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**THE EFFECT OF MANIPULATION OF
FEED INTAKE DURING PREGNANCY
ON LAMB BIRTH WEIGHT**

T A R S O N O

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The Effect of Manipulation of Feed Intake during Pregnancy on Lamb Birth Weight

A thesis presented in partial fulfilment
of the requirements for the degree of
Master of Applied Science in Animal Science
at Massey University
Palmerston North
New Zealand

T A R S O N O

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ABSTRACT

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This study tested the hypothesis that increased maternal nutrition during early and mid-pregnancy could affect placental and fetal development at mid-gestation and fetal weight at term.

Mixed-aged Romney ewes ($n=136$) were mated at a synchronised oestrus and then randomly allocated to a M ("maintenance", average live weight 54.5 ± 1.5 kg) or H ("high", equal to 1.5M, average live weight 55.3 ± 1.5 kg) feeding level from day 19 of pregnancy (P19). At P47, the M group was divided into two groups and each group was allocated to either a M or H feeding level until P102. Thirty ewes (10 per group) were slaughtered at P102-104. The remaining ewes from each group were further subdivided into either a M or H feeding level from P102 to P136. These ewes were slaughtered at P136-140. Maintenance requirements for a 55 kg ewe were assumed to be 11 MJ ME/day at an energy concentration of 10 MJ ME/kg DM.

Over the period from P19 to P102, mean herbage dry matter (DM) intake ranged from 0.98 to 1.24 kg ewe⁻¹ day⁻¹ resulting in ewe live weight changes of 3.1, 4.8 and 5.9 kg for the MM, MH and HH groups ($P<0.05$) respectively. From P102 to P131, mean herbage DM intake ranged from 0.97 to 1.66 kg ewe⁻¹ day⁻¹ resulting in ewe live weight changes of 5.2, 9.0, 8.4, 14.0, 9.2 and 14.8 kg for the MMM, MMH, MHM, MHH, HHM and HHH groups ($P<0.05$) respectively. Feeding level had no significant effect on placental and fetal weights at either of the two slaughter periods (P102-104 and P136-140). Placental weights at P102-104 were 658.0 ± 49.5 , 612.1 ± 49.5 and 676.7 ± 50.6 g, and fetal weights were 1281.7 ± 50.4 , 1296.0 ± 50.8 and 1258.2 ± 53.4 g for

the MM, MH, and HH groups, respectively. At P136-140 placental weights were 583.2 ± 81.9 , 545.8 ± 72.8 , 602.3 ± 77.4 , 551.5 ± 72.8 , 622.5 ± 84.6 and 547.3 ± 86.7 g, and fetal weights were 4535.9 ± 175.4 , 4640.5 ± 162.7 , 4836.6 ± 166.3 , 4651.5 ± 159.3 , 4408.5 ± 186.1 and 4389.2 ± 189.1 g for the MMM, MMH, MHM, MHH, HHM and HHH groups, respectively.

Pelt weights were significantly ($P < 0.05$) affected by pregnancy rank at P102 but final ewe live weights and carcass weights were not. Other components (*i.e.*, total placentome and total cotyledon) were significantly ($P < 0.05$) heavier in twins than in singles but were not affected by feeding level. Ewes carrying twin fetuses had significantly ($P < 0.05$) more placentomes and tended to have more caruncles than single-bearing ewes. Caruncle occupancy was significantly ($P < 0.05$) higher in twins than in singles (87% vs 80%, respectively).

Pre-partum nutritional treatments from P102 to P136 affected final ewe live weights, carcass weights and pelts weights ($P < 0.05$). Pregnancy rank had no effects on final ewe live weights or pelt weights but did affect carcass weights at P136. Carcasses of ewes carrying a single fetus were heavier than those of ewes carrying twins. Single-bearing ewes had lower weights of mammary glands, uterus, myoendometrium, fetal membranes, total placentomes, and total cotyledons, and had lower placentome numbers compared to ewes carrying twins ($P < 0.05$) at P136. Weights of gravid uterus, total caruncle weights and total caruncle numbers were not affected pregnancy rank at day 136 of gestation.

Based on the comparison of these results with earlier studies, it can be concluded that quite severe nutritional treatments are required to influence placental and fetal weights at P102-104 and P136-140.

