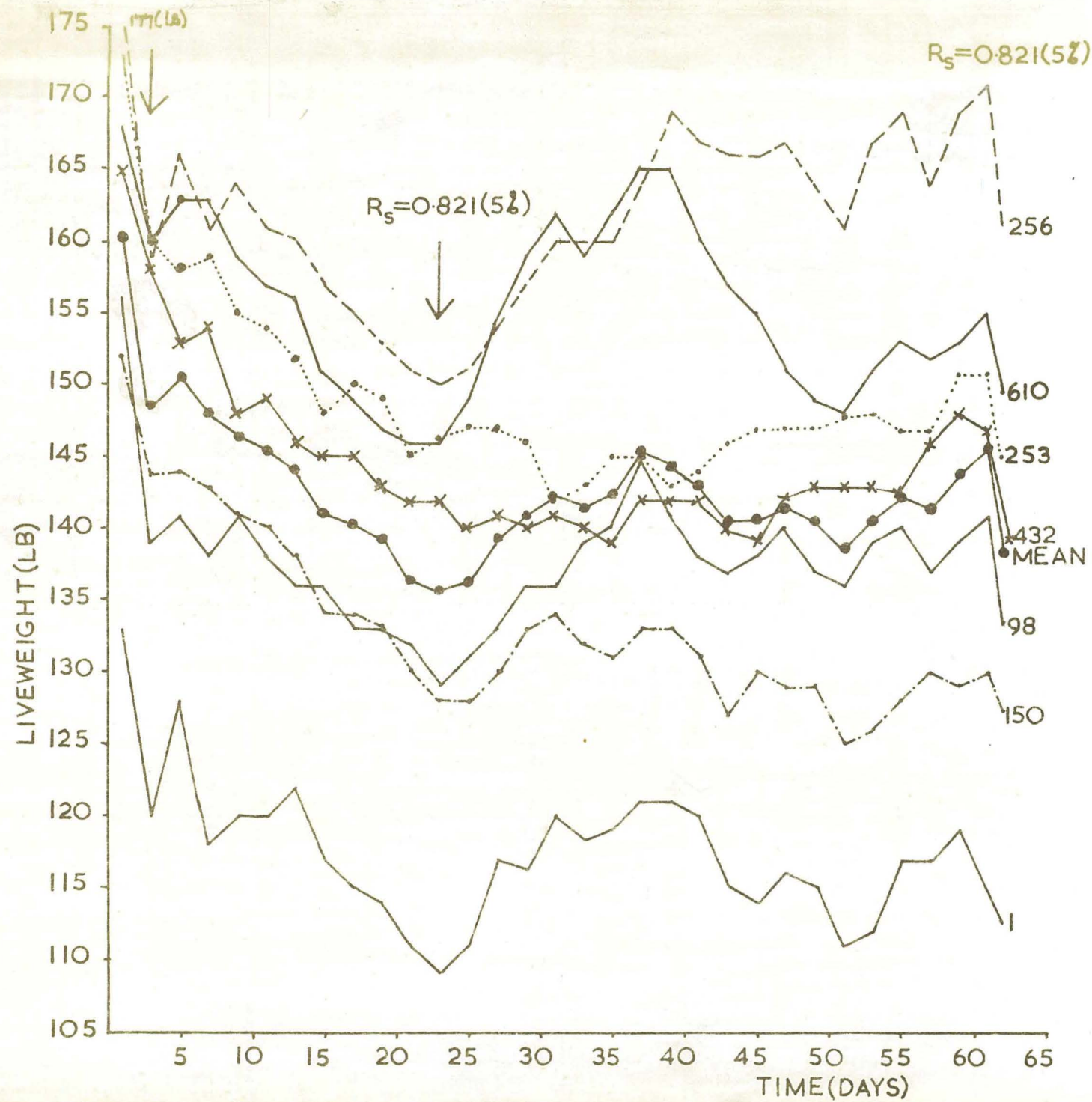


FIG.5.  
 INDIVIDUAL LIVWEIGHT (LB.)  
 GROUP LPS:HP EWES.

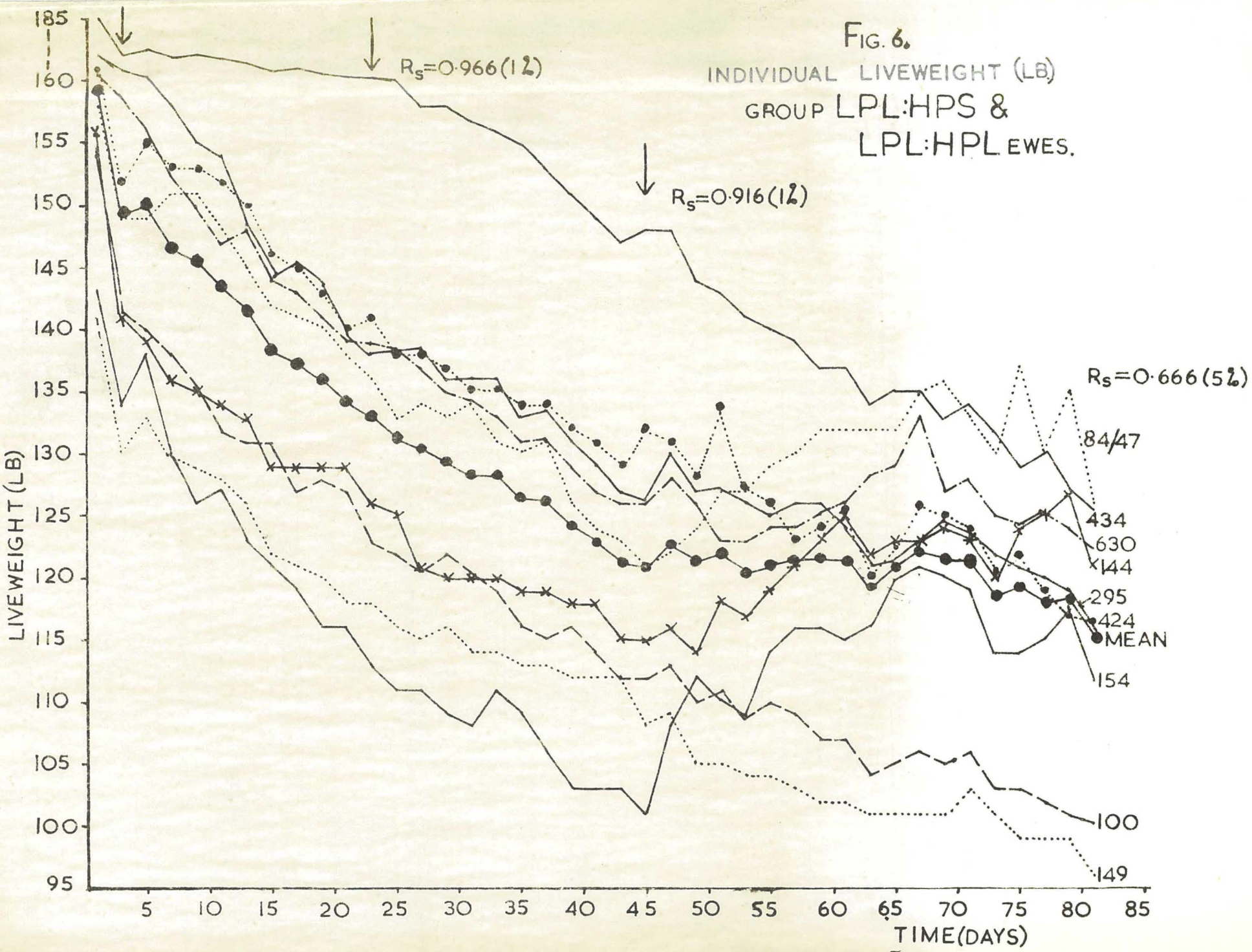


TABLE 3. Liveweight loss of sheep that died during the experiment (lb.)

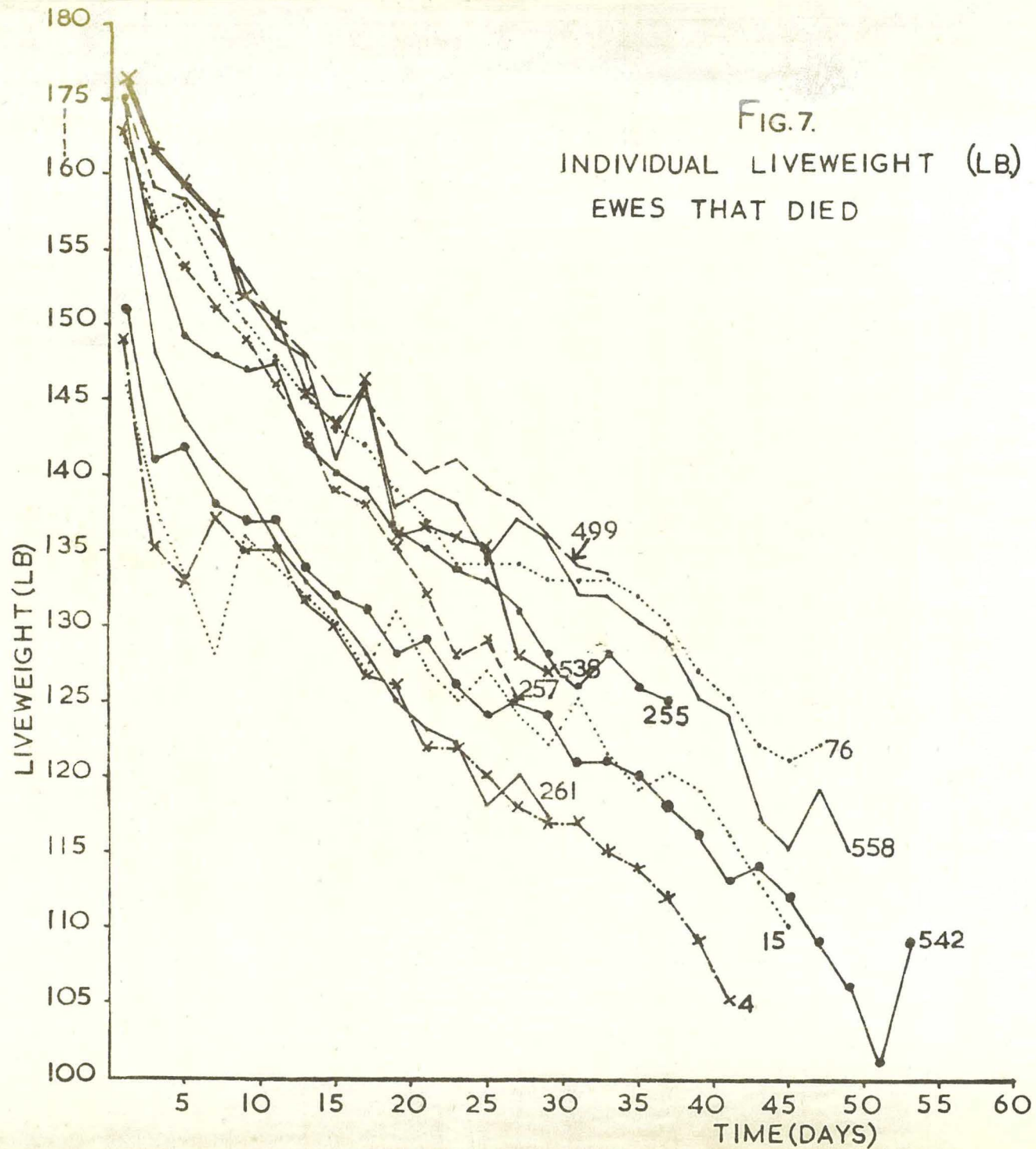
<u>Animal Number (Group)</u>	<u>Liveweight Loss</u>	<u>Percentage Loss</u>	<u>Days *</u>
257 (LPL)	32	20.3	24
538 (LPL:HPS)	37	22.5	26
261 (LPL:HPL)	32	21.6	26
499 (LPL)	26	16.3	30
255 (LPL:HPL)	32	20.3	34
4 (LPL:HPS)	30	22.2	38
76 (LPL:HPS)	35	22.2	44
558 (LPL:HPS)	49	29.8	46
542 (LPS:HP)	33	23.4	50
Mean	34.0	22.0	

\* These days were the time at which the last liveweight was taken although deaths occurred later.

Post-mortem examination of these sheep showed the following pathological changes: The blood clotting power was poor in sheep no's. 261, 499, 4, 558 but normal in 538 and 542. The heart had a normal appearance in no's. 538, 499, 4, 255, 558, 76, and 542. Numbers 261 and 257 showed clots in both chambers, with mobilization of the heart fat evident in no. 257. The heart walls were oedematous in no. 76. The lungs of sheep 499, 255, 4, 257, 542, and 558 had large hydatid cysts. Some calcification was apparent in the fat depots in no. 257 and 261. Number 76 showed adhesions of the pleura. Lesions of necrophorus infection were present in no. 542, a large abscess being observed on the mediastinal lobe of the lungs. Both lungs of no's. 261, 499, 4, 257, 558, and 542 were engorged with blood, suggestive of cardiac incompetence.

Varying degrees of facial eczema damage to the liver were seen in no's. 499, 255, 4, 76, and 542; no. 499 showing evidence of fibrosis of the bile ducts. Hydatid cysts were present in the liver of no's. 538, 255, and 76, and definite paleness of the liver was evident in no's. 499, 255, 538, 4, and 257. The gall bladder was distended in sheep

FIG. 7.  
INDIVIDUAL LIVELWEIGHT (LB)  
EWES THAT DIED



no's. 251, 255, 4, 257, and 76. The kidneys appeared normal in most cases, except no. 255 where white spots in the cortex were observed, while the capsule was adherent in no. 542, although this could have been related to the necrophorus infection of this ewe. The spleen was generally normal except in sheep no's. 499 and 76 where it was grossly enlarged. The pancreas was normal except in 555 where it was engorged with blood, and in 558 there was an extensive associated haemorrhage.

The stomach and intestinal contents were generally very watery, containing little solid food. Haemorrhage of the mucous membranes of the intestines was seen in no's. 499, 255, 4, 76 and 542, and in the stomach of no. 499. Brown scuring was apparent in no's. 538, 499, and 4.

Necrosis of the carcass fats and/or internal fats was evident in no's. 499, 255, 476, and 542. Fat colour was normal except in no's. 76 and 255 where it was icteric. Excess peritoneal fluid was present in no's. 499, and 255. There was no evidence of oedema except in no. 76 where the skeletal musculature was definitely oedematous.

### (c) Discussion

Since the level of nutrition imposed on the sheep was largely dependent on their liveweight changes, it was not unexpected that large reductions in liveweight were evident. In comparison with other experiments on sheep, (Robinson, 1948; Franklin, 1952; Kirton and Barton 1958a), the weight reductions reported here are high although the period of 21 days undernutrition produced a similar reduction in liveweight as was reported by Kirton and Barton (1958a), over a 28-day period by a low plane of nutrition.

Re-alimentation after 21 days of severe undernutrition resulted in a marked initial gain in liveweight, presumably due in part to an increase in weight of the gastrointestinal tract contents. Further high plane feeding did not greatly increase the liveweight, some ewes actually decreasing in bodyweight. A considerable amount of variation is evident. The increase in weight is of the order of 10 lb. during the 38 day re-alimentation period. In marked contrast to this effect, re-alimentation following 42 days of undernutrition was ineffective in increasing liveweight, despite

the fact that a high plane of nutrition was offered for 34 days. Shifts in fluid balance, loss of appetite, a marked reduction in metabolic rate, an unfavourable gastric intestinal tract environment, or a self-imposed starvation are possible ways of explaining this effect. The lower limit of liveweight loss from which recovery was possible may have been attained. This observation does not agree with Franklin (1952), who maintained that sheep which lost most weight, rapidly improved in condition on refeeding. The variable period required after undernutrition for sheep to recover their appetite appears to be related to the depth of undernutrition.

An 18% death loss occurred despite adequate management during the experimental period. The loss of appetite was not in all cases a reliable pre-death symptom. Post-mortem examinations did not reveal any consistent cause of death, although no. 54 was considered to have died from the combined effects of disease and submaintenance feeding, and cardiac incompetence appeared to be the cause of death in no's 257 and possibly 499. It is noted that those sheep which showed pulmonary congestion at death could have died from cardiac incompetence. The importance of disease in the aged Romney ewes, used in this experiment, is emphasized. This could be of considerable practical importance. The liveweight loss of the ewes that died was an average 22.0% (16.3 to 29.8%) of the liveweight at the start of the experiment. Greater reductions in liveweight were observed, however, in some ewes surviving these treatments. One ewe (no. 542) also died after a considerable period of high plane feeding. Consequently the response of aged Romney ewes appears to be due in part to the influence of disease.

Franklin (1952), and Jeffries and Fern (1957), have emphasized that mortality of sheep on a low plane of nutrition was reduced by feeding 'a lot and seldom' rather than 'a little and often', by a reduction in the animals competition for food. Feeding twice daily, as in the present experiment, may therefore have been of considerable importance in contributing to the high death rate. For adequate control of food intake a constant analysis of the liveweight loss of individual sheep is necessary, and it would be preferable to have three sheep only per pen.

(2) Treatment effects on linear measurements on carcass and half-join

In Table 4 group means, standard deviations, and the error mean square (E.M.S.) for some linear carcass measurements are given. The analyses of variance of each of these components is presented in Appendix I.

TABLE 4. Linear carcass measurements (cm.). Group means, standard deviations and error mean squares (E.M.S.).

MEASUREMENT		W.Th.		W.F.		F.		G.	
Group	No.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
C	8	24.2	2.3	24.1	1.9	28.5	1.5	30.4	0.8
LFS	8	23.4	1.5	23.4	2.0	29.2	1.2	29.1	1.2
LFS:HP	7	24.0	2.4	23.5	2.0	28.5	0.9	29.8	0.5
LFL	9	21.1	1.6	20.3	1.2	29.0	2.2	28.8	1.0
E.M.S.		4.01		3.38		2.74		0.91	

No difference due to a short period of undernutrition, or of re-alimentation on the width of thorax (W.Th.) was evident. A prolonged period of undernutrition significantly reduced width of thorax. A short period of undernutrition, or of recovery did not significantly affect width of forequarters (W.F.). Prolonged undernutrition reduced W.F.

There were no significant treatment effects on leg length (F.).

The width of gigots (G.) was reduced by 21 days undernutrition, with no apparent effect of re-alimentation. More prolonged undernutrition appears to have further reduced the width of gigots.

Table 5 presents group means, standard deviations, and the error mean square for measurements taken on the half-join. The results of analyses of variance of these items are given in Appendix I.

TABLE 5. Linear half-loin measurements (mm.). Group means, standard deviations and error mean squares

MEASUREMENT		A		B		C		D		Y		X	
Group	No.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
C	8	61.7	4.5	35.8	3.4	12.1	5.3	11.6	5.4	4.7	1.4	22.0	8.1
LPS	8	57.8	6.1	31.0	3.1	12.1	6.1	10.0	5.0	9.3	3.9	21.7	2.1
LPS:HP	7	58.7	5.5	34.0	5.6	9.0	4.3	8.5	4.6	6.8	3.2	20.1	5.8
LFL	9	56.1	4.5	28.4	1.7	7.6	3.0	7.2	2.8	5.4	2.7	18.8	3.1
E.M.S.		26.89		13.28		32.28		20.50		8.68		27.71	

No significant treatment effects were shown on the cross-sectional length of the longissimus dorsi (l. dorsi), (A), the thickness of backfat at the deepest part of the l. dorsi muscle, (C), the fat over the spinous process (D), or on the thickness of the muscular layer on the lower half of the rib (X). The depth of the l. dorsi muscle was however significantly reduced by 21-days undernutrition, more prolonged undernutrition tending to accentuate this effect. Re-alimentation did not significantly increase measurement B. Statistical analysis showed that 21-days undernutrition significantly increased measurement Y, but the LFL group did not differ from the controls. The recovery group (LPS:HP) did not differ significantly from the other three groups. There does not appear to be any obvious reason for this effect.

### (3) Treatment effects on carcass weight and composition

In Tables 6 and 7 group means, standard deviations and error mean squares for frozen carcass weight and a number of carcass chemical components are presented. The analyses of variance of each of these items is shown in Appendix I.

#### Carcass weight

Analysis of variance showed highly significant treatment effects on carcass weight. A prolonged period of undernutrition resulted in a highly significant reduction in carcass weight, and although the LPS group ewes have a lower carcass weight than Group

TABLE 6. Carcass weight and carcass chemical components (lb.).

Group means, standard deviations, and error mean squares

Group	No.	Carcass weight (lb.)		% Chemical fat		Chemical fat weight (lb.)		Fat-free carcass weight (lb.)		Water as % fat-free carcass	
		Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
C	8	77.4	9.2	44.8	5.9	32.4	7.3	44.8	3.7	72.4	1.2
LFS	8	71.1	9.6	45.1	7.7	32.4	9.1	38.3	3.4	73.7	0.8
LFSHP	7	70.5	4.6	44.0	5.3	29.8	8.0	40.5	3.6	72.6	0.9
LFL	9	60.2	10.4	39.9	5.7	24.1	4.0	35.9	4.2	72.3	0.8
E.M.S.		74.38		39.20		52.80		13.89		3.54	

no statistical differences were shown. No differences were evident between the recovery group and either the LPS group or the control ewes. Further analysis showed that there were significant differences between the LPS and LFL groups, and between the re-alimentated group and the LFL group.

Carcass chemical components:

Percentage and weight of chemical fat (ether extract). No statistically significant treatment effects on the weight or percentage of chemical fat were evident although prolonged undernutrition has reduced the mean weight of chemical fat from 32.4 lb. to 24.1 lb. or by 8.5 lb. This treatment therefore may have been responsible for some fat reduction.

Fat-free carcass weight

A low plane of nutrition for 21 days resulted in a highly significant reduction in the weight of the fat-free body. Prolonged undernutrition however did not further significantly reduce this component. Re-alimentation appears to have resulted in some increase in the weight of the fat-free body since although no significant treatment effects were evident between LPS:HP and LPS ewes, a significant difference is present between the recovery group and the control. This has also resulted in the re-alimentated group being significantly higher than the LFL group.

Water as a percentage of the fat-free carcass weight

No significant effects of undernutrition or re-alimentation were evident on the proportion of water in the fat-free carcass.

Many workers, (Murray, 1922; Behnke, 1942; Behnke, 1953; Pace and Rathbun, 194; Kraybill et al., 1952; Callow, 1947; Babineau and Page, 1955; Ellenberger et al., 195 Reid et al., 1955; Kirton and Barton, 1958; and Garrett et al., 1959), with various species have shown that approximately 72-73% of the fat-free carcass weight is water. The proportion of water is not strictly a constant. Nevertheless the proportion of water in the fat-free body has been regarded by some workers as a 'biological constant' It is therefore of interest to note that the mean value for all experimental ewes was

TABLE 7. Percentage and weight (lb.) of carcass water and protein.  
 Group means, standard deviations, and error mean squares.

Group	No.	% Carcass protein		Weight carcass protein		% Carcass water		Weight carcass water	
		Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
C	8	12.4	1.4	9.4	0.6	42.4	3.5	32.4	3.1
LFS	8	10.6	1.2	7.4	0.4	40.2	4.9	28.2	2.8
LFSHP	7	11.9	1.4	8.4	0.8	44.6	4.8	29.4	4.1
LFL	9	11.6	1.1	7.0	0.7	43.0	4.0	26.0	0.7
E.M.S.		1.72		0.50		19.11		8.34	

72.8% (range 70.9-74.7%) of the weight of the fat-free carcass. Since these figures are estimated on the frozen carcasses, they are likely to underestimate the percentage of water in the fat-free carcass at slaughter due to dehydration during freezer storage.

#### Percentage and weight of carcass water

As with percentage chemical fat and protein no significant treatment effects were apparent on the percentage of carcass water. A short period of undernutrition has resulted in a highly significant reduction in carcass water weight. More prolonged undernutrition did not further significantly reduce the weight of carcass water. Re-alimentation did not produce a significant increase in the weight of water when the recovery group was compared with the LPS group, but since the LPS and C groups are not significantly different and there is a highly significant difference between LPS and LPL ewes, some hydration appears to have occurred on re-alimentation. The largest difference in the mean weight of carcass water between the heaviest (C) and lightest (LPL) groups was 6.4 lb., which accounts for some of the reduction recorded in the fat-free carcass weight. In contrast to the effect of undernutrition on the weight of carcass water no effect was noted on the proportion of water in the fat-free body. A highly significant reduction in carcass protein weight was produced by undernutrition. Apparently undernutrition produced a loss of both protein and water in a constant ratio.

#### Percentage and weight of carcass protein

No treatment effects were apparent on the percentage of carcass protein. Both short and prolonged periods of undernutrition caused a highly significant reduction in the weight of carcass protein, although LPL ewes did not differ significantly from the LPS ewes. Re-alimentation resulted in a significant increase in the weight of carcass protein, although the amount of carcass protein in the recovery group was still highly significantly lower than in the control ewes. This increase in protein weight resulted in highly significant differences between the LPS:HP and LPL groups. Prolonged undernutrition has resulted in a reduction of the mean weight of carcass protein of the order of 2.4 lb., which explains in part the weight loss in the fat-free carcass.

#### (4) Treatment effects on the dissectible components of the leg

The group means, standard deviations, and the error mean squares for leg weight, and a number of leg components are presented in Table 8. The analyses of variance of these items are shown in Appendix I.

##### Leg weight

Analysis of variance revealed a highly significant plane of nutrition effect on the undissected leg weight. A highly significant reduction in the total weight of the leg was produced by 21 days undernutrition, while prolonged undernutrition did not appear to further reduce the total leg weight, although the LPL group mean leg weight was considerably lower (250.1g.) than the LPS ewes. Re-alimentation did not increase the total leg weight and significant differences between the LPS:HP and C groups were evident. No significant difference was found between the LPL and C group although the difference in mean weight was quite marked.

##### Dissectible muscle of leg

The major component of leg weight is muscular tissue, and since both carcass protein and water were significantly affected by plane of nutrition it was not unexpected that undernutrition produced significant differences at the 1% level of probability compared with the control group. Prolonged undernutrition did not further reduce the weight of dissectible muscle of the leg. Re-alimentation did not significantly increase the weight of dissectible muscle and consequently highly significant differences were present between the re-alimentated group (LPS:HP) and the controls. No difference was evident between the LPS:HP group and the prolonged undernutrition group (LPL).

##### Total dissectible fat of leg

No treatment effects were produced on the weight of dissectible fatty tissue. The mean weight of this tissue was appreciably lower in the LPL group than in any other group, and this difference approached significance.

TABLE 8. Weight of the leg and some of its dissectible components (g.).  
Group means, standard deviations, and error mean squares

Group	No.	Leg Weight		Total Dissectible Muscle		Total Dissectible Fat		Total Dissectible Bone		Subcutaneous Fat of Leg		Intermuscular Fat of Leg		Mammary Gland	
		Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
C	8	3638.3	407.2	2320.5	249.7	879.2	160.2	344.1	52.6	424.8	99.4	319.1	36.9	135.2	39.9
LRS	8	3106.6	389.8	1766.2	213.5	946.2	174.2	325.6	32.2	420.8	172.9	377.5	54.1	135.3	14.8
LRS:NP	7	3197.7	318.4	1833.7	182.4	936.7	212.8	313.0	22.8	443.5	127.4	390.0	62.8	131.1	34.9
LPL	9	2856.5	288.5	1703.3	254.8	749.2	82.7	325.5	36.1	324.7	62.4	325.2	45.5	99.2	20.1
E.M.S.		124992.8		52685.4		25675.7		1437.5		14549.7		2870.7		831.2	

#### Dissectible bone of leg

The weight of this tissue does not appear to be sensitive to either prolonged undernutrition or re-alimentation, since no treatment effects on the total weight of dissectible bone of the leg were apparent.

#### Dissectible subcutaneous fat of leg

No significant treatment effects were produced on the weight of the subcutaneous fat of the leg, but it should be noted that the mean weight of this tissue for the LPL group was considerably lower (100.1 g.) than the controls. High variability is present.

#### Dissectible intermuscular fat of leg

Analysis of variance showed that treatment effects were present, but the results do not conform with those expected. A short period of undernutrition produced a highly significant increase in the weight of this tissue, while prolonged undernutrition has further reduced the weight. This resulted in no significant difference being evident between the LPL group and the controls. Recovery resulted in no significant differences being apparent compared with the LPS group, while the LPS:HF ewes were significantly higher than group C. This result appears to be due to the dissection technique.

#### Dissectible mammary gland

Analysis of variance showed a significant plane of nutrition effect on the dissected mammary gland. No effects were evident due to short periods of undernutrition or re-alimentation on this tissue. Prolonged undernutrition however resulted in a significant reduction in the weight of the mammary gland, mainly due to a nutritional effect after 24 days of treatment.

#### (5) Treatment effects on the half-loin and its tissues

Table 9 gives the group means, standard deviations, and error mean squares for the weight of the undissected half-loin, and its dissectible components. Analyses of variance of these data are given in Appendix II.

TABLE 9. Weight of the half-loin and some dissectible components (g.). \*  
Group means, standard deviations, and error mean squares

Group	Half-loin weight		Total dissectible muscle		General muscle		Face muscles		Skin muscle		
	No.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
C	8	2891.5	487.6	1078.5	116.1	453.1	46.6	144.5	20.1	53.5	10.2
IFS	8	2906.8	699.4	853.5	96.0	355.6	48.7	115.0	11.3	54.8	7.5
IFS:HP	7	2652.4	584.2	891.1	143.6	369.0	67.7	120.5	14.5	55.5	11.4
LPZ	9	2199.5	257.1	735.6	93.6	295.3	39.4	113.1	15.4	48.8	14.8
E.M.S.		273805.1		12606.6		2565.6		216.8		131.4	

\* Details pertaining to the longissimus dorsi muscle are given in Table 13.

N.B. Total dissectible muscle includes the longissimus dorsi.

### Half-loin weight

Undernutrition produced significant differences in the undissected weight of the half-loin. Prolonged undernutrition resulted in significant differences being apparent between the LFL group and the control (C) group. A short period of undernutrition or re-alimentation did not have any effect on the total weight of this joint. Considerable variability is evident, and it should be noted that fat was a high proportion of the total weight of this joint.

### Total dissectible muscle of half-loin

A highly significant reduction in the weight of this tissue due to 21 days submaintenance feeding, and a significant reduction on prolonged undernutrition was evident. This resulted in highly significant differences being apparent between the LFL group and the controls. This further emphasizes that muscular tissue is sensitive to undernutrition. Re-alimentation did not increase the total dissectible muscle of the half-loin. Highly significant differences between the LFS:HP and C groups were evident.

### General muscle of half-loin

The general muscle of the half-loin includes all dissected muscle of this joint except the dissectible skin and psaos muscles. Undernutrition produced a highly significant reduction in the weight of this tissue and this difference was significant at the 1% level of probability for 21 days undernutrition. Prolonged undernutrition resulted in a further significant reduction in weight. Re-alimentation did not significantly increase the weight of the general muscle of the half-loin, and highly significant differences between the LFS:HP and the control ewes were shown.

### Weight of dissectible skin muscle of half-loin

No treatment effects were shown on the weight of skin muscle. Considerable variation in the data is evident.

### Dissectible psaos muscles of half-loin

A highly significant reduction in the weight of the psaos muscles due to a short period of submaintenance feeding was apparent. Prolonged undernutrition did not

TABLE 10. Weight of dissectible fat components of the half-join (g.).  
Group means, standard deviations, and error mean squares

Group	No.	Total dissectible fat weight		Subcutaneous fat		Inter-muscular fat		Peritoneal fat	
		Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
C	8	1582.6	451.8	794.5	252.3	264.8	99.4	523.2	200.8
LFS	8	1840.3	670.1	786.5	434.6	385.7	80.3	668.1	202.1
LFS:HF	7	4607.8	484.0	704.4	247.9	391.4	119.2	512.0	166.5
LFL	9	1312.4	237.8	500.7	151.4	297.3	48.6	514.3	129.0
E.M.S.		229692.4		82884.7		7808.6		31004.7	

appear to further reduce the weight of these muscles, although highly significant differences were evident between C and IPL groups. Re-alimentation did not significantly increase the weight of these two muscles. The difference between the recovery group and the control ewes was reduced to a 5% level of significance. No significant differences were apparent between other treatment groups.

#### Total dissectible fat and its components of the half-loin

Table 10 gives the group means, standard deviations, and error mean squares for the weight of total dissectible fat, subcutaneous and intermuscular fats, and the perirenal fat. Analyses of variance of these data are given in Appendix II. Analysis of variance showed no statistically significant treatment effects on the total dissectible fat, or subcutaneous fat weight. The components of the total dissectible fat of this joint were analysed since the total weight of a tissue may not reveal changes in its components. It is interesting to note that significant treatment effects were shown on the intermuscular fat of the half-loin. A significant increase in the weight of this tissue followed a short period of undernutrition, but there was a significant reduction on prolonged undernutrition. This resulted in no differences being evident between the IPL group and the control ewes. Re-alimentation after 21 days undernutrition did not have any significant effect. As with the leg intermuscular fat care must be exercised in interpreting these results as the dissection of this fat does show considerable variability.

#### Treatment effects on the weight of perirenal fat

No significant treatment effects were apparent on the weight of this depot.

The considerable amount of variability in the weight of fat in the half-loin, as in other regions of the carcass, emphasizes that large differences are required for statistical significance to be shown.

#### (6) Treatment effects on the 9-10-11 rib-cut and its physical components

The group means, standard deviations, and error mean squares are presented in Tables 11 and 12. The results of analyses of variance pertaining to these data are given in Appendix II.

TABLE 11. Weight of the 9-10-11 rib-cut and its dissectible muscle components (g.).

Group means, standard deviations, and error mean squares

		<u>Rib-cut weight</u>		<u>Total dissectible muscle</u>		<u>Dissected skin muscle</u>		<u>Dissected general muscle</u>	
<u>Group</u>	<u>No.</u>	<u>Mean</u>	<u>S.D.</u>	<u>Mean</u>	<u>S.D.</u>	<u>Mean</u>	<u>S.D.</u>	<u>Mean</u>	<u>S.D.</u>
C	8	1401.8	276.8	460.8	53.4	33.8	4.4	427.0	49.8
LPS	8	1266.6	302.6	345.1	39.0	30.7	5.2	314.3	35.4
LPS:HP	7	1256.8	256.5	350.0	32.7	32.0	4.8	318.0	29.0
LPL	9	1000.7	101.7	309.4	42.2	25.6	4.6	283.7	40.0
E.M.S.		59122.1		1834.7		23.25		1575.3	

9-10-11 rib-cut weight

No statistically significant treatment effects were apparent due to a short period of undernutrition or re-alimentation, when compared with the controls. Undernutrition however did result in a considerable reduction in the weight of this joint. Severe submaintenance has resulted in a highly significant reduction in the weight of this joint when the LPL group is compared with group C, and significant differences between the LPL ewes and both LPS and LPS:HP ewes. There is a considerable amount of variability present in this region, apparently due to the high fat content.

Total dissectible muscle of 9-10-11 rib-cut

A short period of undernutrition reduced the weight of muscle in both sub-maintenance groups, highly significant differences being present between the LPS and LPL groups and the controls. No significant reduction between LPS and LPL ewes was demonstrated. Re-alimentation did not significantly increase the weight of this tissue. Highly significant differences were present between the recovery group and group C.

Dissectible skin muscle of 9-10-11 rib-cut

There was an absence of significant treatment effects due to a short period of undernutrition, or recovery compared with group C. Further undernutrition resulted in significant differences between the LPL and LPS groups, with highly significant

differences between the extreme low plane group (LPL) and the controls (C).

Dissectible general muscle of 9-10-11 rib-cut

A short period of undernutrition resulted in a highly significant reduction of this tissue. Prolonged undernutrition did not significantly decrease this reduction, although a considerable amount of tissue appears to have been lost as a result of this severe treatment. Re-alimentation did not increase the weight of the general muscle of the 9-10-11 rib-cut, highly significant differences being present between the LFS:HP and C groups. There was no difference due to treatment between the recovery group (LFS:HP), and the prolonged low plane group (LPL).

Total and components of dissectible fat of 9-10-11 rib-cut

No significant treatment effects were evident on the total dissectible fat of rib-cut. Analysis of variance of both the subcutaneous and intermuscular fat of the 9-10-11 rib-cut showed that there were no differences attributable to treatment effects.

TABLE 12. Total dissectible fat and its two components of the 9-10-11 rib-cut (g.)  
Group means, standard deviations, and error mean squares

<u>Group</u>	<u>No.</u>	<u>Total dissectible fat</u>		<u>Subcutaneous fat</u>		<u>Intermuscular fat</u>	
		<u>Mean</u>	<u>S.D.</u>	<u>Mean</u>	<u>S.D.</u>	<u>Mean</u>	<u>S.D.</u>
C	8	793.3	267.3	516.0	221.3	277.3	65.7
LFS	8	796.7	313.6	520.6	260.9	276.1	63.9
LFS:HP	7	778.7	251.4	475.1	183.6	303.5	72.6
LPL	9	576.4	123.6	354.4	120.8	222.0	24.5
E.M.S.		60373.3		40671.3		3407.1	

(7) Changes in chemical composition

Results are presented in terms of the absolute weight of chemical components, and also as percentages. This should clarify treatment effects.

TABLE 13. Weight of the dissectible longissimus dorsi muscle (g.) from the half-join, and some chemical percentage and weight components. Group means, standard deviations, and error mean squares.

Group	No.	Wt. dissected L. dorsi muscle of half-join		Chemical Fat				Water				Protein					
		Mean	S.D.	Percentage	Weight	Mean	S.D.	Percentage	Weight	Mean	S.D.	Percentage	Weight	Mean	S.D.	Percentage	Weight
C	8	430.4	74.3	9.1	2.7	39.2	13.5	68.1	6.1	293.4	59.7	19.7	0.5	84.8	14.5		
IRS	8	328.0	49.0	7.2	1.1	23.6	5.1	72.7	2.0	238.8	37.4	18.9	0.6	62.2	9.2		
IFS:HP	7	346.0	74.1	8.9	1.6	31.0	9.0	72.0	1.9	248.6	51.9	18.1	0.9	63.0	14.3		
IFL	9	278.6	40.0	5.7	1.5	15.8	3.5	74.2	1.5	206.8	30.2	19.2	0.9	53.7	9.2		
E.M.S.		3620.7		3.42		73.64		12.11		2081.3		0.62		143.39			

(a) Treatment effects on the longissimus dorsi muscle from the half-loin (g.) and some chemical percentage and weight components.

Group means, standard deviations, and error mean squares are presented in Table . Appropriate analyses of variance for these data are given in Appendix III.

Weight of the l. dorsi muscle of half-loin

Both periods of undernutrition have produced highly significant reductions in the weight of this tissue compared with the controls. The LPL group does not differ significantly from the LFS group. Re-alimentation does not appear to have significantly increased the weight of this tissue, and the LFS:HP group is significantly lower than the controls. Some increase however appears to have occurred as the LFS:HP group differs significantly from the severe undernutrition group.

Weight and percentages of chemical components of l. dorsi from half-loin

Highly significant reductions in the weight of chemical fat are apparent between the LFS group and the control, and the LPL group and control. Most of this reduction has occurred during the first period of undernutrition; there being no difference between the two undernutrition groups. Although no differences were demonstrated between the LFS:HP and LFS groups, some increase in the weight of chemical fat appears to have resulted from re-alimentation as no differences exist between the recovery group and the control, while highly significant differences occur between the recovery group and the prolonged level of undernutrition (LPL). The ranking order and significance levels for fat percentages are the same as for fat weight except that no significant differences were produced by a short period of undernutrition. This effect appears to be mainly due to the increase in the percentage of water with a short period of undernutrition.

The ranking order of the means for the weight of water and of protein are the same as for the weight of chemical fat.

Significant and highly significant reductions in the weight of water due to short and prolonged periods of undernutrition were shown. No differences were present

TABLE 14. Some chemical percentage and weight components (g.) of the muscle of the leg.  
Group means, standard deviations, and error mean squares

Group No.	Chemical Fat			Protein			Water				
	Percentage	Mean	S.D.	Weight	Mean	S.D.	Weight	Mean	S.D.	Weight	
C	8.4	195.4	48.7	19.0	0.7	440.6	45.1	71.7	1.5	1666.0	179.5
LFS	8.3	146.1	29.4	17.6	0.4	311.2	35.8	73.3	1.6	1295.9	165.1
LFS:NP	7.1	133.8	47.6	18.4	0.7	330.2	40.9	73.4	2.4	1343.1	100.2
LPL	6.8	113.0	17.0	18.4	0.9	315.6	59.7	72.9	3.8	1242.9	199.1
E.M.S.	2.96	1380.2		0.577		2211.3		6.751		28613.4	

between other treatment groups. On a percentage basis the ranking order is reversed compared with the weight of water. Similar significance levels are present, except that on a percentage basis, the recovery group is significantly higher than the control.

As with the weight of water, a short period of undernutrition produced a significant reduction in the weight of protein. No differences were evident between the IPL and IFS groups. Re-alimentation did not significantly increase the weight of protein. On a percentage basis however, undernutrition had no effect on the percentage of protein. The recovery group (IFS:HP) is highly significantly lower than the control, and significantly lower than the undernutrition group.

(b) Weight and percentage chemical components of the muscle of the leg

Group means, standard deviations, and error mean squares are given in Table 14. Analyses of variance for these data are presented in Appendix III.

Except for a highly significant reduction in the percentage of protein due to a short period of undernutrition, there is no evidence that either undernutrition or re-alimentation resulted in a change in the percentage chemical components in the leg muscle.

It will be recalled that highly significant treatment effects were demonstrated on the dissectible muscle of the leg.

Undernutrition reduced the weight of muscle fat, a significant reduction being produced by a short period of submaintenance feeding. Muscle fat is the weight of chemical fat (ether extract) within the muscle. No further reduction in the weight of muscle fat occurred with prolonged undernutrition. Re-alimentation did not increase this fat.

Treatment effects on the weight of water are similar to those for chemical fat and protein weight. For both the weight of protein and water, highly significant reductions were evident due to short periods of undernutrition, with no change being observed with either re-alimentation or prolonged undernutrition.

**TABII 15.** Some chemical percentage and weight components (g.) of the muscle of the half-lobin.  
 Group means, standard deviations, and error mean squares

Group	No.	Chemical fat				Protein				Water			
		Percentage		Weight		Percentage		Weight		Percentage		Weight	
		Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
C	8	13.3	1.1	144.1	21.9	18.2	0.5	197.0	20.2	67.2	0.9	724.8	74.6
LFS	8	13.1	2.9	112.9	33.1	17.9	0.7	152.6	12.0	68.0	2.8	580.3	66.3
LFS:HP	7	11.4	2.3	103.5	32.4	18.0	0.4	160.8	25.0	69.7	1.9	620.0	89.6
LFL	9	11.6	1.9	84.6	12.3	18.1	0.7	133.5	19.1	69.4	1.7	511.7	72.3
E.M.S.		4.69		665.31		0.45		379.13		4.03		5711.1	

Therefore a short period of undernutrition has resulted in a significant reduction in the dissectible muscle of the leg, this weight reduction being due to a loss of chemical fat, protein, and water. Re-alimentation or prolonged undernutrition do not appear to have substantially altered the effect produced by a short period of undernutrition.

(c) Weight and percentage chemical components of the muscle of the half-loin

Group means, standard deviations, and error mean squares are presented in Table 4 and the analyses of variance data are given in Appendix III.

Analyses of variance showed that there were no significant changes in the percentage of chemical fat, water, or protein in the muscle of the half-loin.

The weight of chemical fat was significantly reduced by 24 days undernutrition, and further reduced by prolonged undernutrition. This resulted in highly significant differences being demonstrated between the LPL group and the control. Re-alimentation did not increase the weight of chemical fat in the muscle of the half-loin, a highly significant difference being apparent between the recovery group and the control. No statistical difference is evident between the re-alimentated group and the prolonged undernutrition group.

Treatment effects had the same significant results on both the weight of protein and water components. For these two constituents a short period of undernutrition resulted in highly significant reductions in weight. A further period of undernutrition did not significantly change the amount of this reduction, although for both water and protein, the means of the LPL group are lower than in any other treatment group. For the weight of both protein and water, highly significant differences are evident between the LPS:HP group and the control. The recovery group had a significantly higher amount of both protein and water than the LPL ewes.

Thus undernutrition appears to reduce the weight of the muscle in the half-loin, this weight reduction being due to a decrease in the absolute amount of chemical fat, protein, and water, with no observed change in the percentage of these chemical components.

TABLE 16. Some chemical percentage and weight components (g.) of the muscle of the 9-10-11 rib-cut.

Group means, standard deviations, and error mean squares

Group	No.	Chemical fat				Protein				Water			
		Percentage		Weight		Percentage		Weight		Percentage		Weight	
		Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
C	8	18.3	2.1	84.9	16.7	17.1	0.4	78.9	9.4	63.6	2.0	292.9	30.8
IES	8	18.2	3.4	62.3	11.4	16.6	0.8	57.5	7.4	64.4	2.9	222.7	30.8
IES:HP	7	14.3	2.9	50.1	11.0	17.3	1.3	60.9	7.6	67.5	1.9	236.1	22.5
IFL	9	14.9	2.8	45.6	6.9	17.4	0.5	53.9	7.9	66.8	2.8	207.3	33.3
R.M.S.		8.16		142.58		0.73		66.92		6.44		902.03	

(d) Weight and percentage chemical components of the 9-10-11 rib-cut muscle

Table 16 sets out the appropriate group means, standard deviations, and error mean squares, while analyses of variance results pertaining to these data are given in Appendix IV.