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LIST OF ABBREVIATIONS

°C	degrees celcius
%	percentage
µg	microgram
cm	centimetre
C	control (group)
CIDR	controlled internal drug releaser
Cr	chromium
Cr ₂ O ₃	chromic oxide
CCK	cholecystokinin
CRC	controlled release capsules
CRL	crown rump length
d	day
D	digestibility
day ⁻¹	per day
DM	dry matter
DMI	dry matter intake
DOM	digestible organic matter
DOMI	digestible organic matter intake
EPM	Ellinbank Pasture Meter
FO	faecal output
g	gram
h	hour
H	high (group)
ha	hectare
HFRO	Hill Farming Research Organisation
IL-1	interleukin-1
kg	kilogram
kg ⁻¹	per kilogram
M	maintenance (group)

ME	metabolisable energy
MJ	megajoule
mm	millimetre
OF	oesophageal fistulated
OM	organic matter
P	day of pregnancy
VFI	voluntary feed intake
vs	versus

Statistical:

n	number of experimental units
<i>P</i>	cut of value of significancy
s.e.m.	standard error of the mean

CHAPTER ONE

LITERATURE REVIEW

1.1. INTRODUCTION

Farming systems in New Zealand are based on three main sectors, dairy cattle, beef cattle and sheep, with minor contributions from other species, and have developed on a regional basis reflecting the underlying pasture production in the regions, climatic constraints, and year-to-year variation in pasture growth (Matthews *et al.*, 1999). These farming systems lead to New Zealand farmers' income (hence New Zealand as a whole country) deriving from the sale of animals and animal products. Based on Statistics New Zealand (1997), in 1996, this country generated significant revenue (out of total exports of \$ 19,964 million) from pastoral (\$ 8,798 million) and agricultural (\$ 11,055 million) exports. This means that pastoral-based exports contributed about 80% by value of agricultural exports and 44% of total exports from New Zealand.

As there is only small and limited local market for agricultural products (3.6 million people live in New Zealand), it is inevitable that most agricultural products must be exported to competitive world markets which are sometimes 10 000 km away, constrained by tariff and import barriers, and often in competition with the subsidised output of the local industry. In addition to this, there have been no subsidises for agriculture in New Zealand since 1996, and farmers in this country are exposed to the real market and market prices, often leading to significant changes in the balance of livestock numbers (Matthews *et al.*, 1999).

New Zealand agriculture has remained competitive - (*e.g.*, sheep meats and wool occupy 53 and 30% shares of the world markets, respectively, New Zealand Meat and Wool Board, 1997) - by developing low-cost production systems based on grazed pasture with high output per hectare and per labour unit (Matthews *et al.*, 1999). However, improving the efficiency of livestock production can still increase farmers' income. In sheep farming for

example, about 4,000,000 lambs (15% of newborn lambs) die in the first three weeks of life each year in New Zealand (Morris, 1997). If farmers could improve by 2% the survivability of newborn lambs, they would have additional revenue of \$ 4,000,000 each year (assuming that the price is \$50 per lamb sold).

Improving lambing percentage is the most important factor for increasing profits on sheep farms. Lambing time is the 'crunch' period when the benefits from all the work done before and during mating and throughout pregnancy can be realised with a good lambing percentage (Geenty, 1997). There is evidence, however, that most lamb deaths occur by day three after birth (Hight and Jury, 1970; Dalton *et al.*, 1980), and the deaths are mainly due to dystocia in single-born lambs (45%) and to the starvation/exposure complex in multiple-born lambs (42%) (Hight and Jury, 1970). According to Geenty (1997), lamb mortality ranges from 5-26% between farms, is higher for multiples than singles, and the death rates are heavily influenced by lamb birth weight. Thus, a major goal of sheep farming systems is to increase lamb survival and one method of achieving this is by narrowing the range of birthweights and ensuring that all lambs born are at an optimum birth weight.