It will be recalled that highly significant treatment effects were demonstrated on the total dissectible muscle of the 9-10-11 rib-cut.

Undernutrition for a short period has resulted in highly significant reductions in the weight of chemical fat, protein, and water in the muscle of the rib-cut. Although prolonged undernutrition decreased the mean weight of protein and water, this reduction does not attain significance. Prolonged undernutrition did however significantly reduce the amount of muscle fat. Re-alimentation did not significantly increase the absolute amount of chemical fat, protein, or water, highly significant differences being evident for these three constituents between the recovery group and the control. No significant differences were demonstrated for these three constituents between the re-alimentated group and the LPL group.

In contrast to the treatment effect on the muscle of the half-loin, significant results were evident on the percentage fat, and water of the muscle of the rib-cut, while no significant effects were produced on the percentage of protein. A short period of undernutrition did not reduce the percentage of muscle fat. More prolonged submaintenance feeding however resulted in a further reduction which was significant. It is interesting to note that re-alimentation significantly reduced the percentage of fat in the muscle, so that the recovery group was significantly lower than the control, while no difference was evident between the LPS:HP and LPL groups. Prolonged undernutrition resulted in a significant increase in the percentage of water. Re-alimentation after 21 days undernutrition also increased the percentage of water, so that the recovery group had a higher percentage of water than the control group. The percentage of water was not different between the recovery group and the prolonged undernutrition group.



TABLE 18. Percentage and weight (g.) of water in the subcutaneous fat of the leg, half-loin, and 2-10-11 rib-cut.

Group means, standard deviations, and error mean squares

Group	No.	Subcutaneous fat of leg			Subcutaneous fat of half-loin			Subcutaneous fat of rib-cut					
		Percentage	Mean	S.D.	Percentage	Mean	S.D.	Percentage	Mean	S.D.			
C	8	16.7	5.0	76.8	16.6	9.3	1.9	72.6	26.0	11.1	3.1	53.9	19.4
IPS	8	17.7	6.2	70.4	18.7	11.6	3.4	95.3	84.6	10.4	2.1	50.8	16.5
IPS:HF	7	17.4	3.5	69.7	16.7	10.9	2.8	71.7	15.3	11.4	1.8	51.6	13.5
IFL	9	20.3	6.7	63.1	13.2	10.9	3.3	51.0	5.7	11.3	2.3	38.5	9.4
E.M.S.		31.93		267.14		8.86		2021.9		6.00		227.69	

(e) Weight and percentage chemical components of the dissected subcutaneous fat of the leg, half-loin, and rib-cut

Analyses of variance of these items are presented in Appendix III and IV. The percentage and weight of chemical fat are given in Table 17.

No treatment effects were evident on either the percentage or weight of chemical fat in the subcutaneous fat of the leg, half-loin, or rib-cut. In all these tissues substantial reductions in the weight of chemical fat appear to have occurred.

Percentage and weight of water data for the subcutaneous fat of the leg, half-loin, and rib-cut, are presented in Table 18. There is no evidence that either undernutrition or re-alimentation have affected the hydration of these tissues. This was in contrast to the dehydration noted in the muscular tissue.

The percentage and weight of protein of the subcutaneous fat in the leg, half-loin and rib-cut are given in Table 19. No treatment effects were demonstrated on either the percentage or weight of protein in the subcutaneous fat of the half-loin, or on the weight of protein in the subcutaneous fat of the leg. A short period of undernutrition, or re-alimentation did not affect the percentage of protein in the subcutaneous fat of the leg. Prolonged undernutrition resulted in a significant increase in the percentage of protein, although there is no difference between the IPL group and the control. The percentage and weight of protein in the subcutaneous fat of the rib-cut was significantly reduced by a short period of undernutrition, and significantly increased by prolonged undernutrition, although no difference exists between the IPL group and the control. There has also been a significant increase in the weight of protein in the subcutaneous fat of the rib-cut compared with the LIS group. There does not appear to be any obvious explanation for these effects, but it seems likely to be due to the method of chemical analysis used, and to varying amounts of muscular tissue dissected off with the subcutaneous fat.

(f) Percentage and weight (g.) of chemical components of the dissected intermuscular fat of the leg, half-loin, and 9-10-11 rib-cut

The analyses of variance of these items are presented in Appendix V. The group means, standard deviations, and error mean squares of the percentage and weight of

TABLE 12. Percentage and weight (g.) of protein in the subcutaneous fat of the leg, half-join, and 9-10-11 rib-cut.

Group means, standard deviations, and error mean squares

Group	No.	Subcutaneous fat of leg			Subcutaneous fat of half-join			Subcutaneous fat of rib-cut					
		Percentage	Mean	S.D.	Percentage	Mean	S.D.	Percentage	Mean	S.D.			
C	8	4.9	1.1	20.8	6.4	2.1	0.6	16.5	4.7	2.5	0.9	11.7	3.7
LFS	8	3.8	1.7	14.6	4.9	2.1	0.8	16.8	15.3	1.4	0.8	5.8	2.3
LFS:HP	7	4.2	1.5	16.6	5.2	2.2	1.2	13.6	2.7	2.1	0.6	9.1	1.8
LPL	9	5.9	1.5	18.9	4.2	3.2	0.9	15.1	2.4	3.0	0.5	10.6	2.9
E.M.S.		2.42		27.64		0.86		67.50		0.59		8.03	

TABLE 20. Percentage and weight (g.) of chemical fat in the intermuscular fat of the leg, half-loin, and rib-cut.

Group means, standard deviations, and error mean squares

Group	No.	<u>Intermuscular fat of leg</u>			<u>Intermuscular fat of half-loin</u>			<u>Intermuscular fat of rib-cut</u>					
		<u>Percentage</u>		<u>Weight</u>	<u>Percentage</u>		<u>Weight</u>	<u>Percentage</u>		<u>Weight</u>			
		<u>Mean</u>	<u>S.D.</u>	<u>Mean</u>	<u>S.D.</u>	<u>Mean</u>	<u>S.D.</u>	<u>Mean</u>	<u>S.D.</u>	<u>Mean</u>	<u>S.D.</u>		
C	8	65.9	8.2	209.8	40.1	75.6	3.9	201.3	76.8	75.7	3.5	209.4	46.4
LES	8	51.9	6.9	196.4	40.5	66.8	5.0	259.5	68.7	73.8	6.2	204.7	53.8
LESIMP	7	50.8	2.6	199.6	44.4	62.8	3.3	248.8	87.6	69.2	4.6	210.9	57.5
IFL	9	52.1	4.6	168.1	15.7	60.7	10.3	179.8	37.8	68.5	7.1	152.0	20.8
E.M.S.		36.89		1228.5		43.69		4714.6		32.11		2100.5	



chemical fat and water in these fat depots is given in Tables 20 and 24.

Undernutrition did not affect the weight of chemical fat in the intermuscular fat of the leg, or the half-loin. Treatment effects were demonstrated on the percentage of chemical fat in the intermuscular fat of the leg, and loin. For both these joints a short period of undernutrition lowered the percentage of chemical fat, while further submaintenance feeding, although resulting in a reduction in mean weight and producing a highly significant difference between the LPL and C groups, did not reach significance levels when the LPL group was compared to the LES group. Re-alimentation did not significantly change the percentage of fat in the intermuscular fat of the leg, and the loin. In contrast to these treatment effects on the leg and loin, there was no significant effect, due to a short period of undernutrition, on the percentage or weight of chemical fat in the intermuscular fat of the rib-cut. Prolonged undernutrition significantly reduced the weight and percentage of chemical fat in this rib-cut tissue, when the LPL ewes were compared with the controls. Re-alimentation did not significantly change the percentage or weight of chemical fat in the rib-cut. The recovery group is significantly lower than the control in the percentage of fat.

No treatment effects were shown on either the percentage or weight of water in the intermuscular fat of the rib-cut. Undernutrition however produced a highly significant increase in both the percentage and weight of water present in the loin and leg. Re-alimentation also increased the amount of water present, and the recovery group has a highly significantly greater amount of both percentage and weight of water in these tissues than the control ewes. Prolonged undernutrition did not significantly change the percentage or weight of water in the leg, or loin, except for the percentage of water in the loin, this being highly significantly increased by a prolonged period of sub-maintenance feeding.

The percentage and weight of protein in the intermuscular fat of the rib-cut, leg, and loin do not show any consistent treatment effects, and will not be presented.

The percentage and weight of chemical fat and water in the perirenal fat

The analyses of variance components are presented in Appendix V. Table 22 gives the group means, standard deviations, and error mean squares of the percentage and weight of fat, and water in the perirenal fat.

TABLE 22. Percentage and weight (g.) of chemical components of perirenal fat.  
Group means, standard deviations, and error mean squares

		<u>Chemical fat</u>				<u>Water</u>			
		<u>Percentage</u>		<u>Weight</u>		<u>Percentage</u>		<u>Weight</u>	
<u>Group</u>	<u>No.</u>	<u>Mean</u>	<u>S.D.</u>	<u>Mean</u>	<u>S.D.</u>	<u>Mean</u>	<u>S.D.</u>	<u>Mean</u>	<u>S.D.</u>
C	8	87.9	2.7	463.8	189.1	10.0	2.2	49.9	14.2
LPS	8	90.7	1.8	608.7	196.0	8.4	1.7	54.1	9.5
LPS:HP	7	89.5	2.1	460.2	157.8	9.1	1.7	45.0	9.9
LPL	9	89.7	2.5	464.0	126.4	8.6	2.2	42.2	7.6
E.M.S.		5.56		28461.0		4.11		111.84	

No treatment effects were evident on the percentage or weight of chemical fat, or on the percentage and weight of water in the perirenal fat.

(8) Treatment effects on some non-carass components of liveweight

(a) Weight of gastrointestinal contents and weight of tract

In Table 23 group means, standard deviations, and the error mean squares of the weights of the contents of the gastrointestinal, gastric, and intestinal tract are shown, together with the weight of the empty gastrointestinal tract, and its two segments. The analyses of variance of each of these items are presented in Appendix VI.

No treatment effects on the weight of the contents of the gastrointestinal, gastric or intestinal tract were apparent. This effect is of considerable importance, since variation in the amount of "fill" is regarded as one of the major sources of variation in liveweight. The greater water content of the LPS and LPL groups appears to be the reason for the lack of treatment effects on the gastrointestinal tract contents.

TABLE 23. Weight of the gastro-intestinal contents, gastric contents, intestinal contents, and of the empty gastro-intestinal tract and its two segments (g.).

Group means, standard deviations, and error mean squares

Group	No.	Gastro-intestinal contents		Gastric contents		Intestinal contents		Empty gastro-intestinal tract		Empty gastric tract		Empty intestinal tract	
		Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
C	8	5186	1900	3898	1213	1287	1011	3880	388	1906	157	1974	244
IPS	8	4667	1374	3343	1402	1323	276	2806	228	1370	196	1435	72
IPS:HP	7	6484*	948	5020*	831	1474	322	3255	204	1593	165	1662	71
IFL	9	4832	1369	3591	1209	1240	417	2626	433	1276	230	1349	223
H.M.S.		2146532		1453505		346924		113289		36894		31283	

\* The weight of the gastric contents from one sheep of this group was inadvertently not taken.

Highly significant reductions in the weight of the empty gastrointestinal, gastric and intestinal tracts were produced by a short period of undernutrition. No further reduction in the weight of the gastrointestinal tract, or its two segments were shown after prolonged submaintenance feeding. Re-alimentation after 21 days undernutrition resulted in a significant increase in the weight of these three items, although at the time of slaughter the recovery group (LPS:HP) ewes had highly significantly lower gastrointestinal tracts than the control group. This resulted in the re-alimentated group being highly significantly heavier than the IFL group. The extreme loss in weight of the gastrointestinal tract with prolonged undernutrition is in the order of 37% and 31% for the gastric and intestinal weights respectively, as compared with the control ewes.

(b) Treatment effects on the weight and chemical composition of the heart

In Table 24 group means, standard deviations, and the error mean squares of the weight and chemical composition of the heart are presented. The results of the analyses of variance of these items are given in Appendix VI.

A highly significant reduction in the weight of the heart occurred with prolonged undernutrition. Although a short period of undernutrition did not significantly effect the heart weight, it is interesting to note that the mean heart weight of the LPS ewes is less than the control group.

No significant effects due to treatment were demonstrated on the percentage or weight of chemical fat in the heart, or on the percentage of water present. Prolonged undernutrition however did significantly reduce the weight of water in this organ indicating muscle dehydration.

The percentage and weight of protein in the heart were not affected by a short period of undernutrition, but were significantly reduced by prolonged undernutrition. The weight of protein in the IFL group was highly significantly lower than the control ewes, while no differences were apparent on a percentage basis. Re-alimentation appears to have further reduced the protein present, as the percentage and the weight of protein in the recovery group are highly significantly lower than the control group. No differences are evident between the LPS:HP and IFL groups.

TABLE 24. Treatment effects on the dissected weight (g.), percentage and weight of chemical components of the heart. Group means, standard deviations, and error mean squares

Group	No.	Weight of Heart		Chemical fat		Water		Protein							
		Mean	S.D.	Percentage	Weight	Percentage	Weight	Percentage	Weight						
C	8	302.0	31.0	48.5	3.4	55.7	11.0	65.5	3.7	198.5	27.5	45.1	1.0	45.7	3.1
IPS	8	260.1	15.8	16.6	3.4	44.5	9.5	66.6	3.3	178.2	15.0	15.5	0.8	41.6	3.1
IPS+IP	7	282.8	13.3	19.9	3.3	56.4	10.9	66.3	3.4	187.4	11.5	13.0	0.7	36.9	1.6
IPZ	9	251.6	53.9	20.4	4.3	51.5	15.5	64.7	3.6	162.4	36.6	14.0	1.6	35.3	8.6
E.M.S.		1175.1		13.82		148.71		12.57		659.21		1.39		27.24	

TABLE 25. Weight of the thyroid gland, liver, kidneys, pancreas, and skin plus wool (g.).  
Group means, standard deviations, and error mean squares

Group	No.	Thyroid gland		Liver		Kidneys		Pancreas		Skin plus wool	
		Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
C	8	31.8	1.9	871.5	96.3	146.3	37.2	85.2	13.0	6622.3	1168.9
IPS	8	9.2	2.1	647.5	196.7	134.5	15.4	65.7	12.8	5901.8	579.9
IPS:HP	7	8.6	1.8	810.0	76.1	151.8	14.9	85.8	10.4	6945.8	649.5
IPI	9	6.8	1.5	634.4	130.8	129.6	20.5	64.2	11.0	5785.4	915.1
E.M.S.			3.44		18134.4		575.82		142.14		755357.5

(c) Treatment effects on the weight of the thyroid gland, liver, kidneys, pancreas, and skin plus wool

Group means, standard deviations, and the error mean squares of the weights of the thyroid gland, liver, kidneys, pancreas, and skin plus wool are shown in Table 25. The results of the analyses of variance of these organs are presented in Appendix VI.

A short and prolonged period of undernutrition reduced the weight of the thyroid gland, highly significant differences being apparent between the LPS and LPL groups and between these groups and the control. Re-alimentation did not increase the weight of the thyroid gland, in fact no difference was shown between the recovery group and the LPL group. It should also be noted that there was a large initial fall in the thyroid gland weight with undernutrition, in the order of 78% for the LPL ewes, and 71% for the LPS group compared with the control ewes.

A period of 21 days undernutrition highly significantly reduced the weight of the liver, while a further period on a low plane of nutrition did not appear to increase this loss, although the LPL group have a lower mean liver weight than any other treatment group. Re-alimentation significantly increased the liver weight, there being no difference between the recovery group and the control group at slaughter.

No significant effect due to plane of nutrition was produced on the weight of both kidneys, but it is noted that the mean weight of the LPL ewes was lower than in any other group.

A low plane of nutrition produced a highly significant reduction in the weight of the pancreas after 21 days submaintenance feeding, and no apparent further change on prolonged undernutrition. Re-alimentation resulted in a highly significant increase in weight, there being no difference between the recovery group and the controls. The LPL ewes had highly significantly lower pancreas weights than the control ewes, and the LPS:HP group.

Statistical analysis showed that a short or prolonged period of undernutrition did not significantly produce a significant effect on the weight of skin plus wool. Re-alimentation significantly increased the weight, there being no difference between

the recovery group and the controls. These results are difficult to interpret due to the varying lengths of wool present between the treatment groups. It is interesting to note however that the mean length of wool based on three measurements per sheep at the mid-shoulder, trunk, and leg positions, for all sheep except the control ewes, was 3.4 cm. on 8 January. The mean length of wool on the ewes in the LFS, LFS:HP, and IPL groups was 4.0 cm., 5.1 cm., and 4.2 cm. respectively at the time of slaughter. This would support the view that a real treatment effect was present on the weight of the skin per se.

(d) Treatment effects on the weight of the omental and mesenteric fat depots, spleen, genital tract plus urinary bladder

Group means, standard deviations, and error mean squares for these items are given in Table 26, while the results of the analyses of variance are given in Appendix VI.

TABLE 26. Weight of the omental and mesenteric fat depots, spleen, genital tract plus urinary bladder (g.).  
Group means, standard deviations, and error mean squares

<u>Group</u>	<u>No.</u>	<u>Omental fat</u>		<u>Mesenteric fat</u>		<u>Spleen</u>		<u>Genital tract plus urinary bladder</u>	
		<u>Mean</u>	<u>S.D.</u>	<u>Mean</u>	<u>S.D.</u>	<u>Mean</u>	<u>S.D.</u>	<u>Mean</u>	<u>S.D.</u>
C	8	2264.6	770.7	794.8	193.7	115.1	15.2	244.5	90.8
LFS	8	2085.8	422.8	777.1	77.3	105.2	12.2	244.6	81.1
LFS:HP	7	2235.2	704.4	931.5	257.3	104.0	11.1	190.4	63.3
IPL	9	2045.5	668.0	675.0	153.8	123.7	53.8	206.5	33.7
E.M.S.		426147.8		31830.5		951.71		4894.8	

No significant treatment effects on the omental or mesenteric fat depots, the spleen, or the genital tract plus urinary bladder were apparent. It is noted that the mean weight of mesenteric and omental fat in the IPL ewes was considerably lower than the control ewes, which may suggest that the treatment was starting to produce some effect.

(9) Internal organs as indices of composition

Table 27 presents some correlations between the weight and chemical composition of the heart and the carcass.

TABLE 27. Correlations between the weight and chemical composition of the heart, and the carcass, for 32 ewes

	<u>Heart</u>							
	<u>Weight</u>	<u>Fat-free weight</u>	<u>% Water</u>	<u>Water Wt.</u>	<u>% protein</u>	<u>Protein Wt.</u>	<u>% fat</u>	<u>Fat Wt.</u>
Corresponding Carcass Component	0.416 (s)	0.621 (s.s.)	0.345 (n.s.)	0.615 (s.s.)	-0.078 (n.s.)	0.507 (s.s.)	0.143 (n.s.)	0.092 (n.s.)

These results indicate that the weight, fat-free weight, water weight, and the weight of protein in the heart are significantly related to the same components in the carcass, but that this organ is unlikely to be of use in predicting carcass composition. It is of interest to note that the amount of chemical fat in the heart does not appear to be related to that in the carcass.

Some correlations between selected internal organ weights and carcass composition are given in Table 28.

TABLE 28. Correlations between some internal organ weights and the weight of the fat-free carcass, weight of carcass protein, and carcass chemical fat, for 32 ewes

	<u>Heart wt.</u>	<u>Kidneys wt.</u>	<u>Liver wt.</u>	<u>Omental fat wt.</u>	<u>Mesenteric fat wt.</u>	<u>Omental + Mesenteric fat wt.</u>
Hot carcass wt.	+0.416 (s)	+0.125 (n.s.)	+0.267 (n.s.)			
Fat-free carcass wt.	+0.542 (s.s.)	+0.099 (n.s.)	+0.479 (s.s.)			
Carcass protein (lb.)			+0.595 (s.s.)			
Chemical fat				+0.579 (s.s.)	+0.429 (s)	+0.608 (s.s.)

These results indicate that these organ weights are unlikely to be of use in predicting carcass weight, or its composition.

### DISCUSSION

An outline of the significant treatment effects on the carcass and non-carcass components of liveweight are presented in Table 29.

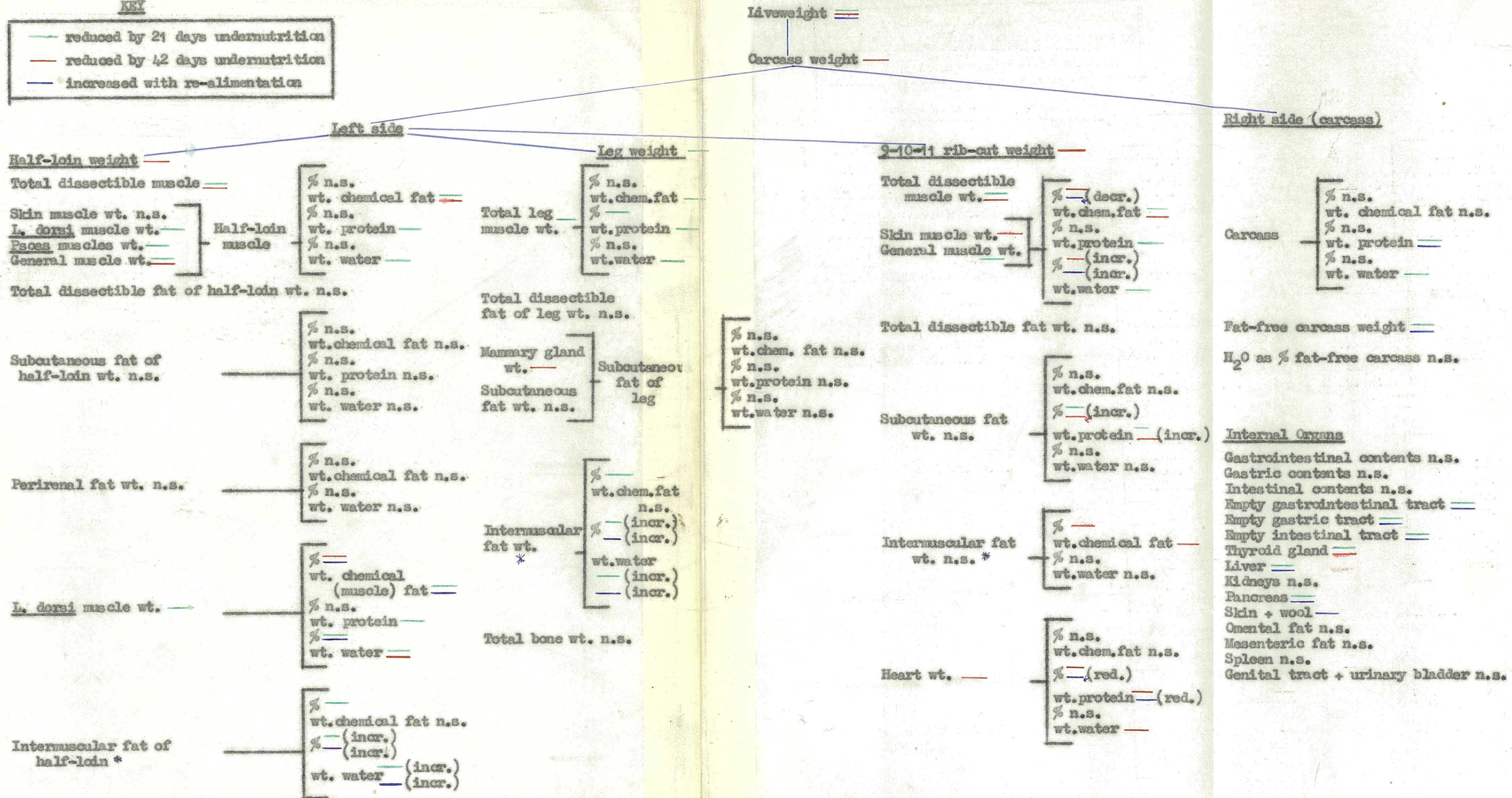
As was expected a short and prolonged period of undernutrition both significantly reduced liveweight, while re-alimentation tended to increase liveweight. This reduction in liveweight resulted in a significant decrease in carcass weight on prolonged undernutrition, but a short period of undernutrition, or of re-alimentation, did not significantly change carcass weight. Kirton and Barton (1958a) also showed that a short period of undernutrition did not reduce the carcass weight.