Maternal nutrition during pregnancy has been widely reported to have an effect on lamb birth weight and hence lamb survival. Much research into maternal nutrition and its effect during pregnancy has been conducted at feeding levels equal to or below animal requirements. Smeaton *et al.* (1985), for example, observed a 25% decrease in lamb birth weight when ewes were fed below maintenance during mid- and late pregnancy. Moreover, lambs born to ewes underfed during mid- and late pregnancy also have lower body fat reserves and a reduced chance of survival (Geenty, 1997). Undernutrition during pregnancy can also restrict mammary growth and development (Russel *et al.*, 1977). Interestingly, recent research at Massey University (Cooper *et al.*, 1998) has shown that, when ewes were offered pasture allowances during the first 100 days of pregnancy at a level 50% above their maintenance (M) requirements, heavier placentas and fetuses were produced compared to control treatment ewes fed at a maintenance

allowance. If this enhanced placental growth could subsequently increase fetal growth, then placental enhancement might lead to improved lamb birth weight.

Manipulating ewe pasture intakes during pregnancy, for example, by changing pasture allowance, could thus result in placental and fetal growth differences. If this was successful, the formulation of feeding strategies that ensured optimum growth of the ovine fetus could enhance survival of the under-sized newborn.

This chapter reviews the literature on this subject and includes sections on: lamb birthweight and newborn lamb survival; factors affecting pasture intake; placental and fetal development; maternal nutrition and fetal growth; and finally the purpose and scope of the study.

1.2. LAMB BIRTHWEIGHT AND NEWBORN LAMB SURVIVAL

Lamb mortality is a major cause of reduced productivity of sheep, and has been the subject of many investigations for several decades as it affects total revenue for sheep farmers. For practical perspectives, lamb mortality is categorised into four periods (Sargison, 1997) including losses occurring between conception and mid-pregnancy (stage of ultrasound scanning), losses occurring between scanning and lambing, perinatal lamb losses, and lamb losses between one week to weaning.

There is plenty of evidence (*e.g.*, Bradford *et al.*, 1974; Doney *et al.*, 1976; Rhind *et al.*, 1984; Sargison, 1997), from studies using multiple ovulation and embryo transfer technologies, that about 20-40% of embryos die at or around the time of implantation (day 19). Losses of embryos/fetuses from day 30 to parturition, which are principally due to placental and fetal exposure to infectious agents (abortion) (Sargison, 1997), are low when compared with deaths occurring later during the perinatal period (Moore *et al.*, 1960; Quinlivan *et al.*, 1966; Sargison, 1997). According to the MAF Animal Health Laboratory data, it is estimated that the national incidence of abortion in

lowland flocks and rotationally grazed hill flocks is about 2-3%, although in individual naïve flocks, abortion storms causing up to 70% losses occasionally occur (Sargison, 1997). Perinatal lamb losses, which refers to lamb deaths occurring between the last week of gestation and the first week of life, accounted for 5-27% (Hight and Jury, 1970; Dalton *et al.*, 1980). Such quoted figures, however, need to be interpreted with caution, because of considerable variation within and between flocks, districts, seasons, sheep breeds, ewe age groups and farm management systems (Knight *et al.*, 1979; Dalton *et al.*, 1980; Hinch *et al.*, 1986; Merrell, 1995; Sargison, 1997).

Among the many factors involved in embryonic, fetal and lamb survival, viability of the newborn lamb possibly has the greatest impact on overall profitability (Sargison, 1997). This is apparent when we consider how much the value of lamb losses during the perinatal period is. For example, each year in New Zealand approximately 4,000,000 lambs (15% of newborn lambs) - all of which are inherently viable at birth - die in the first three weeks of life (Morris, 1997). In fact there is also about a 25-30% difference between the number of fetuses present at scanning and the number of lambs docked (Sargison, 1997). Morris (1997) noted that aside from the tremendous wastage, these losses have important animal welfare implications and represent one component of this country's agricultural practices which is of increasing concern in terms of a potential imposition of non-tariff trade barriers. Thus, as the New Zealand sheep industry becomes more centred on lamb production, lamb survival will become critical to its profitability (Sargison, 1997), by putting lamb meat products more competitively into international markets.