It is of considerable interest to note that no treatment effects were shown on the chemical fat in the carcass, or the dissectible fat in the leg, half-loin, or 9-10-11 rib-cut, nor in the various chemical fat determinations made. No treatment effects were demonstrated on the physical or chemical components of the perirenal fat. Prolonged undernutrition reduced the dissectible weight of the mammary gland. Except for the protein in the subcutaneous fat of the 9-10-11 rib-cut no treatment effects were demonstrated on the chemical fat, protein, or water in the subcutaneous or perirenal fat depots studied. Although a high variability of these data are evident these results indicate that surplus fat cannot readily be removed from mature Romney ewes by submaintenance feeding without endangering their lives. This result is supported by the observation of Kirton and Barton (1958a) but is at variance with a number of workers, (Mitchell et al. 1928; Pomeroy, 1941; Robinson, 1948; Widdowson and McEance, 1956; Garn and Brozek, 1956; Keys and Brozek, 1953; Riney, 1953). A high variability of the fat content of the tissues, a change in the susceptibility of fat depots to undernutrition with aging, or species differences in resistance to undernutrition are possible ways of explaining the lack of agreement in this respect. If the treatment size had been increased to 26 there would have been a good chance ( $P = 0.75$ ) of detecting a difference of 20 per cent. of the mean weight of carcass chemical fat. A sample size of 11 animals would however, have been sufficient for a similar precision in the percentage of carcass chemical fat, (Snedecor, p.280).

TABLE 29. Summary of the Effects of Undernutrition and Re-alimentation on Carcass Composition and Non-carcass Components

KEY

- reduced by 21 days undernutrition
- reduced by 42 days undernutrition
- increased with re-alimentation



\* Effects probably influenced by dissection technique

In contrast to most other workers the treatments imposed were more severe and prolonged, and the measurement of response more sensitive.

Analysis of the treatment effects on the intermuscular fat weight, and the chemical components of this tissue in the leg, half-loin, and 9-10-11 rib-cut produced unexpected results. Undernutrition was not effective in reducing the chemical fat in this tissue, except in the 9-10-11 rib-cut, and this supports observations noted in other fat depots. There was however, a variable treatment response on the water and protein contents of these intermuscular fat depots. These results appear to be due to the variable amount of muscular tissue dissected with the intermuscular fat. This appears to be the first time the chemical components of this fat depot have been studied. Because of this effect the weight of intermuscular fat in these three joints appears to be unsuitable as a criterion of response in investigations of the nature reported here.

In contrast to the lack of treatment effects on both dissectible and chemical fat were the marked treatment effects and low variability on the weight of the chemical fat-free carcass. That a short period of undernutrition reduces the fat-free carcass weight agrees with the observation of Kirton and Barton, (1958a). More prolonged undernutrition appears to result in less reduction, while re-alimentation significantly increased the weight of the fat-free carcass. When the fat-free carcass was partitioned into its various components, it was found that a low plane of nutrition for 21 days had caused a reduction of both protein and water but further undernutrition did not significantly increase this loss. The weight of carcass protein was significantly increased by re-alimentation. Protein and water loss together are indicative of muscular tissue being reduced. This was confirmed by an analysis of the physical and chemical components of the muscles in the half-loin, leg, 9-10-11 rib-cut, and in the l. dorsi muscle. The greater part of the loss of protein and water in the carcass and these muscles appears to have occurred during the first 21 days of undernutrition, although prolonged undernutrition significantly reduced the dissectible weight of the skin muscle in the 9-10-11 rib-cut, the general muscle

of the half-loin, and significantly reduced the weight of water in the l. dorsi muscle. Re-alimentation did not significantly increase the weight of protein in the muscle of these sample joints, but the weight of water was increased in the l. dorsi muscle. It is of considerable interest to note that muscle fat was reduced by undernutrition in the l. dorsi, half-loin, leg, and rib-cut muscles, with prolonged undernutrition further reducing this component. This was the only fat depot, other than the mammary gland, and probably the intermuscular fat depots which was shown conclusively to be reduced by undernutrition. Except for the chemical fat in the l. dorsi muscle re-alimentation was ineffective in increasing the muscle fat. A number of workers, (Jackson, 1915; Pomeroy, 1944; Robinson, 1948; Wallace, 1948; Addis et al. 1936<sub>a</sub>, and b; Kosterlitz and Campbell, 1945; Keys et al. 1950; Kirton and Barton, 1958<sub>a</sub>) have shown that muscular tissue and its components are sensitive to undernutrition. That there is a lag period after re-alimentation before protein repletion commences is supported by Summers and Fisher, (1960). The muscular dehydration noted as a result of undernutrition also seems to be a characteristic feature.

Treatments were not effective in either reducing or increasing the weight of bone in the leg, and since the weight of bone in the leg is closely related to the total carcass bone weight, (Barton and Kirton, 1958) it was unlikely that bone in other regions of the carcass would be affected by undernutrition, or re-alimentation. Pomeroy (1944), and Keys et al. (1950), maintained that bone weight was reduced by severe undernutrition, while Robinson (1948), and later Kirton and Barton, (1958<sub>a</sub>) with sheep found that bone weight was not reduced by undernutrition. Variation in the lability of bone constituents between ages and species could account for the lack of agreement between investigators.

Relationships between chemical and physical components of the carcass and various sample joints have been reported by a number of workers, (Chatfield, 1926; Hopper, 1944; Hankins and Howe, 1946; Callow, 1948; Kirton, 1957; Shorland et al. 1947; and Ulyatt, 1960). It is of interest to note that closely similar results were found on the dissected muscular and fatty tissue weights as on the chemical components of these tissues. In general the chemical components tend to be a more sensitive

criterion of treatment effects, and provide more information on the components of tissue weight changes than do changes in dissectible tissue weights.

The use of sample joints has been discussed by several authors, (Palsson, 1939; Barton and Kirton, 1958; Shorland et al., 1947; Hankins, 1953; Lush, 1926; Hankins and Ellis, 1939; Hankins et al., 1943; Hopper, 1944; Hankins and Howe, 1946; Hankins, 1947; Palsson and Verges, 1952; and McMeekan, 1940-41) who have emphasized their value in the determination of carcass composition. The composition of these three samples could be expected to be representative of the whole carcass and its changes. The leg, an early developing joint, would be expected to be less affected by undernutrition than the half-loin. While there is little indication of a differential effect on fat depots in these joints, the muscle components of the half-loin appear to have been more affected by undernutrition than the muscular tissue in the leg. The high proportion of muscular tissue, and the low proportion of fatty tissue could provide the reason for the significant reduction in the weight of the leg, and the converse appears to explain the absence of significant effects due to 21 days of undernutrition on the half-loin and 9-10-11 rib-cut weight. More prolonged undernutrition appears to have had a greater effect on the half-loin and 9-10-11 rib-cut weight than on the leg weight. This explanation was justified in view of the absence of significant treatment effects on fatty tissue, and the amounts of chemical fat in these regions.

No treatment effects were demonstrated on the proportion of water in the fat-free carcass. The mean value was 72.8% (70.9 - 74.7%). This percentage although derived from frozen carcass weight is in agreement with several authors using the sheep or some other species, (Murray, 1922; Behnke, 1942; Behnke, 1953; Pace and Rathbun, 1945; Kraybill et al., 1952; Callow, 1947; Babineau and Page, 1955; Ellenberger et al., 1950; Reid et al., 1955; Kirton and Barton, 1958; Garrett et al., 1959; Kirton et al., 1960). Some of these workers have regarded this figure as a 'biological constant', and it has been extensively used in the indirect determination of carcass composition. Increased hydration at lower levels of fatness has been noted for the

sheep (Mitchell et al., 1928), and the pig, (Clawson et al., 1955). Other factors thought to affect the composition of the fat-free carcass or body include thyroxine treatment, (Kirton and Barton, 1958) pregnancy, (Dewar, 1957; Keys et al., 1950) and edema, (Keys et al., 1950) resulting from undernutrition and disease. It is concluded that the composition of the fat-free body is not strictly a constant and that the use of this in the indirect determination of body composition could lead to appreciable errors in critical studies.

Significant treatment effects were shown on the following linear measurements; W.Th., W.F., G., and B. Linear measurements on the carcass, and the cross-sectional area of the loin have been studied by several authors, (Pålsson, 1939; McMeekan, 1940-44; Walker and McMeekan, 1944; Kraybill et al., 1954; Hammond, 1932; Whiteman and Whatley, 1953; and Robinson et al., 1956). Bodwell et al. (1959) showed that some carcass measurements cannot be taken with a high repeatability. Further studies showed that the value of carcass measurements for predicting the yield of wholesale and retail joints may have been overestimated, (Bodwell et al., 1959).

It is of considerable interest to note that no treatment effects were shown on the weights of the gastrointestinal, gastric, or intestinal contents. An apparent increased wateriness of the contents of the tract seemed to be a characteristic feature of a reduction in food intake. Substitution of dry matter by water at low levels of food intake has been noted by Ritzman and Benedict, (1938); Nevens, (1928); Quin, (1943); and Phillips, (1960). Kirton and Barton, (1958) have recorded a similar observation during a self-imposed starvation in ewes, while Blaxter, (1948) noted that hyperthyroidism increased the amount of water in the tract in wethers.

A low plane of nutrition for 21 days produced a significant reduction in the weight of the empty gastrointestinal tract, gastric, and intestinal tracts. Prolonged undernutrition did not accentuate this effect. Re-alimentation significantly increased the weight of these three items. This further emphasizes that the weight of the empty gastrointestinal tract is sensitive to the effects of undernutrition, (Jackson, 1915; Pomeroy, 1944; Robinson, 1948; Kirton and Barton, 1958a) despite the fact that the weights of the contents of the tract were not affected.

Treatment effects could not be demonstrated on the weight of the omental or mesenteric fat depots. These results agree with those of Robinson, (1948), and Kirton and Barton, (1958a). Robinson, (1948) was unable to demonstrate a fat loss from the internal depots of ewes which had lost up to 50 per cent. of their original body weight. The weights of omental, and mesenteric fat depots were significantly correlated with the weights of carcass chemical fat, but the relationship is not close enough for predictive purposes. This confirms the observation of Lush, (1926), and Kirton, (1957).

No treatment effects were demonstrated on the weights of the kidneys, spleen, or genital tract plus urinary bladder. Keys et al., (1950); Pomeroy, (1944); Robinson, (1948); and Kirton and Barton, (1958a) maintained that the weight of the kidneys was sensitive to plane of nutrition. There does not appear to be any apparent reason for the discrepancy of these results. Pitts, (1951); Brody, (1945); Kraybill et al., (1954) reported close relationships between the weight of the heart, liver, and kidneys with the fat-free carcass or body weight. Kirton, (1957) also noted highly significant correlations between the fat-free carcass weight and liver weight, kidney weight, and heart weight, but the relationships were lower than that reported by previous workers. The present results indicate that there is no relation between the weight of the kidneys and carcass weight, or the weight of the fat-free carcass.

The lack of treatment effects on the weight of the spleen were in disagreement with evidence reported by Jackson, (1913); Stewart, (1919); Kudo, (1921); Jackson, (1915); and Keys et al., (1950), but agrees with the results noted by Robinson, (1948), and Kirton and Barton, (1958b) with sheep. It is possible that the spleen of the sheep is more resistant to undernutrition than those of other species or alternatively the degree of undernutrition in the sheep investigations was less severe than for those species in other experiments where treatment effects were shown. Considerable variability in spleen weight is apparent.

The thyroid gland showed a large reduction in weight after 21 days of under-

nutrition, and a further decrease in weight on prolonged undernutrition. Re-alimentation was without appreciable effect. This large reduction was in the order of 78% for the LPL ewes and 71% for the LIS groups respectively compared with the control ewes. The observation that the thyroid gland atrophies with inanition is supported by Keys et al. (1950). Kirton and Barton, (1958b) with sheep were unable to demonstrate a decrease in the weight of the thyroid gland, or in follicular cell heights after undernutrition, (Munford and Kirton, 1958). This may have been due to the stress of undernutrition imposed by Kirton and Barton (1958) not being severe enough. This decrease in thyroid gland weight could be expected to result in a decreased activity of the thyroid gland, (Brown-Grant et al. 1954; 1957; Brown-Grant, 1956; Pipes et al. 1960; Brown-Grant and Rethes, 1960), probably as a result of increased ACTH secretion. The decrease in thyroid activity and therefore metabolic rate, (Blaxter and Wood, 1951; Benedict and Ritzman, 1927; Ritzman and Benedict, 1938; Cresswell, 1958), may be the primary reason why prolonged undernutrition did not greatly increase the tissue losses shown to occur as a result of 21 days of undernutrition.

The loss in liver weight attributable to a low plane of nutrition for 21 days reported here is in agreement with that noted by Jackson, (1932); Kosterlitz and Campbell, (1945); Pomeroy, (1944); Robinson, (1948); Keys et al. (1950); Widdowson and McCance, (1956); and Kirton and Barton, (1958). A further period of undernutrition does not appear to have greatly increased this loss. This organ appears to be one of the first components of body weight to recover in weight on re-alimentation. The highly significant correlations reported between Liver weight and the weight of the carcass protein, or the fat-free carcass confirm the results noted by previous authors, but the relationships are not high enough for predictive purposes.

A prolonged period of undernutrition reduced the weight of the heart, this weight reduction being due to a highly significant decrease in the weight of protein and of water. In contrast to the effect on other muscular tissues studied the

weight of the heart, protein, or water weight, were not significantly reduced by a short period of undernutrition. Cardiac muscle may be more resistant to the effects of undernutrition than skeletal muscle. The percentage or weight of chemical fat in the heart was not affected by the treatments imposed, and this is also at variance with results noted for skeletal muscle. Re-alimentation reduced the percentage and weight of protein in the heart. That a reduction in the weight of the heart is a characteristic feature of undernutrition has been confirmed by Pomeroy, (1944); Keys et al. (1950); and Kirton and Barton, (1958). It is of interest to note that the weight, fat-free heart weight, water weight, and protein weight were significantly correlated with the same chemical entities in the carcass, but these correlations are not sufficiently high for predictive purposes. This appears to be the first time the chemical composition of the heart has been related to that of the carcass. Other internal organs (liver, lungs) would appear to be less useful than the heart for predicting carcass composition due to the high incidence of disease.

Re-alimentation was ineffective in increasing the weight of the chemical components studied, except for carcass fat-free body weight, protein weight, and the weight of chemical fat in the l. dorsi muscle. A decrease in the weight of heart protein was noted following re-alimentation. In contrast to this general effect on carcass components, the gastrointestinal tract, liver, and pancreas showed marked increases in weight on re-alimentation. Consequently there appears to be a considerable lag after re-alimentation before the aged Romney ewe recovers its original composition. The lack of recovery in the thyroid gland is also indicative of delayed physiological recovery. Recovery seems to be related to the depth, and length of undernutrition. Those animals in the groups given a prolonged low plane of nutrition would appear to have reached the limit of liveweight loss from which recovery was possible, at least during the relatively short time of the experiment.

This experiment emphasizes the desirability in critical experiments to determine with precision the major components of the liveweight loss (or gain) arising from various treatments in animal experiments.

SUMMARY

1. An experiment involving 50 mature Romney ewes is described. These animals were randomized into 6 treatment groups each of 8 ewes, so that mean liveweights were approximately equal. The average liveweight of the ewes at the beginning of the experiment was 160.8 lb. The six treatment groups were the control, (C); low plane of nutrition for 21 days, (LPS); low plane of nutrition for 21 days followed by high plane, (LPS:HP); low plane of nutrition for 42 days, (LPL); low plane of nutrition for 42 days followed by high plane for a short period, (LPL:HPS); low plane of nutrition for 42 days followed by high plane for a long period, (LPL:HPL). The last two groups were not considered in the data analyses.
2. The period of 21 days undernutrition produced a mean reduction of 20.0 lb. in liveweight, in addition to a pre-experimental liveweight loss. The total loss in liveweight was 30.6 lb. at the end of the 21 day period. A further 21 days of undernutrition produced an additional 12.8 lb. reduction in liveweight. At slaughter the LPL group had a mean liveweight of 115.4 lb. Re-alimentation for 38 days, after 21 days of submaintenance feeding, increased the liveweight to a level comparable to the control group at slaughter. After 42 days undernutrition, re-alimentation was ineffective in increasing liveweight.
3. An 18 per cent. death loss occurred.
4. Despite significant effects on liveweight, the prolonged period of undernutrition was the only treatment that significantly reduced the carcass weight. Re-alimentation did not significantly increase carcass weight.
5. No significant treatment effects were demonstrated on carcass fat, dissectible fat in the leg, 9-10-11 rib-cut, or half-loin, or their chemical components, or on the perirenal fat and its chemical components. A significant reduction in the weight of the mammary gland was noted after 42 days undernutrition.
6. The measure of intermuscular fat was found to be unsatisfactory.

7. A period of 21 days undernutrition reduced the weight of the fat-free carcass, carcass water, and protein weight. Re-alimentation after 21 days significantly increased the weight of the fat-free carcass.
8. Carcass weight and water loss are indicative of muscular tissue reduction. This was confirmed for the dissectible muscle of the leg, half-loin, 9-10-11 rib-cut, and their chemical components. Prolonged undernutrition did not accentuate this effect. Re-alimentation was not effective in increasing the dissectible weight, protein, or water weight in the muscles.
9. The muscle fat in the half-loin, l. dorsi, leg, and 9-10-11 rib-cut muscles was reduced by undernutrition.
10. Undernutrition resulted in muscular dehydration.
11. No treatment effects could be shown on the weight of bone from the leg.
12. The proportion of water in the fat-free carcass was not affected by the treatment imposed.
13. The weight of the heart was significantly reduced by prolonged undernutrition, this reduction being due to a loss of protein and water. No treatment effects were shown on the percentage or weight of chemical fat in the heart. Re-alimentation further reduced the weight of heart protein. The chemical composition of the heart was highly correlated with carcass composition, but these relationships are not sufficiently high for predictive purposes.
14. No treatment effects were demonstrated on the weight of the gastrointestinal contents.
15. A short period of undernutrition lowered the weight of the empty gastrointestinal tract, and its two components, while prolonged undernutrition did not accentuate this effect. Re-alimentation increased the weight of these three items.
16. A short and prolonged period of undernutrition both produced a marked reduction in the weight of the thyroid gland. Re-alimentation did not increase thyroid gland weight.

17. No treatment effects were demonstrated on the weight of the omental or mesenteric fat depots, the kidneys, spleen, or genital tract plus urinary bladder. While the omental and mesenteric fat depots, and the liver were significantly related to carcass composition these relationships were not high enough for predictive purposes. The weight of the kidneys was not related to carcass composition.
18. A short period of undernutrition reduced the weight of the liver and pancreas, while re-alimentation increased the weight of these two organs.
19. The recovery after undernutrition is influenced by the depth and length of undernutrition.

PART II

HAEMATOLOGICAL STUDIES

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## CHAPTER I

### REVIEW OF LITERATURE

Changes in blood constituents are frequently used to study alterations in metabolism, or pathological conditions. Their interpretation may be difficult or misleading if the conditions are accompanied by a reduced food intake, which may itself bring about changes in blood constituents. There have been few systematic experiments made on the blood constituents of ruminants.

Several authors (Josland, 1933; Filmer, 1933; Hamersma, 1934; Underwood *et al.*, 1939; Allcroft, 1944; Dukes, 1947; Mehrotra *et al.*, 1954; Fraser, 1929-30; Gresswell, 1958; Weeth *et al.*, 1959; White *et al.*, 1956; Williams and Christian, 1959; Mallick and Kehar, 1954; and Meyer *et al.*, 1955), have reported values of blood constituents for the sheep, but few have presented values for the differential white cell count. The breed of sheep, age, feeding level, pregnancy, and sex have usually not been specified. Disease is probably one of the most important factors causing changes in blood constituents, but it does not appear to have been quantitatively analysed. Coffin (1953) presents "normal" values and ranges of blood cellular elements for the sheep. These are given in Table 30.

TABIE 30. "Normal" values and ranges of blood cellular elements for the sheep

R.B.C. m/c.mm.	Hbg g/100 c.c.	P.C.V.%	M.C.H. u.u.g.	M.C.V. c.u.	M.C.H.C. %	Platelet m.
8.5-13.5	9-14.5	33-46	9.0-13	33.5-43.0	33-35	0.25-0.7
Reticulocytes %	Total W.B.C. thou./c.m.m.	Neutrophils %	Eosinophils %	Basophils %	Lymphocytes %	Monocyte %
0	4-12	40 (20-50)	6 (0-15)	0.2 (0-2)	52 (40-70)	4 (1-12)

Fraser (1930) reported that an increase in lymphocytes and eosinophils, and a decrease of monocytes indicates a recovery from the diseased condition, while an extension of lesions is indicated by a decrease of monocytes and eosinophils. Gresswell (1958) found significant differences between Cheviot and Romney sheep for

Hbg, P.C.V.%, and leucocyte counts. Seasonal variation in the blood composition of sheep have been noted by Mullick and Kehar, (1954), and Cresswell, (1958). Sex and age differences have been shown by Reeve, (1948). Blood volume appears to change with the state of pregnancy in sheep, (Barcroft et al., 1939; Reeve, 1948). Each animal appears to have a distinctive haematological picture, and this may be an expression of physiological individuality.

Little appears to be known of the physiological functions of leucocytes. The number of leucocytes in circulation varies at different times of the day and during digestion, (Maximow and Bloom, 1948). Consequently the leucocyte counts frequently have only a relative value. Some leucocytes are phagocytic, all are prominent in various types of inflammation. Large numbers of lymphocytes may degenerate in the lymphatic tissue, and also by migration into the intestinal cavity. The manner and site of destruction of erythrocytes are not clarified, but the spleen, liver, bone marrow, and the circulatory system itself are possible sites of degeneration. It will be recalled in this connection that the weight of the liver was significantly decreased by undernutrition, and increased on re-alimentation, but the weight of the spleen was not significantly changed by undernutrition or re-alimentation.

Meyer et al. (1955) found that the haematocrit value increased with sheep deprived for 36 hours of food and water. White et al. (1956) found at intakes of 400 g. or more of dried grass, that there were no marked changes in the blood components of wethers. Further intake reductions resulted in a lowering of circulating eosinophils, lymphocytes, and of total leucocytes. Haemoglobin and haematocrit also appeared to be reduced. This indicates that large reductions in food intake may take place before major changes in the blood constituents occur. Widdowson and McCance (1956) noted an increase in circulating haemoglobin of growing and adult rats during starvation. Williams and Christian (1959), found that the neutrophil per cent., lymphocyte per cent haemoglobin, and haematocrit of non-pregnant ewes was not influenced by a sudden reduction of food intake. A pronounced eosinopenia was noted with pregnant ewes, (Weeth et al., 1959). Haemococentration was also present during fasting.

McCance (1960) found that undernutrition in pigs decreased the erythrocytes and the total leucocytes, while haemoglobin increased.

Selye and Heuser (1955-56) claim that the organism responds in a stereotypical manner to a variety of widely different factors, including infections, cold, and muscle fatigue, which place the body in a state of stress. The adrenal cortex is thought to be of major importance in the response of an organism to stress, (Sayers, 1950; Thom et al. 1953). Stress affects the adrenal cortex by acting through the hypophysis causing increased secretion of corticotrophic hormone, which acts on the adrenal cortex resulting in the release of corticoid hormones, (Selye, 1948). Long et al. (1940) present evidence which supports the belief that the adrenal cortex influences carbohydrate and protein metabolism. It appears that in the fasting state, and under the influence of cortical hormone there occurs an increased rate of protein catabolism. In normal animals there is a marked diminution in the number of circulating eosinophils in response to the injection of adrenotropic hormone (ACTH), epinephrine, insulin, and to the stimuli of various stresses, (Sayers, 1950; Thom et al. 1953). Eosinopenia (decrease in the per cent. of eosinophils) can also be produced by a number of stresses, such as handling in mice (Spiers and Meyer, 1949); fasting in rats, (Butler and Morgan, 1953); and parturition or injection of ACTH in cows, (Merrill and Smith, 1954). Thus since Hills et al. (1948) reported that the number of circulating eosinophils is indicative of adrenal cortical secretion, the eosinophil response test has been widely used as an assay of adrenocortical function, (Spiers and Meyer, 1951). Wynne (1954) found that a three-day infusion of ACTH resulted in the complete disappearance of eosinophils from the blood of a pregnant ewe. Benson and Cowie (1957) claim that changes in the levels of 17-hydroxycorticoids in the blood affect the numbers of eosinophils and lymphocytes in circulation.

A lymphopenia (decrease in the per cent. of lymphocytes) also appears to occur as a result of increased adrenocortical activity, (Dougherty and White, 1944, 1947; Merrill and Smith, 1954). A lymphopenia has been used as an index of adrenocortical activity, but the drop in circulating lymphocytes in man is not as great as the

eosinophils, and the response is variable, (Forsham et al., 1948). However, Sayers (1950) was of the opinion that the number of circulating lymphocytes is a valuable index of adrenocortical activity in those species in which the lymphocytes make up the greatest percentage of the white blood cells, as in the bovine. Benson and Cowie (1957) conclude that eosinophil and lymphocyte counts are useful indicators in studying changes in the levels of 17-hydroxycorticoids in the circulation of ruminants. Schultz (1959) suggested that the variability in response between individuals may be useful in selection for physiological function in calves.

Schultze (1957) found that the fattest calves tended to have the highest eosinophil counts. Arthaud et al. (1959) however, found that the erythrocyte, haemoglobin, or haematocrit were of no value in predicting the total gain of beef bulls. They concluded that the number and volume of red cells, and the haemoglobin level was largely influenced by environmental factors. With human subjects Borner et al. (1960) found that when the relative weight, (actual weight as a percentage of expected weight) and therefore "body fat" increased venous haematocrit also tended to increase. The criterion of "body fat" would appear to be unreliable.

Turner and Hodgetts (1959) demonstrated with sheep that jugular haematocrit was increased by excitement, exercise, or injection of intravenous adrenaline or nor-adrenaline, and decreased with soothing, isolation from visual and auditory stimuli or anaesthesia. The effects were attributed to the filling or contraction of the spleen, this organ being the site for the dynamic storage of red blood cells. This emphasizes the difficulty of defining a normal value for jugular haematocrit of individual sheep, and the necessity for training, quietness, and gentleness when handling sheep. A fall in of haematocrit during quiet "holding" or due to pharmacological tranquilisers (e.g. chlorpromazine) occurs, (Turner and Hodgetts, 1960) and this was attributed to splenic relaxation.

## CHAPTER II

### MATERIALS AND METHODS

Samples of blood were taken after the morning feeding period on the day prior to the slaughter of each group of animals. The ewe was restrained in a standing position with the head held slightly to the left. Wool was removed from the neck region overlying the jugular vein. Sampling with the sheep in a standing position was considered to be more convenient than with the animal in an upright sitting position as used when the control group were sampled. The jugular vein was closed off with the thumb of the left hand above the brisket, and the area palpated with the forefinger of the right hand until the vein was clearly evident. A sterilized, dry, 14 gauge needle with a diamond point was then firmly pushed into the vein at an angle of  $45^{\circ}$  and in line with the vein. Occlusion of the vein was as brief as possible in order to avoid possible haemoconcentration. The first few drops of blood were discarded. A 5 ml. sample of blood was run into a bottle containing potassium oxalate, and thoroughly mixed by repeated inversion. Two smears were made from the blood in the needle.

Blood samples were analysed by the Biological Services Laboratory, Palmerston North. Estimations of the constituents studied are frequently carried out in medical, veterinary, and agricultural investigations, and the techniques are well standardized. Detailed accounts are given by Coffin (1953). The magnitude of the errors involved are considered by Darmady and Davenport (1958). Reynolds (pers. comm.) considered that the lymphocyte and monocyte count may not be completely reliable due to the difficulty of differentiation between these two types of cells by workers inexperienced with sheep blood.

## CHAPTER III

### RESULTS

In Table 31 group means and standard deviations of the last recorded liveweight prior to the pre-slaughter starvation period and some components and indices of blood are given. Components of variance are presented in Appendix VII. The group means for these blood characteristics are shown graphically in Figs. 8 and 9. The number of individuals in each group is given in parenthesis in Table 31. Due to the high death rate in the LPS:HES and LPS:HPL groups previously described, the results for the surviving ewes in these groups were excluded from statistical analysis, but the group means are given in graphical form to indicate possible trends. One ewe (no.15) which was slaughtered with the LPL group was excluded from analysis as it was killed in extremis.

#### Sedimentation rate

No sedimentation of erythrocytes was evident over a period of one hour in all samples studied. This is to be expected in the absence of acute generalized infection.

#### Total erythrocytes (m./cmm.)

Statistical analysis showed that both a short period (21 days) and a more prolonged period (42 days) of undernutrition had no significant effect on the total erythrocyte count. A period of 41 days ad lib. feeding after 21 days undernutrition appears to have significantly reduced the erythrocyte count. It is of interest to note that no significant difference is apparent between the recovery group (LPS:HP) and the prolonged undernutrition group, (LPL).

#### Haematocrit % (P.C.V.)

These results are presented, although the P.C.V. is normally in direct proportion to the erythrocyte count and haemoglobin level, but is less subject to error. As with the erythrocyte count, both a short and prolonged period of undernutrition were without effect on the P.C.V. Re-alimentation resulted in a highly significant reduction in P.C.V. Significant differences between the LPS:HP and LPL are evident. The results

TABLE II. Some haematological components of blood. Group means and standard deviations

Item	Group C (8)		Group IFS (8)		Group IFL (8)		Group IFS:HP (7)	
	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
Liverweight (lb.)	148.1	11.0	135.1	11.6	119.5	9.9	145.7	15.5
Red Blood Cells (m./c.mm.)	11.028	1.635	12.075	1.639	10.637	1.726	9.028	1.187
Haematocrit % (P.C.V.)	43.0	5.4	46.2	7.5	43.8	5.6	37.1	3.2
Haemoglobin (g./100 ml.)	11.9	1.2	13.5	1.6	13.0	1.5	11.4	0.9
Mean Cell Volume (c.u.)	39.1	3.7	38.2	2.3	41.5	3.7	41.3	2.7
Mean Cell Haemoglobin (u.u.g.)	10.9	0.9	11.2	0.6	12.3	0.8	12.7	1.2
Mean Cell Haemoglobin Conc. (%)	27.8	1.3	29.4	2.0	29.8	1.3	30.8	1.3
Total Leucocytes (per.c. mm.)	13,771	2,177	9,037	2,006	8,162	1,984	10,000	2,565
Heterophils (%)	23.0	8.7	29.3	9.7	23.7	13.1	29.4	7.5
Eosinophils (%)	12.7	4.7	6.2	5.5	3.6	3.4	4.4	3.0
Basophils (%)	0.3	-	-	-	-	-	-	-
Lymphocytes (%)	62.1	7.4	62.7	11.3	72.1	13.6	66.0	6.9
Monocytes (%)	1.7	-	1.6	-	0.5	-	0.1	-

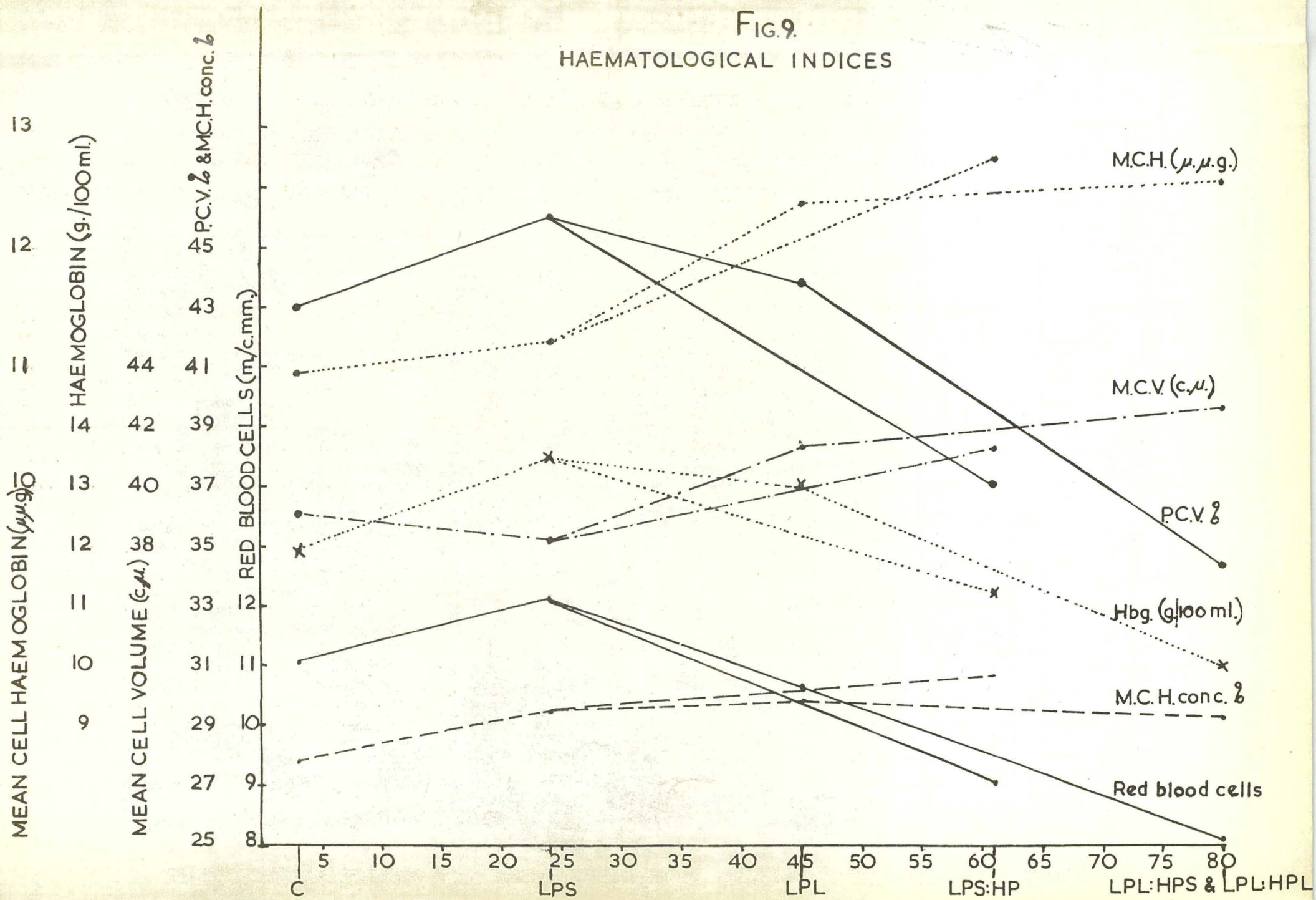
Definitions. P.C.V. - the volume occupied by the total red cells expressed as a % of the whole blood.

M.C.V. (mean cell volume) - the average volume of a single red cell in c.u.

M.C.H. (mean cell haemoglobin) - average weight in u.u.g. of Hbg in a single red blood cell.

M.C.H.C. (mean cell haemoglobin conc.) - the % of Hbg in 100 ml. of packed red cells as distinct from whole blood.

FIG. 9.  
HAEMATOLOGICAL INDICES



of re-alimentation further reducing the P.C.V. may have been largely due to the previous 24 days undernutrition, insufficient time being observed for the animals to recover physiologically. No significant differences were shown between the LFS:HP group and the control.

#### Haemoglobin (g./100 ml.)

The amount of haemoglobin was significantly increased by a short period of undernutrition. More prolonged undernutrition did not have any apparent effect. Re-alimentation appears to have reduced the haemoglobin, and there was no significant difference between the LFS:HP group and the control ewes. Haemoconcentration appears to have resulted from a short period of undernutrition, while re-alimentation has restored the haemoglobin to a normal level.

#### Mean Cell Volume (c.u.)

No effects attributable to the nutritional treatments imposed were evident on the volume of the erythrocytes.

#### Mean Cell Haemoglobin (u.u.g.)

Prolonged submaintenance feeding resulted in a highly significant increase in the mean weight of haemoglobin in a single red blood cell. Re-alimentation produced a highly significant increase in M.C.H., although no differences were evident between the LFS:HP and LPL groups.

#### Mean Cell Haemoglobin Concentration % (M.C.H. conc.%)

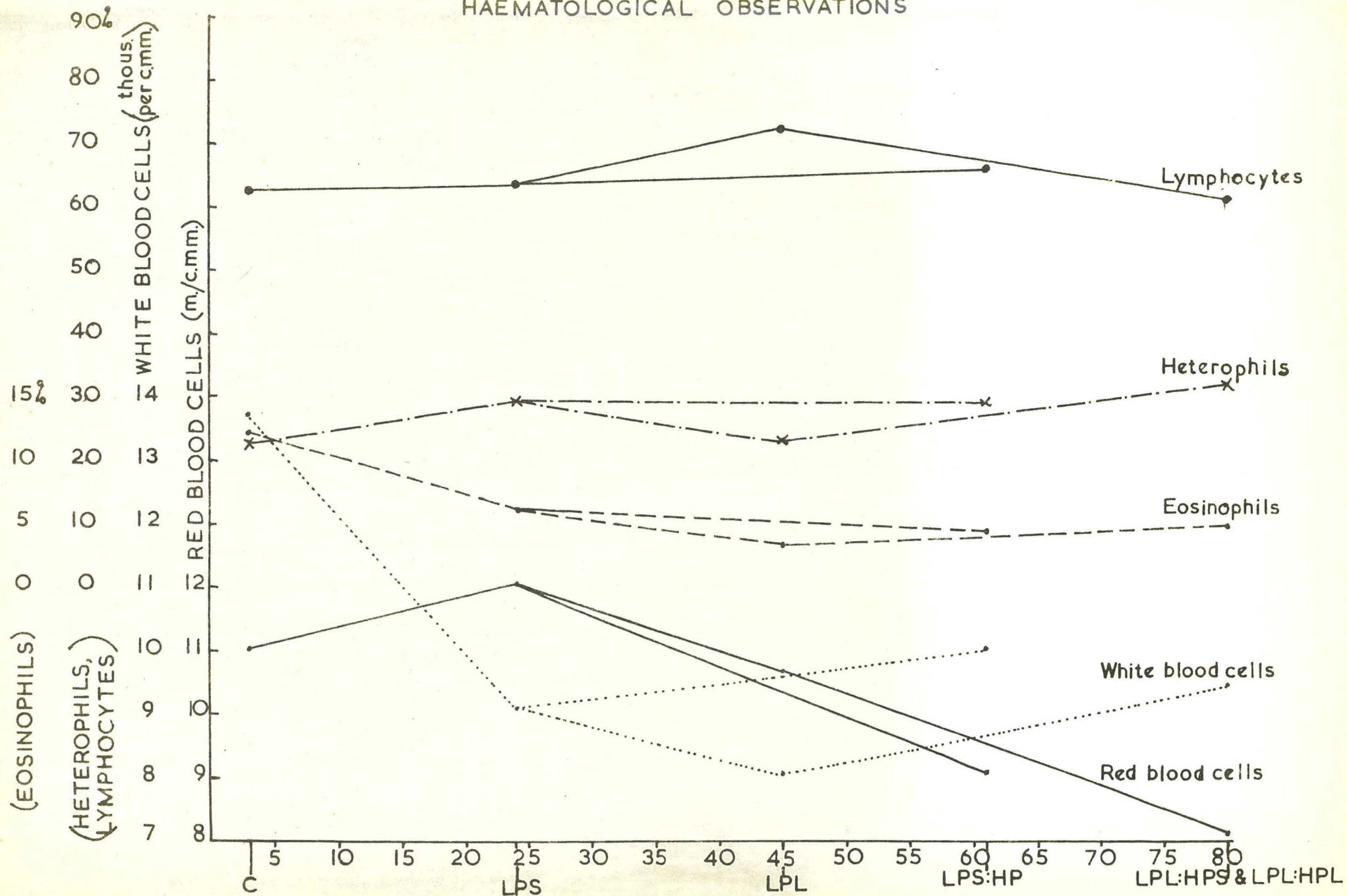
A prolonged period of undernutrition increased the M.C.H. conc.%, indicating haemoconcentration. Re-alimentation also appears to have resulted in some increase although there are no apparent differences between the LFS:HP and either LFS or LPL groups, the recovery group is highly significantly higher than the control group.

#### Total Leucocytes (per cmm.)

Statistical analysis showed that a short period of undernutrition resulted in a highly significant reduction in the total leucocyte count. Further undernutrition does not appear to have accentuated this effect. Re-alimentation after 24 days

FIG. 8.

HAEMATOLOGICAL OBSERVATIONS



submaintenance feeding did not significantly change the total leucocytes, the recovery group having a highly significantly lower count than the control ewes.

Heterophils, per cent. of Leucocytes

No treatment effects were shown on the percentage of heterophils.

Eosinophils, per cent. of Leucocytes

A highly significant reduction as a result of 21 days undernutrition was demonstrated. Further submaintenance feeding does not appear to have accentuated this effect. Re-alimentation did not increase the percentage of eosinophils, the recovery group being highly significantly lower than the controls. There was no apparent difference between the LPS:HP and LPL group.

Lymphocytes, per cent. of Leucocytes

No significant treatment effects on the percentage of lymphocytes were demonstrated. Considerable variability between animals is evident.

Consideration of the means of the re-alimentated group and the LPS:HPS and LPL:HPL groups, shown graphically in Figs. 8 and 9, indicate that changes in all components studied tend to be similar in the recovery group and the LPL:HPS and LPL:HPL groups.

### DISCUSSION

The effect of the plane of nutrition on blood composition is complicated by the disproportionate liveweight loss, and the presence of obvious disease symptoms, particularly of hydatid cysts in the lungs and liver, and to a lesser extent by facial eczema lesions observed at slaughter. This could have an important influence on the results.

It is of interest to note that the values of blood constituents reported all remained within the normal ranges given by Coffin (1953), except for the H.C.H. conc. per cent., and the lymphocyte percentage. This appears to indicate that changes in the cellular elements studied are unlikely to be of use as indicators of how a particular animal is reacting to the treatment imposed during an experiment.

No effects were evident on the total red blood cells, or haematocrit values. The erythrocytes and haematocrit values were however decreased by re-alimentation following a short period of undernutrition. This disagrees with the results of Meyer et al. (1955); and White et al. (1956), but Williams and Christian (1959) noted that the haematocrit was not influenced by a sudden reduction in food intake. A decrease in the amount of haemoglobin appeared to have resulted from re-alimentation. Prolonged undernutrition has increased M.C.H. and M.C.H. conc. per cent., which indicates that there was some haemoconcentration occurring. Further increases were noted on re-alimentation. In view of the recent findings of Turner and Hodgetts (1955) however, a bias could have been introduced into these values since the control group were less accustomed to handling than other groups. This reduces the reliability that can be placed on the results reported.

The marked decrease in the total leucocytes that appears to have occurred mainly during the initial period of undernutrition was reflected in a highly significant reduction in the eosinophils. This observation is supported by White et al. (1956) and Weeth et al. (1959). The eosinophil change was greater than could be attributed to the normal very large variability. The reduction in eosinophils is due to an increased secretion of ACTH, and this may have resulted in an increased rate of protein catabolism.

No treatment effects were shown on the lymphocytes, and this disagrees with the observation of Dougherty and White, (1947); and Merrill and Smith, (1954). The response of the lymphocytes to stress is however, less reliable than the decrease in eosinophils, (Forsham et al. 1948). This result together with the reduction in the weight of the thyroid gland as a result of undernutrition suggests an increased ACTH secretion and a decreased TSH secretion.