1.2.1. Causes of Perinatal Lamb Mortality and the Association with High and Low Birthweights

Many studies have been carried out to investigate the relationship between birthweight and mortality, and post-mortem studies (*e.g.*, Dalton *et al.*, 1980; Duff, 1981) concluded that there was a definite relationship between the cause of death and lamb birthweight. Trials carried out at Whatawhata by Dalton *et al.* (1980) showed that the mortality rate is high for lambs that are

born at very low weights and lambs that are very heavy at birth. On the basis of observations on 10,048 lambs born, a birth weight range of 3.5 to 5.5 kg was identified as being optimal. However, even within this weight range there is still lamb wastage of at least 13% (Morris and Garrick, 1998), suggesting there are some other factors involved in lamb mortality such as diseases and maternal failure. There are at least three major causes of lamb wastage as follows.

The first major cause of lamb deaths is dystocia. This is defined as “parturition considered likely to result in injury to the lamb or ewe” and is usually associated with a long and difficult parturition. It may arise from disproportion between fetal size and the dam’s pelvic size, malpresentation of the lamb or uterine inertia. The death of the lamb may occur during or immediately after parturition, or alternatively the lamb may survive but die from starvation as a result of impaired maternal or lambs behaviour (Morris and Garrick, 1998). The death rate caused by dystocia is about 30%, primarily in large single-born lambs (McCutcheon *et al.*, 1981; Morris, 1997).

The second cause of lamb deaths is starvation/mismothering/exposure. These lambs usually die between 1 and 3 days after birth and show evidence of activity (*i.e.*, having breathed and walked), extensive depletion of body reserves and a gut devoid of food. Exposure occurs when the rate of heat loss exceeds heat production, causing a decline in deep body temperature until the animal can no longer function and dies. Starvation involves a depletion of body reserves until there is insufficient energy remaining for metabolism and heat production (Morris and Garrick, 1998). Lamb death caused by starvation/exposure is about 30% of all deaths and occurs particularly in small multiple-born lambs (McCutcheon *et al.*, 1981; Morris, 1997).

The last cause of lamb deaths is a group referred to as “other causes”. These include deaths, which cannot be simply categorised into either of the above causes, such as congenital deformities or misadventure (Morris and Garrick, 1998), and chondrodysplasia (spider syndrome) in the Suffolk breed

(West *et al.*, 1994; Sargison, 1997). Iodine deficiency (goitre) (Caple *et al.*, 1982), selenium deficiency (white muscle disease) (Scales, 1974), and lamb losses due to wild pigs (Bruere and West, 1993) have been associated with high perinatal lamb mortality rates (Sargison, 1997).

Dystocia and starvation/mismothering/exposure have been the most often diagnosed by several investigators (Hight and Jury, 1970; Knight *et al.*, 1979; Dalton *et al.*, 1980; Duff, 1981; Hinch *et al.*, 1986), all of whom have concluded that the two mortality types are associated with high and low birthweights respectively. Unfortunately, there are several possible aetiologies for both diagnoses (Sargison, 1997). Further investigation concluded that dystocia injury alone may not result in lamb death, which may only occur when the lamb is subsequently subjected to cold stress or undernutrition. Likewise, the diagnosis of “starvation/mismothering/hypothermia” has several aetiologies, including dystocia (Sargison, 1997). Thus, it is likely that combination of events occurring pre-partum, during parturition, and post-partum result in many post-partum deaths.

1.2.2. Efforts to Improve Lamb Survival by Using Manipulation of Birthweight

In sheep flocks with a high incidence of multiple births, an essential consideration is the whole question of keeping the lambs alive after they are born because there is no point in having more twins and triplets if they fail to survive. As mentioned earlier the problem with multiple births is that high mortality rates are inevitable because of lower birthweights, suggesting that improving birthweights of multiple lambs could increase their survival.

Three strategies which could increase lamb birth weight and hence lamb survival are manipulating hormonal systems, pre-lamb shearing or by manipulating pre-partum maternal nutrition.

The effect of manipulating hormonal systems on lamb birth weight

It has been known that hormones such as insulin, glucocorticoids, thyroid hormones, growth hormone, and placental lactogen, have an important role in the regulation of fetal growth. They act on both tissue accretion and differentiation, and enable a precise and orderly pattern of growth to occur during late gestation. In part, their actions may be mediated by other growth factors such as the insulin-like growth factors (IGFs). The hormones act through both metabolic and non-metabolic mechanisms to ensure that the growth rate is commensurate with the nutrient supply (Fowden, 1995).