The hypothesis that the re-alimentated group had not recovered physiologically is supported by the fact that the LFS:HP group did not differ in total leucocytes, M.C.H., M.C.H. conc. per cent., eosinophils, or total leucocytes from the LPL group. Reference to Figs. 8 and 9 also show that in all observations made the mean of the LFS:HP group is very close to the mean of the LFS:HFS and LPL:HPL ewes.

Because of their greater energy requirements, grazing animals may show larger changes in blood constituents than penned animals.

SUMMARY

No treatment effects were demonstrated on M.C.V., the percentage of heterophils, or lymphocytes.

Undernutrition significantly increased Hbg., M.C.H., and M.C.H. conc. per cent., and reduced total leucocytes, and eosinophils. No effect of undernutrition was evident on total erythrocytes or haematocrit values.

Re-alimentation after 24 days undernutrition reduced the haematocrit, laemoglobin and total red blood cells, but increased M.C.H., and M.C.H. conc. per cent. No effect attributable to re-alimentation on total leucocytes, or eosinophils was demonstrated.

The recovery group (LES:HP) did not differ in total erythrocytes, M.C.H., M.C.H. conc. per cent., eosinophils, or total leucocytes from the prolonged undernutrition group.

PART III

MUSCLE-FIBRE DIAMETER

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## CHAPTER I

### REVIEW OF LITERATURE

Of the three main components of the carcass, muscle is the most important.

Striated muscle, attached to the skeleton, consists of large multinucleated called muscle fibres, varying in length from 1 to 41 mm., (Maximow and Bloom, 1944), and in thickness from 10 to 100  $\mu$ . or more. The muscle fibre is covered with a thin, structureless membrane, the sarcolemma, 150  $\text{\AA}$  to 250  $\text{\AA}$  thick, applied to the outside of the fibre (Bennett and Porter, 1953). The fibres consist of two parts - the protoplasmic mass or sarcoplasm, and cross-striated fibrils or myofibrils. The mass of the fibres consists of various albuminous substances of which the most important are myosin, and myogen, although carbohydrates, fats, lipoids, myoglobin, cytochrome, and other metabolic entities are present. Muscles are surrounded by fibrous tissue called the epimysium. This is continued into the muscle as the perimysium which breaks the muscle into bundles of fibres called fasciculi. Each fibre is isolated by fibrous tissue called endomysium.

The major factors which appear to influence muscle mass are species, age, breed, weight, sex, nutrition, and exercise.

Striated muscles originate from the mesoderm. A detailed account of the embryology of striated muscles is given by Boyd (1960). Growth of muscular tissue is generally accepted to occur by hyperplasia during the embryonic and early foetal stages, and essentially by the hypertrophy of existing fibres in late pre-natal, and throughout post-natal life. The increase in cell numbers is apparently due to differentiation of interstitial cells surrounding the primary muscle fibres, (Boyd, 1960). Schafer (1912) estimated that muscle fibres enlarged from 200 to 400 per cent. from mid-foetal life until birth, compared to a five-fold increase from birth to adult life. Adams *et al.* (1953) however, stated that previous attempts to measure the cross-sectional diameter of muscle fibres at different stages of foetal life had not yielded uniform results. Mearns (1947) concluded that growth of muscles occurs by hyperplasia in pre-natal life

and by hypertrophy in post-natal life. Joubert (1955) presents evidence to support this hypothesis. With the ovine foetus he found a marked change in the cross-sectional diameter of the fibres in foetuses 800 to 1200 g. in weight, but he emphasized that it cannot be inferred that cell division ceases abruptly at about this stage of foetal development. Subsequently Joubert (1956b) with various muscles from the sheep foetus concluded that the rate of development differed between muscles, and that the increase in the weight of muscles during the first two-thirds of pre-natal life was due primarily to hyperplasia, while during the last one-third of intra-uterine life hypertrophy of existing fibres contributes primarily to weight changes. The age of the foetuses was determined on the basis of the crown-rump length measurement, which may not be very reliable.

The influence of nutrition on the size and composition of muscular tissue has received a considerable amount of emphasis. Jackson (1945) with albino rats noted a marked reduction in muscular tissue with both undernutrition and starvation. Pomeroy (1944) with pigs, and Robinson (1948) and Wallace (1948) with sheep showed that muscular tissue is reduced with submaintenance feeding but less muscular tissue was lost than fatty tissue. Addis et al. (1936a) with fasting rats found that the muscle skin, and skeleton accounted for 62 per cent. of the total loss in weight. The liver protein was particularly sensitive to the level of nutrition, (Addis et al. 1936b). During fasting considerable amounts of body protein appear to be catabolized, (Kosterlitz and Campbell, 1945). Keys et al. (1950) maintain that in both man and animals marked atrophy of the skeletal musculature is a characteristic feature of undernutrition. They claim that the proportional loss of skeletal muscle is close to that of the body as a whole. The individual muscle fibres were reduced in size, but the nuclei appear to resist degeneration, with smooth and skeletal muscles responding in a similar manner. Kirton and Barton (1958a) found that a low plane of nutrition produced a loss of both protein and water in a constant ratio, this being reflected in a highly significant reduction in the weight of the fat-free carcass. Some of this loss may have resulted from a reduction of protein and water from adipose tissue. Ju and Nasset (1959) found that during undernutrition various body organs lose nitrogen at different

rates. On repletion, the rate of gain of nitrogen varied inversely with the rate of nitrogen loss during depletion of the organ. It is of interest to note that the concentrations of chloride and sodium in skeletal muscle of man, the pig, and the fowl, are reduced by about one-third if severe bleeding takes place before death, (Widdowson and Southgate, 1959). Summers and Fisher (1960) with chickens reported that on depletion there was a marked loss in carcass nitrogen, but on repletion there was a lag in the gain of carcass nitrogen. Fourman and McConkey (1958) and McConkey (1959) emphasized that a relative increase in extracellular fluid surrounding shrunken muscle cells was necessary if these cells, with a large surface area:volume ratio were to retain a surrounding film of interstitial fluid. Dickerson and McCance (1960) found that the amount of water, chlorine, and sodium was increased in the pectoral muscles of cockerels which had been underfed. They suggested that underfeeding may result in overhydration of cells. Extracellular fluid is withdrawn from the muscles into the circulation during bleeding (Widdowson and Southgate, 1959).

A high plane of nutrition increases the size of the muscle fibre, while a low plane reduces fibre size. Waters (1909) with beef carcasses reported that during fattening the average diameter of muscle fibres may increase from 20 to 50  $\mu$ , while conversely undernutrition reduces fibre size. Hammond (1932) reviewing his own and other workers results concluded that nutrition influences the size of the muscle fibres, with the latest maturing muscles being most affected. Robertson and Baker (1933) found that muscle fibres of full-fed yearling steers were appreciably greater in diameter than "rough-fed" steers. The fibres of "half-fed" animals occupied an intermediate position. This indicates a relationship between fibre size and muscle nutrition. The decrease in muscle fibre volume on inanition could indicate a loss of cytoplasmic material rather than that of a special protein reserve source, (Roche and Hoerner, 1933). McMeekan (1940-41) observed that pigs reared on a high nutritional plane until 16 weeks of age, had fibres approximately 50 per cent. larger than their low plane counterparts. This difference in fibre diameter was closely related to differences in the weights of the pig, and of the muscle. Joubert (1954) found that muscle fibres increased in

diameter with sheep on a supermaintenance diet, and decreased on a submaintenance diet. He found indications of a maximum limit of fibre size being attained with a high liveweight, suggesting that above a certain level additional gains in muscle weights are due to deposition of fat. Everitt (1960) has also used muscle fibres as an indication of muscle hypertrophy. The diameter of the component fibres of a muscle is known to vary along the length of the muscles. Meara (1947) found that a specimen from the middle of the muscle will represent the muscle fibre diameter of a particular muscle. Hammond (1932) considered that samples taken from a late maturing muscle would be most likely to show nutritional effects, these differing between muscles.

Exercise appears to result in increased vascularity and hypertrophy of muscles, (Eliot et al. 1943; Joubert, 1956).

Various methods have been used to measure muscle fibre diameters. These include; measurement with an ocular micrometer, (Buchthal and Lindhard, 1939); transverse drawings on graph paper, (Paff, 1930); teasing out free-hand shavings in a drop of dilute glycerine and measuring the cross-sectional diameter with an ocular micrometer, (Hammond and Appleton, 1932); and macerating with dilute nitric acid before mounting, (Robertson and Baker, 1933). The technique used by Hammond and Appleton (1932) has also been used by Meara (1947), McMeekan (1940-44), and Joubert (1956a). This method appears to result in least distortion of the fibre. Joubert (1956a) noted that muscles preserved in 10 per cent. formalin tend to become hardened. He found a decrease in the cross-sectional diameter of muscle fibres after overnight fixation, but no subsequent change in diameter over a period of six months.

McMeekan (1940-44) found the fibre diameter in the longissimus dorsi to be closely related to muscle weight. Joubert (1954) with sheep reported that fibre diameter was closely related to muscle weight ( $r = 0.965$ ), carcass weight ( $r = 0.945$ ), and liveweight ( $r = 0.962$ ), in Verge's (1939) submaintenance ewes. Except for the correlation between fibre diameter and carcass weight, the correlations were higher with the submaintenance sheep than the supermaintenance sheep. This indicates that an animal on a submaintenance diet draws on its muscle reserves. Joubert (1956a) also showed that fibre diameter was closely related to the width of the longissimus dorsi ( $r = 0.929$ ), and to the depth of

this muscle ( $r = 0.984$ ).

The size of the muscle fibres has been considered to be an important factor in the tenderness of meat. Sartorius and Child (1938) found that large bundles of fine fibres were more tender than smaller bundles of thick fibres. Hammond and Appleton (1932) found a correlation coefficient of 0.74 between tenderness and fibre diameter. Tenderness appears to decrease with age in cattle, (Hiner and Hankins, 1950). Hiner et al. (1953) reported that the relationship between resistance to shearing and fibre diameter varied from +0.34 to +0.75. Tenderness and fibre diameter seem to be more closely related in mature than immature animals. Brady (1936-37) noted that the size of the muscle fibre was related to the size of the bundle, the latter influencing the shear stress, texture, and tenderness scores of beef. The tenderness of meat is influenced by a number of factors other than muscle fibre diameter, Kiehl et al. (1958) has emphasized that tenderness is the most important factor influencing consumers' preference.

## CHAPTER II

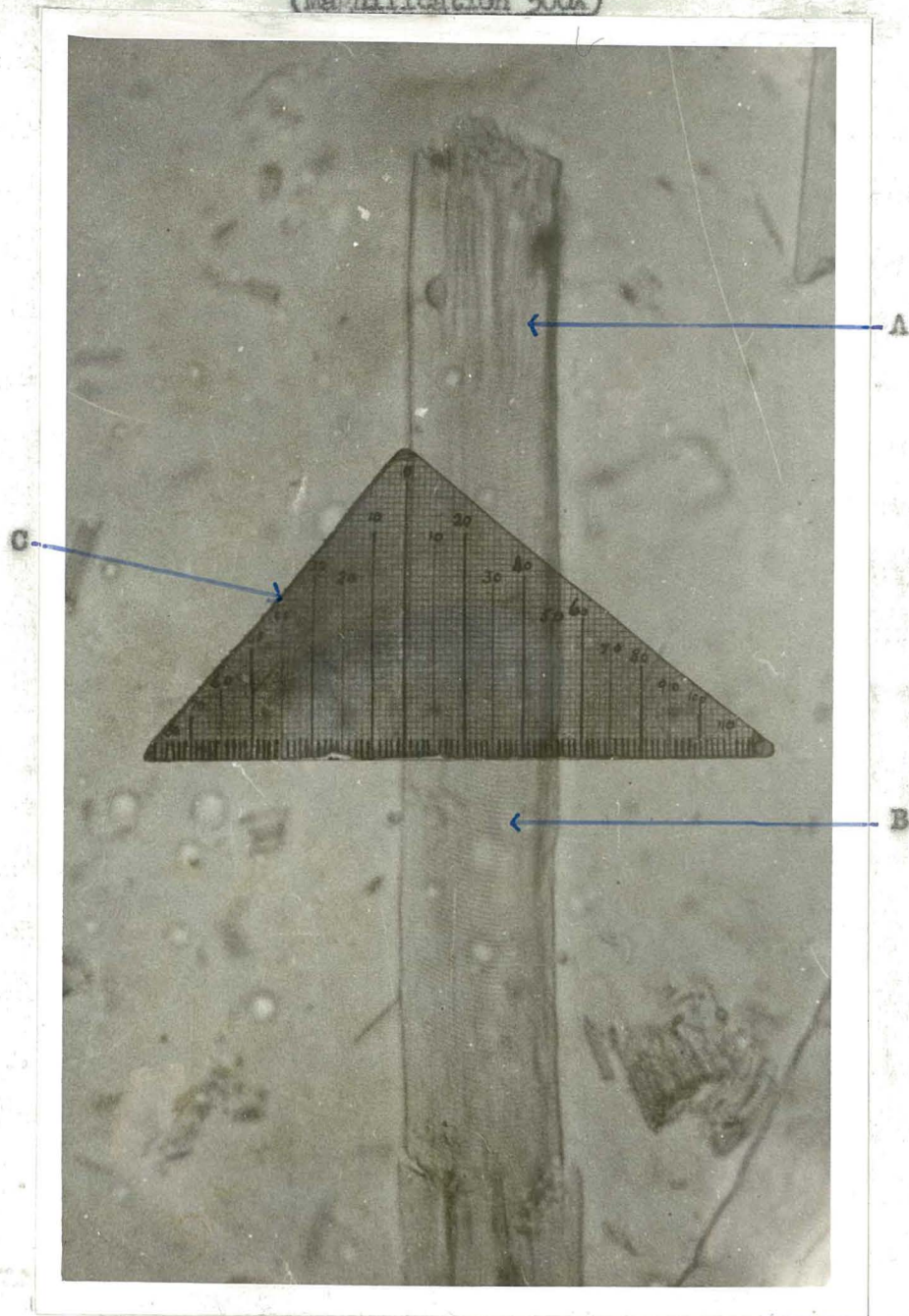
### MATERIALS AND METHODS

After weighing the longissimus dorsi from the half-loin, the muscle was cut along its length to give a 17 x 2 x 2 cm. sample. Three sections each 2 cm. long were cut from this sample, one square from the middle of the sample, and two a unit distance from each end. These were placed in a 10 per cent. formalin solution buffered with potassium phosphates to give a physiological solution with a pH of 7.2. It was thought that this solution would tend to reduce distortion of the samples. The solution was made up as follows: 100 ml. of 40 per cent. formaldehyde, 900 ml. of distilled water, 7.97 g. of potassium phosphate ( $KHPO_4$ ), and 3.97 g. of potassium dihydrogen phosphate ( $KH_2PO_4$ ). Samples were stored for approximately four months. Sections each about 10 mm. thick were cut with razor blades in a blade holder, and then teased out with needles in a drop of a 10 per cent. solution of glycerine. A standard concentration of glycerine solution is essential to ensure a constant refractive index of the suspension fluid. Fibres were readily separated. When fibres were teased out the watch glass was tilted, and a needle repeatedly drawn through the solution, so that single muscle fibres only remained in solution. A drop of this solution was then transferred to a hollow slide. A coverslip was not used. The diameter of the muscle fibres were then measured using a projection microscope, at a magnification of 500X. Rapid counting was facilitated by means of a counter, and distribution sheet. Counting always commenced in the left hand corner of the slide, the first 50 normal, clearly defined fibres being determined as the slide was moved from left to right and vice versa. The time taken to section, mount, and measure 50 fibres was approximately 25 minutes.

A muscle fibre is illustrated in Fig.10.

FIG. 10. Isolated striated muscle fibre from the longissimus dorsi  
muscle showing the method of measurement of the cross-section diameter.

(Magnification 500X)



A = sarcolemma

B = myofibrils

C = scale in microns (u.)

## CHAPTER III

### RESULTS

Before measuring all the muscle fibres an analysis of variance was carried out on the three samples (anterior, mid, posterior) for five sheep. Fifty fibres per sample were measured. The analysis of variance of these measurements is given in Table 32.

TABLE 32. Analysis of variance of muscle-fibre diameter for five sheep, and three positions

<u>Source of variation</u>	<u>s.s.</u>	<u>d.f.</u>	<u>M.S.</u>	<u>F.</u>	<u>F. Reqd.</u>	<u>Result</u>
Between sheep	11,084	4	2770	20.93	2.38 (3.34)	**
Between positions	356	2	178	1.34	3.00 (4.62)	n.s.
Interaction (S x P)	1,629	8	203.62	1.53	1.95 (2.53)	n.s.
Error	97,256	735	132.32			
Total	110,322	749				

This showed that there are highly significant differences between sheep, but the difference between positions was not significant. Isolation of the components of variance showed there was little to be gained by increasing the number of fibres counted per sample. The mid-sample adequately represented the three sections studied for each muscle.

#### Effect of plane of nutrition on muscle fibre diameter

In Table 33 are given the group means, standard deviations, and ranges of fibre-diameter in the longissimus dorsi muscle. The analysis of variance is presented in Table 34.

TABLE 33. Average muscle-fibre diameter, Group means, standard deviations, and ranges (u.)

<u>Group</u>	<u>Number</u>	<u>Mean</u>	<u>S.D.</u>	<u>Range</u>
G	8	51.7	14.0	18-96
LPS	8	49.7	11.9	18-82
LPS:HP	7	47.9	13.5	24-86
LPL	9	47.8	10.5	20-80

TABLE 34. Analysis of variance of muscle-fibre diameter measurements (u.)

<u>Source of variation</u>	<u>S.S.</u>	<u>d.f.</u>	<u>M.S.</u>	<u>F</u>	<u>F. Reqd</u> <u>(1%)</u>	<u>Result</u>
Between treatments	4038.5	3	1346.1	1.150	2.95 (4.57)	n.s.
Between sheep within Tr.	32753.6	28	1169.7	8.215	1.46 (1.69)	**
Within sheep (error)	223269.9	1568	142.3			
Total	260062.0	1599				

It is evident that no effects of the different treatments on muscle-fibre diameter were demonstrated, but considerable variation between sheep was present.

Relationships between average muscle-fibre diameter and some components of carcass composition and carcass measurements

A number of relationships are presented in Table 35. The mean muscle-fibre diameter of the longissimus dorsi is not related to liveweight or carcass composition. However, the mean muscle-fibre diameter is related to the dissected weight of the longissimus dorsi from the half-loin, weight of protein, and of water in this muscle. The correlation coefficient between the percentage of water in the l. dorsi and the mean muscle fibre diameter approaches significance. It is of interest to note that

**TABLE 35. Relationships between average muscle-fibre diameter and some components of carcass composition and carcass measurements for 32 ewes.**  
Independent variate = Mean muscle-fibre diameter (u.)

<u>Dependent variate</u>	<u>Correlation coefficient</u>	<u>Regression equation</u>	<u>Sy.x.</u>
Live weight (lb.) at slaughter	+ 0.2533 (n.s.)	$Y = 0.8126x + 91.03$	15.37
Carcass weight (lb.)	+ 0.2400 (n.s.)	$Y = 0.5145x + 44.15$	10.30
Fat-free carcass wt. (lb.)	+ 0.2912 (n.s.)	$Y = 0.2931x + 25.31$	4.76
Wt. carcass protein (lb.)	+ 0.2554 (n.s.)	$Y = 0.0622x + 5.01$	1.16
% carcass protein	- 0.0181 (n.s.)	$Y = 0.0052x + 11.93$	1.44
Wt. carcass water (lb.)	+ 0.2977 (n.s.)	$Y = 0.2237x + 17.92$	3.55
% carcass water	- 0.0206 (n.s.)	$Y = 0.0181x + 42.77$	4.36
Wt. <u>l. dorsi</u> (dissected) (g.)	+ 0.5264 (* *)	$Y = 8.7133x - 85.91$	49.35
Wt. <u>l. dorsi</u> protein (g.)	+ 0.5518 (* *)	$Y = 1.8626x - 26.12$	13.94
% <u>l. dorsi</u> protein	+ 0.3347 (n.s.)	$Y = 0.0642x + 15.91$	0.89
Wt. <u>l. dorsi</u> water (g.)	+ 0.4480 (*)	$Y = 4.9709x - 0.57$	49.12
% <u>l. dorsi</u> water	- 0.4010 (*)	$Y = 0.3325x + 88.22$	3.76
Width <u>l. dorsi</u> muscle (A) (mm.)	+ 0.3813 (*)	$Y = 0.4198x + 37.84$	5.04
Depth <u>l. dorsi</u> muscle (B) (mm.)	+ 0.2283 (n.s.)	$Y = 0.2131x + 21.65$	4.50

variation in muscle-fibre diameter appears to affect the width of the cross-section of this muscle (measurement A), while no effect is evident on muscle depth (measurement B). The relationships where significant are not sufficiently high for predictive purposes. Standard errors of estimate are also high. Changes in the main chemical components of the longissimus dorsi muscle therefore appear to influence the constituent fibre-diameter of this muscle.

### DISCUSSION

Although muscular tissue was reduced by undernutrition no treatment effects were demonstrated on the diameter of the muscle fibres. Several workers, (Waters, 1909; Hammond, 1932; Robertson and Baker, 1933; Roche and Hoerner, 1933; McMeekan, 1940-4 and Joubert, 1954) have shown that a low plane of nutrition reduces muscle fibre size while a high plane increases fibre size. The weight of the chemical fat, protein and water in the l. dorsi were reduced by undernutrition. This reduction in chemical constituents does not appear to have been great enough to show an effect on the cross-sectional diameter of the l. dorsi muscle. It should however, be emphasized that within a muscle a wide range exists in the cross-sectional diameter of the muscle fibres. As a consequence of this, large treatment differences would be necessary before statistically significant results were obtained. The amount of water, chlorine and sodium in the muscles could have been increased by undernutrition, (Dickerson and McCance, 1960), but the extracellular fluid may be withdrawn during bleeding, (Widdowson and Southgate, 1959).

The mean muscle fibre diameter was not related to liveweight, carcass weight, or carcass chemical composition. This is at variance with the result found by McMeekan (1940-41), and Joubert (1954). Muscle fibre diameter was however related to the l. dorsi muscle weight, which supports the observations of Joubert (1954) and McMeekan (1940-41). Changes in the chemical composition of the muscle, particularly protein appear to affect the constituent fibre-diameter of the l. dorsi muscle. The finding that muscle fibre-diameter was related to the width (measurement A), but not significantly related to the depth (measurement B) of the l. dorsi muscle, is at variance with the results of Joubert (1956a). There is no apparent reason for this discrepancy of results.

SUMMARY

No treatment effects were demonstrated on the mean muscle fibre-diameter of the longissimus dorsi muscle.

The mean muscle fibre-diameter was not significantly related to liveweight, carcass weight, the chemical composition of the carcass, the percentage of protein in the l. dorsi muscle, or the depth (measurement B), of this muscle.

Significant relationships were found between the mean muscle fibre-diameter and the weight of the dissected l. dorsi from the half-join, weight of protein, and the percentage and weight of water in this sample. A significant correlation was also found between fibre diameter and the width (measurement B) of the l. dorsi muscle.

The relationships where significant are not however sufficiently high for predictive purposes.

BIBLIOGRAPHY

- Abercrombie, M. (1957). The Biol. Action of Growth Substances. S.E.B. Symp. XI:235.
- Adams, R.D., Denny-Brown, D. and Pearson, C.M. (1953). Diseases of Muscle -  
(quoted by Joubert, 1955).
- Addis, T., Poo, L.J. and Lew, W. (1936a). J. Biol. Chem. 115:111.
- Addis, T., Poo, L.J. and Lew, W. (1936b). J. Biol. Chem. 115:117.
- Allcroft, W.M. (1944). J. Agric. Sci. 34:323.
- Annison, E.F. (1960). Aust. J. Agric. Res. 11:58.
- Andik, L., Bank, J. and Donhoffer, S.Z. (1957). Arch. exp. Path. Pharmacol. 231:55.
- Andik, L., Bank, J. and Donhoffer, S.Z. (1958). Acta. physiol. Hung. 13:125.  
(quoted by Wilson and Osbourn, 1960).
- Armstrong, D.G., Blaxter, K.L., Graham, N. McC. and Wainman, F.W. (1959).  
Animal Prod. 1:1.
- Arthaud, R.L., Schultze, A.B., Koch, R.M. and Arthaud, V.R. (1959). J. Animal Sci.  
18:314.
- Asdell, A.S. and Crowell, M.F. (1935). J. Nutr. 10:13.
- Asdell, S.A. (1949). J. Dairy Sci. 32:60.
- Babineau, L. and Page, E. (1955). Canad. J. Biochem. Physiol. 33:970.
- Baker, G.A. and Giulbert, H.R. (1942). J. Animal Sci. 1:293.
- Balch, C.C. (1949-50). Brit. J. Nutr. 4:361.
- Ball, Z.B., Barnes, R.H. and Visscher, M.B. (1947). Amer. J. Physiol. 150:511.
- Barrcroft, J., Kennedy, J.A., and Mason, M.F. (1939). J. Physiol. 95:159.
- Barker, S.B. (1951). Physiol. Rev. 31:205.
- Barton, R.A. (1957). Massey Agricultural College Sheepfarming Annual. p.61.
- Dean, H.W. (1948). J. Animal Sci. 7:50.
- Behnke, R.A. (1944-42). Harvey Lect. 37:198.
- Behnke, R.A. (1953). Annals N.Y. Acad. Sci. 56:1095.
- Benedict, F.C. and Ritzman, E.G. (1923). Carnegie Inst. Washington, Publ. no. 324.
- Benedict, F.C. and Ritzman, E.G. (1927). Carnegie Inst. Publ. no. 377.
- Bennett, H.S. and Porter, K.R. (1953). Amer. J. Anat. 93:61.
- Benson, G.K. and Cowie, A.T. (1957). J. Dairy Res. 24:252.
- Benton, D.A., Harper, A.E. and Elvehjem, C.A. (1955). J. Biol. Chem. 218:693

- Berg, B.N. and Simms, H.S. (1960a). *J.Nutr.* 71:242.
- Berg, B.N. and Simms, H.S. (1960b). *J.Nutr.* 71:255.
- Blaxter, K.L. (1944). *Proc.Brit.Soc.An.Prod.* p.85.
- Blaxter, K.L. (1948). *J.Agric.Sci.* 38:1.
- Blaxter, K.L., Reineke, E.P., Grampton, E.W. and Petersen, W.E. (1949).  
*J. Animal Sci.* 8:307.
- Blaxter, K.L. (1950). *Nutr.Abst. and Rev.* 20:1.
- Blaxter, K.L. and Wood, W.A. (1951). *Brit.J.Nutr.* 5:11.
- Blaxter, K.L. and Graham, N. McC. (1955). *Proc.Nutr.Soc.* 14:15.
- Blaxter, K.L., Graham, N. McC. and Wainman, F.W. (1955). *Proc.Nutr.Soc.* 14:4.  
(1956). *Brit.J.Nutr.* 10:69.
- 
- Bloor, W.R. (1924). *J.Biol.Chem.* 59:543.
- Bodwell, C.E., Everitt, G.G., Harrington, G. and Pomeroy, R.W. (1959). *An.Prod.* 1:51.
- Bodwell, C.E., Harrington, G., Pomeroy, R.W. and Williams, D.R. (1959).  
5th Meeting European Meat Res. Workers, Paris.
- Bomer, W., Kolb, J., Moll, E. and Schroder, J. (1960). *Klin. Wocheuscher.* 38:21.
- Boyd, J.D. (1960) in The Structure and Function of Muscle. Edit. by G.H. Bourne.  
Academic Press, London.
- Brady, D.E. (1936-37). *Proc.Amer.Soc.An.Prod.* p.246.
- Brobeck, J.R. (1946). *Physiol. Rev.* 26:544.
- Brobeck, J.R. (1960). Recent Progress in Hormone Research. p.439. Academic Press.
- Brody, S. (1945). Bioenergetics and Growth. Reinhold Publ. Corp., New York.
- Brown-Grant, K., Harris, G.W. and Reichlin, S. (1954). *J. Physiol.* 126:29.
- Brown-Grant, K. (1956). *J. Physiol.* 131:58.
- Brown-Grant, K., Harris, G.W. and Reichlin, S. (1957). *J. Physiol.* 136:364.
- Brown-Grant, K. and Pethes, G. (1960). *J. Physiol.* 151:40.
- Brozek, J. (1955). *Annals N. York Acad. Sci.* 63:491.
- Buchthal, F. and Lindhard, J. (1939). *K. Danske Vedenskab. Selskab Biol. Meddelel.*  
14:184. (quoted by Mearns, 1947).
- Butler, L.C. and Morgan, A.F. (1953). *Proc.Soc.Exp.Biol.Med.* 83:655.
- Butler, O.D., Garber, M.J. and Smith, R.L. (1956). *J. Animal Sci.* 15:891.

- Callow, E.H. (1947). *J.Agric.Sci.* 37:113.  
\_\_\_\_\_ (1948). *J.Agric.Sci.* 38:174.  
\_\_\_\_\_ (1958). *J.Agric.Sci.* 51:361.
- Cannon, W.B. (1929). *Physiol. Rev.* 9:399.
- Chatfield, C. (1926). U.S. Dept. Agric. Circ. 389.
- Child, C.M. (1920). *Biol. Bull. Wood's Hole.* 39:147. (quoted by Hammond, 1944).
- Clark, R.T. (1934). *Anat. Record.* 60:125.
- Clawson, A.J., Sheffy, B.E. and Reid, J.T. (1955). *J. Animal Sci.* 14:1122.
- Cochran, W.G. and Cox, G.M. (1950). Experimental Designs. Wiley & Sons, Inc. New York.
- Coekrem, F.F. (1960). Personal communication.
- Coffin, D.L. (1953). Manual of Vet. Clin. Pathology. Comstock Publ. Assoc.
- Comfort, A. (1954). *Biol. Rev.* 29:284.
- Cresswell, E. (1958). Ph.D. Thesis. M.A.C. Library.
- Darmady, E.M. and Davenport, S.G.T. (1958). Haematological Technique. Churchill Ltd. London.
- Deuel, H.J. (Jr.) (1955). The Lipids, Vol. 2. Interscience Publ., Inc., New York and London.
- Dewar, A.D. (1957). *J. Endocrin.* 15:246.
- Dickerson, E.E. and Gowen, J.W. (1947). *Science.* 105:496.
- Dickerson, G.E. (1954). Dynamics of Growth Processes. Edit. by E. J. Boell, Princeton Univ. Press.
- Dickerson, J.W.T. and McCance, R.A. (1960). *Brit. J. Nutr.* 14:331.
- Dougherty, T.F. and White, A. (1944). *Endocrin.* 35:1.  
\_\_\_\_\_ (1947). *J. Lab. Clin. Med.* 32:584.
- Dukes, H.H. (1955). The Physiology of Domestic Animals. Comstock Publ. Assoc. New York.
- Duncan, D.B. (1957). *Biometrics* 13:164.
- Edwards, F.R. and Holley, K.T. (1939). *Amer. Soc. An. Prod. Rec. Proc.* p. 376.
- Ellenberger, H.B., Newlander, J.A. and Jones, C.H. (1950). Univ. of Vermont and State Ag. College Bull. no. 558.
- Ellis, S. (1956). *Pharmacol. Rev.* 8:485. (quoted by Olson and Vester, 1960).
- Eliot, T.S., Wigginton, R.C., and Corbin, K.B. (1943). *Anat. Record.* 85:307.
- El-Sheikh, A.S., Halet, C.V., Pope, A.L. and Casida, L.E. (1955). *J. Animal Sci.* 14:91

- Erschoff, B.H. (1952). *Vitamins and Hormones* 10:79.
- Evans, H.M. and Bishop, K.S. (1922). *J. Metabol. Res.* 1:319.
- Everitt, G.C. (1960). *Proc. N.Z. Soc. An. Prod.* 20:58.
- Falconer, D.S. and Isaacsen, J.H. (1959). *J. Heredity* 50:290.
- Falconer, D.S. (1960). *Genetical Res.* 1:91.
- Fenton, P.F. and Dowling, M.T. (1953). *J. Nutr.* 42:319.
- Ferguson, N.L. (1954). *Brit. J. Nutr.* 8:269.
- Filmer, J.F. (1933). *Aust. Vet. J.* 9:163.
- Folley, S.J. and French, T.H. (1948). *Biochem. J.* 43: proc. IV.
- Fourman, P. and McConkey, B. (1958). *Lancet*, ii.554. (quoted by Dickerson and McGance, 1959).
- Forsham, P.H., Thorn, G.W., Prunty, F.T.G. and Hills, A.G. (1948).  
*J. Clin. Endocrinol.* 8:15.
- Franklin, M.C. (1952). *Aust. J. Agric. Res.* 3:168.
- Fraser, A.C. (1929-30). *Univ. Cambridge Inst. An. Pathology (Dir. Report)* p.114.
- French, M.H. and Ledger, H.P. (1957). *Emp. J. Exp. Agric.* 25:10.
- Frens, A.M. (1949). *Landbouwk. Tijdschr.* 61:916. (quoted by Kirton, 1957).
- Fullerton, H.W. (1956-57). *Proc. Nutr. Soc.* 15:66.
- Garn, S.M. and Brozek, J. (1956). *Science* 124:682.
- Garrett, W.N., Meyer, J.H. and Lofgreen, G.P. (1959). *J. Animal Sci.* 18:528.
- Garton, G.A. (1960). *Nutr. Abst. and Rev.* 30:1.
- Gerwing, J., Long, D.A. and Pitt-Rivers, R. (1958). *J. Physiol.* 144:229.
- Graham, N. McC., Wainman, F.W., Blaxter, K.L. and Armstrong, D.G. (1959).  
*J. Agric. Sci.* 52:13.
- Greenbaum, A.L. (1953). *Biochem. J.* 54:400.
- Guilbert, H.R. (1942). *J. Animal Sci.* 1:3.
- Halberg, F. and Bock, F. (1953). *Soc. Exp. Biol. Med. Proc.* 83:338.
- Hanersma, P.J. (1934). *Onderstepoort J. Vet. Sci.* 2:153.
- Hammond, J. (1932). Growth and Development of Mutton Qualities in the Sheep.  
Oliver and Boyd, Edinburgh.
- \_\_\_\_\_ (1940). *Chem. and Industry* 59:521.
- \_\_\_\_\_ (1944). *Proc. Nutr. Soc.* 2:8.

- Hankins, O.G. and Ellis, N.R. (1939). *Amer.Soc.An.Prod.* p.134.
- Hankins, O.G. and Titus, H.W. (1939). *Food and Life.* U.S. Dept.Agric. p.450.
- Hankins, O.G., Knopp, B. and Phillips, R.W. (1943). *J. Animal Sci.* 2:42.
- Hankins, O.G. and Howe, P.E. (1946). *U.S. Dept.Agric.Tech.Bull.* no.926.
- Hankins, O.G. (1947). *U.S. Dept.Agric.Bull.* no.944.
- \_\_\_\_\_ (1953). *Proc. 6th Recip.Meat.Conf.* p.131.
- Harkness, M.L.R., Harkness, R.D. and James, D.W. (1958). *J.Physiol.* 144:307.
- Harris, G.W. (1955). *Giba Foundation Colloquia on Endocrinol.* 8:531.
- Hart, E.B., Eloehjem, C.A. and Stenbock, H. (1930). *J.Nutr.* 2:277.
- Hausberger, F.X. (1958). *Diabetes* 7:211. (quoted by Wertheimer and Shafrir, 1960).
- Havel, R.J. and Goldfein, A. (1959). *J. Lipid Res.* 1:102.
- Hetherington, A.W. and Ranson, S.W. (1942). *Endocrinol.* 31:30.
- Hilditch, T.P. and Pedelty, W.H. (1941). *Biochem.J.* 35:932.
- Hilditch, T.P. (1956). *The Chemical Constituents of Natural Fats.* Chapman & Hall Ltd London.
- Hills, A.G., Forsham, P.H. and Finch, C.A. (1948). *Blood* 3:755. (quoted by Merrill and Smith, 1954).
- Hiner, R.L. and Hankins, O.G. (1950). *J. Animal Sci.* 9:341.
- Hiner, R.L., Hankins, O.G., Slocane, H.S., Fellers, C.R. and Anderson, E.E. (1953). *Food Research* 18:364.
- Hopper, T.H. (1944). *J. Agric. Res.* 68:239.
- Houssay, B.A. (1955). *Human Physiology.* McGraw-Hill Book Comp.
- Hughes, G.P. and Harker, K.W. (1950). *J.Agric.Sci.* 40:403.
- Hill, P. (1960). *J.Agric.Sci.* 55:317.
- Huseby, R.H., Ball, Z.B. and Visscher, M.B. (1945). *Cancer Res.* 5:40.
- Huseby, R.H., and Ball, Z.B. (1945). *Anat.Rec.* 92:135.
- Hutchinson, J.C.D. (1947). *Brit.J.Nutr.* 1:219.
- Jackson, C.M. (1913). *Amer.J.Anat.* 15:1.
- \_\_\_\_\_ (1915). *Amer.J.Anat.* 18:75.
- \_\_\_\_\_ (1917). *Amer.J.Anat.* 21:321. (quoted by Leathem, 1959).
- \_\_\_\_\_ (1932). *Amer.J.Anat.* 51:347.
- \_\_\_\_\_ (1937). *Anat.Rec.* 68:371.

- Jeffries, B.C. and Fern, J.T. (1957). *J. Dept. Agric. Sc. Aust.* 61:133.
- Josland, S.W. (1933). *N.Z. J. Sci. and Tech.* 14:304.
- Joubert, D.M. (1954). *J. Agric. Sci.* 45:164. (1955). *Proc. Brit. Soc. An. Prod.* p.49.
- \_\_\_\_\_ (1954). *Proc. Brit. Soc. An. Prod.* p.49.
- \_\_\_\_\_ (1955). *Nature (Lond.)* 175:936.
- \_\_\_\_\_ (1956a). *J. Agric. Sci.* 41:59.
- \_\_\_\_\_ (1956b). *J. Agric. Sci.* 47:382.
- Ju, J.S. and Nasset, E.S. (1959). *J. Nutr.* 68:633.
- Jungas, R.L. and Ball, E.G. (1960). *J. Biol. Chem.* 235:1894.
- Kelly, D.C., Guerrant, R.E. and Macdintosh, D.L. (1953). *Proc. 6th Recip. Meat Conf.* p.112.
- Kennedy, G.C. (1952). *Proc. Royal Soc. B.* 140:578.
- \_\_\_\_\_ (1957a). *Proc. Soc. Endocrinol.* p.XIX.
- \_\_\_\_\_ (1957b). *J. Endocrinol.* 16:9.
- Keys, A., Brozek, J., Henschel, A., Mickelsen, O. and Taylor, H.L. (1950). *The Biology of Human Starvation*. Vols. 1 and 2, University of Minnesota Press.
- Keys, A. and Brozek, J. (1953). *Physiol. Rev.* 33:245.
- Kiehl, E.R., Rhodes, V.J., Brady, D.E. and Naumann, H.D. (1958). *Mo. Res. Bull.* no.652.
- Kirton, A.H. (1957). Unpublished M. Agric. Sci. Thesis. M.A.C. Library.
- Kirton, A.H. and Barton, R.A. (1958a). *J. Agric. Sci.* 51:265.
- \_\_\_\_\_ (1958b). *J. Agric. Sci.* 51:282.
- Kirton, A.H., Ulyatt, M.J. and Barton, R.A. (1959). *Nature (Lond.)* 184:1724.
- Koch, R.M., Schleicher, E.W. and Arthaud, W.H. (1958). *J. Animal Sci.* 17:604.
- Kosterlitz, H.W. and Campbell, R.M. (1945). *Nutr. Abst. and Rev.* 15:1.
- Kraybill, H.F., Bitter, H.L. and Hankins, O.G. (1952). *J. Appl. Physiol.* 4:575.
- Kraybill, H.F., Hiner, R.L. and Farnworth, W.M. (1954). *J. Animal Sci.* 13:548.
- Kudo, T. (1921). *J. Exp. Zool.* 33:435. (quoted by Wallace, 1948).
- Lasley, E.L. and Kline, E.A. (1957). *J. Animal Sci.* 16:485.
- Leatham, J.H. (1959). *Recent Progress in Hormone Research*. Academic Press.
- Lepkovsky, S. (1948). *Adv. in Food. Res.* 1:105.
- Ierner, I.M. (1953). *Genetic Homeostasis*. Oliver and Boyd, Edinburgh.

- Ljunggren, H. (1957). *Acta Endocrinol.* 25: Suppl. 33.
- Ljunggren, H., Ildros, D. and Luft, R. (1959). *Brit. J. Nutr.* 13:485.
- Litwack, G., Harkes, L.V. and Elvehjem, G.A. (1952). *Proc. Soc. Exp. Biol. Med.* 81:444.
- Long, C.N.H., Katzin, B. and Fry, H.G. (1940). *Endocrinol.* 26:309.
- Long, C.N.H. (1957). *Proc. Soc. Endocrinol.* 14:35.
- Lush, J.L. (1926). *J. Agric. Res.* 32:727.
- Lush, J.L., Christensen, P.W., Wilson, G.W. and Black, W.H. (1928). *J. Agric. Res.* 36:554.
- Macomber, D. (1933). *New Engl. J. Med.* 209:1160.
- Marshall, F.H.A. and Hammond, J. (1945). *Bull. Minist. Agric.* no. 39.
- Mason, K.E. (1933). *Amer. J. Anat.* 52:153.
- Maximow, A.A. and Bloom, W. (1950). *A Textbook of Histology.* Saunders Comp. Philadelphia and London.
- Mayer, J. (1953). *Physiol. Rev.* 33:472.
- \_\_\_\_\_ (1955). *Nutr. Abstr. and Rev.* 25:871.
- Maynard, L.A. and Loosli, J.K. (1956). *Animal Nutrition.* McGraw-Hill Publ. New York.
- McCance, R.A. (1960). *Brit. J. Nutr.* 14:59.
- McConkey, B. (1959). *Clin. Sci.* 18:95.
- McDonald, I.W. (1959). *Aust. J. Sci.* 22:235.
- McMeekan, C.P. (1940). *J. Agric. Sci.* 30:276, 387, 511.
- \_\_\_\_\_ (1944). *J. Agric. Sci.* 31:1.
- Mears, P.J. (1947). *Onderstepoort J. Vet. Sci.* 21:329.
- Mehrotra, P.N., Mullick, D.N. and Kehar, N.S. (1954). *J. Animal Sci.* 13:1026.
- Mendes, G.B. and Waterlow, J.C. (1958). *Brit. J. Nutr.* 12:74.
- Merritt, P. (1954). *M.A.C. Sheepfarming Annual* p.97.
- Merrill, W.G. and Smith, V.R. (1954). *J. Dairy Sci.* 37:546.
- Meyer, J.H., Weir, W.C. and Smith, J.D. (1955). *J. Animal Sci.* 14:160.
- Meyer, J.H., Lucker, C.E. and Smith, J.D. (1956). *J. Nutr.* 60:121.
- Meyer, J.H. and Weir, W.C. (1960). *Ann. Rep. California's Contributing Project.*
- Mitchell, H.H., Karmilade, W.G. and Hamilton, T.S. (1928). *Univ. Ill. Agric. Exp. Sta., Bull.* no. 317.
- Mitchell, H.H. (1944). *Ind. Eng. Chem. (Anal. ed.)* 16:696.

- Moulton, C.R., Trowbridge, P.F. and Haigh, L.D. (1921). Univ. Mo. Agric. Exp. Sta., Res. Bull. no. 43.
- 
- (1922). Univ. Mo. Agric. Exp. Res. Bull. no. 54 and 55.
- 
- (1923). Univ. Mo. Agric. Exp. Sta., Res. Bull. no. 61.
- Moustgaard, J. (1959). Reproduction in Domestic Animals. Academic Press, New York.
- Mullick, D.N. and Kehar, N.D. (1954). J. Animal Sci. 13:1027.
- Munford, R.E. and Kirton, A.H. (1958). N.Z.J. Agric. Res. 1:83.
- Murray, A.J. (1922). J. Agric. Sci. 12:103.
- Nevens, W.B. (1928). J. Agric. Res. 36:777.
- Olson, R.E. and Vester, J.W. (1960). Physiol. Rev. 40:677.
- Osbourn, D.F. and Wilson, P.N. (1960). J. Agric. Sci. 54:278.
- Pace, N. and Rathbun, E.N. (1945). J. Biol. Chem. 158:685.
- Palsson, H. and Verges, J.B. (1952). J. Agric. Sci. 42:1,93.
- Palsson, H. (1955). Progress in the Physiology of Farm Animals. Vol. 2. Butterworths London.
- Parry, H.B. (1960). Nature, (Lond.), 177:288.
- Paschkis, K.E. (1958). Cancer Res. 18:984.
- Passmore, R., Strong, J.H. and Ritchie, F.J. (1958). Brit. J. Nutr. 12:113.
- Patterson, R.E. (1947). J. Animal Sci. 6:237.
- Pennington, R.J. (1952). Biochem. J. 51:251.
- Phillips, G.D. (1960). J. Agric. Sci. 54:231.
- Pipes, G.W., Crossie, J.H. and Turner, G.W. (1960). Proc. Soc. Exp. Biol. Med. 104:491.
- Pitta, G.C. (1951). Fed. Proc. 10:105.
- Pomeroy, R.W. (1944). J. Agric. Sci. 31:50.
- Pratt, G.W.M. and McCance, R.A. (1960). Brit. J. Nutr. 14:75.
- Prosser, C.L. (1955). Biol. Rev. 30:229.
- Purser, A.F. and Roberts, C. (1959). An. Prod. 1:189.
- Quin, J.I. (1943). Onderstepoort J. Vet. Sci. 18:91.
- Randle, R.J. (1957). S.E.B. Symposia XI : p.183.
- Rennie, I. (1956-57). Nutr. Soc. Proc. 15 and 16 : p.61.

- Raymond, W.F., Harris, C.E. and Kemp, C.D. (1954). *J. Brit. Grassl. Soc.* 9:209.
- Reeve, E.B. (1948). *Nutr. Abst. and Rev.* 17:811.
- Reid, J.T., Trimberger, G.V., Asdell, S.A., Turk, K.L. and Smith, S.E. (1951).  
*J. Dairy Sci.* 34:510.
- Reid, J.T., Wellington, G.H. and Dunn, H.O. (1955). *J. Dairy Sci.* 38:1344.
- Reid, J.T. (1956). *Proc. Cornell Nutr. Conf. Feed Manuf.*
- Riney, T. (1955). *N. Z. J. Sci. and Tech. (B)* 36:429.
- Ritzman, E.G. and Benedict, F.C. (1938). *Nutritional Physiology of the Adult Ruminant*  
Carnegie Inst., Washington.
- Robertson, D.D. and Eaker, D.D. (1933). *Univ. Mo. Res. Bull.* no. 200.
- Robinson, P. (1948). *J. Agric. Res.* 38:345.
- Robinson, T.J., Binet, F.E. and Doig, A.G. (1956). *Aust. J. Agric. Res.* 7:345.
- Roche, A. and Hoerner, G. (1933). *C.R. Soc. Biol.* 144:1027. (quoted by Kosterlitz and  
Campbell, 1945).
- Sartorius, M. and Child, A.M. (1938). *Tech. Bull. Mo. Agric. Exp. Sta.* no. 131.
- Sayers, G. (1950). *Physiol. Rev.* 30:244.
- \_\_\_\_\_ (1957). *Ciba Found. Coll. Endocrinol.* 11:138.
- Schalk, A.F. and Amadon, R.S. (1928). *Bull. N. Dakota Agric. Exp. Sta.* no. 216.
- Scheer, B.T., Soule, D.F., Fields, M. and Deuel, H.J. (1947). *J. Nutr.* 33:583.
- Schoenheimer, R. and Rittenberg, D. (1937). *J. Biol. Chem.* 120:155.
- Schultze, M.O. (1955). *J. Nutr.* 56:25.
- Schultz, A.B. (1957). *J. Dairy Sci.* 40:672.
- \_\_\_\_\_ (1959). *J. Dairy Sci.* 42:166.
- Schweigert, B.S. (1955). *Amer. Meat Inst. Found. Circ.* no. 19.
- \_\_\_\_\_ (1957). *Amer. Meat Inst. Found. Sp. Report* no. 1.
- Selye, H. (1948). *Textbook of Endocrinology.* Acta Endocrinol., Canada.
- Selye, H. and Hauser, G. (1955-56). *5th Ann. Report on Stress.* M.D. Publ. Inc.  
New York.
- Shafer, E.A. (1912). *Textbook of Microscopic Anatomy.* Longmans, Green; London.
- Shafir, E., Sussman, K.E. and Steinberg, D. (1959). *J. Lipid Res.* 1:109.
- Shapiro, B. and Weitheimer, E. (1956). *Metabolism* 5:79.
- Shaw, J.C., Ensor, W.L., Tellechea, A.F. and Lee, S.D. (1960). *J. Nutr.* 71:203.

- Shorland, F.B., De la Mare, P.B.D., Sorrell, D.M.P. and Barnicoat, G.R. (1947).  
N.Z.J.Sci.Tech. (A) 29:76.
- Shorland, F.B. (1955). Progress in Chemistry Fats and Other Lipids. p.275.  
Pergamon Press Ltd., London.
- \_\_\_\_\_ (1958). Proc.N.Z.Soc.An.Prod. 18:99.
- Smith-Pilling, S.H. and Barton, R.A. (1954). N.Z.J. Agric. 88:98.
- Snedecor, G.W. (1959). Statistical Methods. Iowa State College Press.
- Sobel, H. and Marmorston, J. (1958). Recent Progr. Hormone Res. 14:457.
- Solomon, D.H. and Dowling, J.T. (1960). Ann.Rev.Physiol. 22:615.
- Speirs, R.S. and Meyer, R.K. (1949). Endocrinol. 45:403.
- \_\_\_\_\_ (1951). Endocrinol. 48:316.
- Steinberg, V. (1947). Beret. Forsgslab. Copenhagen no.227. (quoted by Moustgaard, 19)
- Stephens, D.J. (1944). J.Clin.Endocrinol. 1:257.
- Stewart, W.L. (1931). Vet.Record. 11:1033.
- Stewart, C.A. (1919). Amer.J.Physiol. 48:67.
- Summers, J.D. and Fisher, H. (1960). J.Nutr. 72:153.
- Taylor, J.C., Alder, F.E. and Rudman, J.R. (1957). Nature (Lond.) 179:197.
- Taylor, J.C. (1959). Nature (Lond.) 184:2021.
- Terroine, E.F., Brenckmann, E. and Feuerbach, A. (1922-23). Arch.int.Physiol. 20:466.
- Terroine, E.F., Feuerbach, A. and Brenckmann, E. (1924). Arch.int.Physiol. 22:233.  
(quoted by Widdowson et al., 1960).
- Thomas, B.H., Outhbertson, C.C. and Beard, F. (1934). Amer.Soc.An.Prod.Rec.Proc. p.193.
- Thom, G.W., Jenkins, D. and Laidlaw, J.C. (1953). Rec.Progr.Hormone Res. 8:171.
- Trowbridge, P.F., Moulton, G.R. and Haigh, L.D. (1918). Univ.Mo.Res.Bull. no.29.
- Turner, A.W. and Hodgetts, V.E. (1959). Aust.J.Exp.Biol.Med.Sci. 37:399.
- \_\_\_\_\_ (1960). Aust.J.Exp.Biol.Med.Sci. 38:79.
- Ulyatt, M.J. (1960). Unpublished M.Agric.Sci. Thesis. M.A.C. Library.
- Underwood, E.J., Harvey, R.J. and Beech, A.A. (1939). Aust.J.Exp.Biol.Med.Sci. 17:202.
- Verges, J.B. (1939a). Suffolk Sheep Soc. Year Book. Ipswich. (quoted by Falson, 19)
- \_\_\_\_\_ (1939b). Proc. 4th Int.Cong.An.Breeding. Zurich.

- Walker, D.E. and McLeskan, C.P. (1944). *N.Z.J.Sci.Tech.(A)* 26:51.
- Wallace, L.R. (1948). *J.Agric.Sci.* 38:93,243,367.
- \_\_\_\_\_ (1960). *N.Z.J.Agric.* 101:9.
- Waters, H.J. (1909). *Proc.Ann.Meat Soc.Prom.Agric. Portland, U.S.A.* p.70.  
(quoted by Joubert, 1954).
- Weeth, H.J., Torell, G.R. and Cassard, D.W. (1959). *J. Animal Sci.* 18:694.
- Weitze, M. (1940). *Store Nordeske Videnskabsboghandel Kbenhavn.* (quoted by Dickerson, 1954).
- Werner, S.C. (1939). *Proc.Soc.Exp.Biol.Med.* 41:101.
- Wertheimer, E. and Shapiro, B. (1948). *Physiol.Rev.* 28:457.
- Wertheimer, E. and Shafrix, E. (1960). Recent Advances in Hormone Research.  
Academic Press, New York and London.
- White, R.R., Christian, K.R. and Williams, V.J. (1956). *N.Z.J.Sci.Tech.(A)* 38:440.
- Whitman, J.V. and Whatley, J.A. (1953). *J. Animal Sci.* 12:591.
- Whitman, J.V., Loggins, P.F., Chambers, D., Pope, L.S. and Stephens, D.F. (1954).  
*J. Animal Sci.* 13:832.
- Widdowson, E.M. and McCance, R.A. (1956). *Brit.J.Nutr.* 10:363.
- Widdowson, E.M. and Southgate, D.A.T. (1959). *Biochem.J.* 72:200.
- Widdowson, E.M., Dickerson, J.W.T. and McCance, R.A. (1960). *Brit.J.Nutr.* 14:457.
- Williams, V.J. and Christian, K.R. (1959). *N.Z.J.Agric.Res.* 2:677.
- Willits, C.O. (1951). *Anal.Chem.* 23:1058.
- Wilson, P.N. (1952). *J.Agric.Sci.* 42:369.
- \_\_\_\_\_ (1954a). *J.Agric.Sci.* 44:67.
- \_\_\_\_\_ (1954b). *J.Agric.Sci.* 45:110.
- \_\_\_\_\_ (1958a). *J.Agric.Sci.* 50:198.
- \_\_\_\_\_ (1958b). *J.Agric.Sci.* 51:4.
- \_\_\_\_\_ (1960). *J.Agric.Sci.* 54:105.
- Wilson, P.N. and Osbourn, D.F. (1960). *Biol.Rev.* 35:324.
- Winchester, G.F. and Morris, M.J. (1956). *J. Animal Sci.* 15:722.
- Wynne, K. (1954). *Aust.J.Sci.* 17:36.
- Yadkin, J. (1958). *Progress in Cardiovascular Diseases*, 1:116.
- Zimmer, R., Weill, J. and Dubois, H. (1944). *New Engl.J.Med.* 230:303.  
(quoted by Leatham, 1959).

A P P E N D I X

A copy of all the data used in this thesis  
has been lodged with the Sheep Husbandry Department,  
Massey Agricultural College.

LIST OF APPENDICES

- I. Analyses of variance of liveweight, linear measurements, carcass weight, and some of its components.
- II. Analyses of variance of dissectible components of the half-loin, and 9-10-11 rib-cut.
- III. Analyses of variance of the chemical components of the l. dorsi, leg, and half-loin muscles.
- IV. Analyses of variance of chemical components of the 9-10-11 rib-cut muscle, and the subcutaneous fat of the leg, half-loin, and 9-10-11 rib-cut.
- V. Analyses of variance of the chemical components of some dissection tissues.
- VI. Analyses of variance of some non-carcass components of liveweight.
- VII. Analyses of variance of haematological components of blood.

The Conventional Abbreviations used in the text are as follows:

n.s.	results not statistically significant
s.	results statistically significant at the 5% level
s.s.	results statistically significant at the 1% level
F.	the variance ratio for specified conditions
$\bar{x}$	mean value
b	regression coefficient
d.f.	degrees of freedom
S.D.	standard deviations
S.S.	sums of squares
r	correlation coefficient
S <sub>y,x</sub>	sample standard error of Y estimated from X

APPENDIX I. Analyses of variance of treatment effects on liveweight  
linear measurements, carcass weight and some of its components.

Mean squares and significance levels are presented

<u>Item</u>	<u>Source of variation</u>		<u>Significance</u>
	<u>Plane of nutrition</u>	<u>Within subclass</u>	
Liveweight (lb.)	1381.1	118.7	s.s.
Width of thorax (W.th. cms.)	16.49	4.01	s.
Width of forequarter (W.F. cm.)	24.72	3.38	s.s.
Leg length (F. cm.)	0.85	2.74	n.s.
Width of gigots (G. cm.)	4.09	0.91	s.
Measurement A. (mm.)	46.33	26.89	n.s.
Measurement B. (mm.)	89.66	13.28	s.s.
Measurement C. (mm.)	42.33	23.28	n.s.
Measurement D. (mm.)	29.90	20.50	n.s.
Measurement Y. (mm.)	33.80	8.67	s.
Measurement X. (mm.)	18.20	27.71	n.s.
Frozen carcass weight (lb.)	433.0	74.4	s.s.
Carcass % chemical fat	41.89	39.19	n.s.
Carcass weight chemical fat (lb.)	131.27	52.80	n.s.
Fat-free carcass wt. (lb.)	118.66	13.89	s.s.
Water as % fat-free carcass	3.32	3.53	n.s.
Carcass % protein	4.496	1.725	n.s.
Carcass wt. protein (lb.)	9.873	0.504	s.s.
Carcass % water	11.803	19.114	n.s.
Carcass wt. water (lb.)	60.706	8.341	s.s.
Leg wt. (g.)	888331	124993	s.s.
Leg dissectible muscle (g.)	645473	52685	s.s.
Leg dissectible fat (g.)	69836	25676	n.s.
Leg dissectible bone (g.)	1000	1437	n.s.
Leg intermuscular fat (g.)	10124	2870	s.
Leg dissected subcutaneous fat (g.)	19927	14549	n.s.
Dissected mammary gland (g.)	2642.6	831.2	s.
Degrees of freedom	3	28	

APPENDIX II. Analyses of variance of treatment effects on dissectible components of the half-loin, and 9-10-11 rib-cut (g.).  
Mean squares and significance levels are presented

<u>Item</u>	<u>Source of variation</u>		
	<u>Plane of nutrition</u>	<u>Within subclass</u>	<u>Significance</u>
Half-loin weight (g.)	940173.6	273805.1	s.
Half-loin dissectible muscle (g.)	169481.3	12606.6	s.s.
Half-loin dissectible fat (g.)	397249.6	229692.4	n.s.
General muscle of half-loin (g.)	35531.3	2565.6	s.s.
Dissected skin muscle of half-loin (g.)	84.31	131.4	n.s.
Dissected psoas muscle of half-loin (g.)	1385.3	246.8	s.s.
Dissected subcutaneous fat of half-loin (g.)	161853.6	82884.7	n.s.
Dissected intermuscular fat of half-loin (g.)	31565.3	7808.6	s.
Dissected perirenal fat of half-loin (g.)	46087.0	31004.7	n.s.
Rib-cut wt. (g.)	241218.0	59122.1	s.
Dissectible muscle of rib-cut (g.)	35349.3	1834.7	s.s.
Rib-cut dissected skin muscle (g.)	105.6	23.2	s.s.
Rib-cut dissected general muscle (g.)	32249.6	1575.2	s.s.
Total dissected fat of rib-cut (g.)	98866.3	60373.3	n.s.
Subcutaneous fat of rib-cut (g.)	52042.0	40671.3	n.s.
Intermuscular fat (dissected) of rib-cut (g.)	9704.3	3407.1	n.s.
Degrees of freedom	3	28	

APPENDIX III. Analyses of variance of treatment effects on the chemical components of the l. dorsi, leg, and half-loin muscles.

Mean squares and significance levels are presented

<u>Item</u>	<u>Source of variation</u>		
	<u>Plane of nutrition</u>	<u>Within subclass</u>	<u>Significance</u>
Dissectible <u>l. dorsi</u> of half-loin (g.)	33391.3	3620.7	s.s.
% water in <u>l. dorsi</u>	55.666	12.107	s.s.
Wt. water in <u>l. dorsi</u> (g.)	10763.0	2081.39	s.s.
% protein in <u>l. dorsi</u>	3.186	0.625	s.s.
Wt. protein in <u>l. dorsi</u> (g.)	1456.0	143.39	s.s.
% fat in <u>l. dorsi</u>	20.590	3.448	s.s.
Wt. fat in <u>l. dorsi</u> (g.)	835.33	73.642	s.s.
% fat in leg muscle	5.213	2.963	n.s.
Wt. fat in leg muscle (g.)	10121.0	1380.2	s.s.
% protein in leg muscle	2.493	0.577	s.
Wt. protein in leg muscle (g.)	29767.0	2211.3	s.s.
% water in leg muscle	4.393	6.751	n.s.
Wt. water in leg muscle (g.)	296373.6	28613.4	s.s.
% fat in half-loin muscle	7.810	4.698	n.s.
Wt. fat in half-loin muscle (g.)	5155.2	665.31	s.s.
% protein in half-loin muscle	0.146	0.448	n.s.
Wt. protein in half-loin muscle (g.)	5904.0	379.13	s.s.
% water in half-loin muscle	11.170	4.036	n.s.
Wt. water in half-loin muscle	66513.6	5711.1	s.s.
Degrees of freedom	3	28	

APPENDIX IV. Analyses of variance of treatment effects on the chemical components of the 9-10-11 rib-cut muscle, and the subcutaneous fat of the leg, half-loin, and 9-10-11 rib-cut.

Mean squares and significance levels are presented

<u>Item</u>	<u>Source of variation</u>		<u>Significance</u>
	<u>Plane of nutrition</u>	<u>Within subclass</u>	
% fat in muscle of rib-cut	34.90	8.166	s.
Wt. fat in rib-cut muscle (g.)	2506.12	142.58	s.s.
% protein in rib-cut muscle	0.970	0.729	n.s.
Wt. protein in rib-cut muscle (g.)	1009.0	66.92	s.s.
% water in rib-cut muscle	26.796	6.446	s.
Wt. water in rib-cut muscle (g.)	11484.1	902.03	s.s.
% fat in leg subcutaneous fat	46.236	49.438	n.s.
Wt. fat in leg subcutaneous fat (g.)	19232.0	10921.6	n.s.
% fat in half-loin subcutaneous fat	10.480	14.742	n.s.
Wt. fat in half-loin subcutaneous fat (g.)	126038.6	62531.2	n.s.
% fat in rib-cut subcutaneous fat	8.383	9.051	n.s.
Wt. fat in rib-cut subcutaneous fat (g.)	43946.4	35353.4	n.s.
% water in leg subcutaneous fat	15.000	31.928	n.s.
Wt. water in leg subcutaneous fat (g.)	266.66	267.14	n.s.
% water in half-loin subcutaneous fat	7.000	8.857	n.s.
Wt. water in half-loin subcutaneous fat (g.)	2776.6	2021.9	n.s.
% water in rib-cut subcutaneous fat	1.610	6.003	n.s.
Wt. water in rib-cut subcutaneous fat (g.)	415.14	227.69	n.s.
% protein in leg subcutaneous fat	7.330	2.242	s.
Wt. protein in leg subcutaneous fat (g.)	57.666	27.642	n.s.
% protein in half-loin subcutaneous fat	2.213	0.858	n.s.
Wt. protein in half-loin subcutaneous fat (g.)	16.333	67.500	n.s.
% protein in rib-cut subcutaneous fat	4.026	0.598	s.s.
Wt. protein in rib-cut subcutaneous fat (g.)	52.713	8.033	s.s.
Degrees of freedom	3	28	

APPENDIX V. Analyses of variance of treatment effects on the  
chemical components of some dissection tissues.

Mean squares and significance levels are presented

<u>Item</u>	<u>Source of variation</u>		<u>Significance</u>
	<u>Plane of nutrition</u>	<u>Within subclass</u>	
% fat in leg intermuscular fat	409.33	36.891	S.S.
Wt. fat in leg intermuscular fat (g.)	2743.0	1228.5	n.s.
% fat in half-loin intermuscular fat	353.63	43.69	S.S.
Wt. fat in half-loin intermuscular fat (g.)	11885.3	4711.6	n.s.
% fat in rib-cut intermuscular fat	100.98	32.107	S.
Wt. fat in rib-cut intermuscular fat (g.)	6878.20	2100.56	S.
% water in leg intermuscular fat	290.513	24.007	S.S.
Wt. water in leg intermuscular fat (g.)	7012.33	686.89	S.S.
% water in half-loin intermuscular fat	255.19	17.056	S.S.
Wt. water in half-loin intermuscular fat (g.)	5562.06	497.06	S.S.
% water in rib-cut intermuscular fat	48.340	19.276	n.s.
Wt. water in rib-cut intermuscular fat (g.)	598.04	280.76	n.s.
Perirenal % chemical fat	10.923	5.558	n.s.
Perirenal chemical fat wt. (g.)	42548.6	28461.0	n.s.
Perirenal % water	4.270	4.109	n.s.
Perirenal water wt. (g.)	229.46	111.84	n.s.
Degrees of freedom	3	28	

APPENDIX VI. Analyses of variance of the weights of some non-carcase components of liveweight.

Mean squares and significance levels are presented

<u>Item</u>	<u>Source of variation</u>		<u>Significance</u>
	<u>Plane of nutrition</u>	<u>Within subclass</u>	
Gastrointestinal contents (g.)	4472134	2146532	n.s.
Gastric contents (g.)	3619810	1453505	n.s.
Intestinal contents (g.)	77113	346924	n.s.
Empty gastrointestinal tract (g.)	2579335	113289	s.s.
Gastric tract empty (g.)	646735	36894	s.s.
Intestinal tract empty (g.)	642975	31283	s.s.
Heart wt. (g.)	3973.6	1175.1	s.
Heart % chemical fat	23.000	13.821	n.s.
Heart wt. chemical fat (g.)	232.00	148.71	n.s.
Heart % protein	9.333	1.392	s.s.
Heart wt. protein (g.)	181.00	27.214	s.s.
Heart % water	5.666	12.571	n.s.
Heart wt. water (g.)	1966.0	659.21	s.
Liver wt. (g.)	115396.3	18134.4	s.s.
Thyroid gland (g.)	1133.41	3.440	s.s.
Pancreas (g.)	1130.0	142.14	s.s.
Skin (g.)	2462079	755357	s.
Omental fat (g.)	93875.6	426147.8	n.s.
Mesenteric fat (g.)	86824.6	31830.5	n.s.
Kidneys (g.)	837.33	575.82	n.s.
Genital tract plus bladder (g.)	5442.6	4894.8	n.s.
Spleen (g.)	708.43	951.71	n.s.
Degrees of freedom	3	28	

APPENDIX VII. Analyses of variance of treatment effects on  
haematological components of blood.

Mean squares and significance levels are presented

<u>Item</u>	<u>Source of variation</u>		
	<u>Plane of nutrition</u>	<u>Within subclass</u>	<u>Significance</u>
Total red blood cells (m/c.mm.)	11.808	2.467	s.s.
Haematocrit %	109.533	33.076	s.
Haemoglobin (g./100 ml.)	6.753	1.934	s.
Mean cell volume (c.u.)	20.766	9.988	n.s.
Mean cell haemoglobin (u.u.g.)	5.776	0.842	s.s.
Mean cell haemoglobin conc. %	10.560	2.444	s.
Total leucocytes (per c. mm.)	44484960	4757376	s.s.
Neutrophils (%)	94.433	102.00	n.s.
Eosinophils (%)	135.166	18.666	s.s.
Lymphocytes (%)	167.566	106.518	n.s.
Degrees of freedom	3	26	