Insulin affects fetal growth by increasing the mitotic drive and nutrient availability for tissue accretion but it has a little effect on tissue differentiation. Evidence for this is that in fetal sheep, bodyweight and crown rump length (CRL) at term were reduced by 30-40% and 20-30%, respectively, when insulin deficiency was induced in mid- to late pregnancy by either pancreatectomy or streptozotocin (Fowden, 1995). In contrast, the main effects of cortisol *in utero* are on tissue differentiation and maturation. Cortisol appears to act directly on the cells to alter gene transcription or post-translational processing of the gene products. Cortisol may also initiate the transition from the fetal to the adult modes of growth regulation by inducing the switch from IGF-II to IGF-I gene expression in the fetal liver.

Thyroid hormones have an important role in fetal growth and development, and have been shown to affect both tissue accretion and differentiation (Fowden, 1995). However, the effect of these hormones in body growth is not consistent. For example, bodyweight, CRL and limb lengths at term were reduced by 30%, 9% and 25%, respectively, after fetal thyroidectomy and were restored to normal values when thyroxine (T_4) replacement treatment was given from the day of the thyroid ablation (Fowden and Silver, 1995). Further investigation by Fowden (1995) has shown that thyroid hormones stimulate fetal growth by both metabolic and non-metabolic mechanisms. The main metabolic action of T_4 , which is important in controlling fetal growth, is the stimulation of O_2 utilisation by fetal tissues. Umbilical O_2 uptake, for

example, is reduced by 20-30% after fetal thyroidectomy and is restored to normal value when fetal T_4 concentrations are maintained after thyroidectomy by exogenous T_4 administration. Conversely, increasing fetal T_4 concentrations above normal levels raised O_2 consumption by the sheep fetus. As a consequence of its low O_2 uptake, the thyroidectomised fetus will have less energy available for growth.

Effects of recombinant bovine growth hormone (bGH) treatment of pregnant ewes on maternal metabolism, placental development and fetal growth were examined (Jenkinson *et al.*, 1999). There was an increase in the weights of myoendometrium and of the gravid uterus in bGH-treated ewes. It was concluded that administration of exogenous bGH to pregnant ewes could stimulate fetal growth, but only after about day 100 of gestation. This response seems most likely to reflect changes in maternal nutrient partitioning or placental function, rather than placental size. These studies suggest a role of growth hormone of maternal or placental origin in the regulation of fetal growth.

Thus, the manipulation of hormones seems to have great potential for the regulation of fetal growth. Injection of exogenous hormones to multiple-bearing ewes may result in increased placental development and fetal growth, and hence increased lamb birthweight. However, it is questionable whether this would be acceptable to consumers at the present time.

The effect of pre-lamb shearing on lamb birth weight

Shearing of pregnant ewes at pasture beyond day 100 of gestation (Dabiri *et al.*, 1994; Dabiri *et al.*, 1995; Dabiri *et al.*, 1996) generally has not led to an increase in birthweights of lambs (Morris, 1997). Birthweight responses in housed ewes are greatest when shearing occurs relatively early in pregnancy, perhaps reflecting both an effect on placental development and the greater time available for altered fetal growth before parturition (Black and Chestnutt, 1990; Morris, 1997). These facts led Massey University to conduct research on pre-lamb shearing to determine whether shearing earlier

in pregnancy leads to a greater increase in fetal growth, as measured by birthweight, than shearing in late pregnancy (Morris, 1997).

Results from this study showed that there was a highly significant interaction between time of shearing and lamb birthweight. Time of shearing did not affect birthweight of single-born lambs (*i.e.*, 5.8; 6.0; 5.6 and 6.2 kg representing ewes shorn at P70; P100; P130 and unshorn, respectively), but birthweight of twin lambs increased with earlier maternal shearing (*i.e.*, 5.0; 4.7; 4.6 and 4.3 kg representing shearing at P70; P100; P130 and unshorn, respectively). It was concluded that while shearing had no effect on the birthweights of singles, those of twins have increased progressively as the shearing took place early in pregnancy, reaching a maximum of 0.7 kg (per lamb) in ewes shorn at day 70 of pregnancy (Morris, 1997).

Recent studies at Massey University (Kenyon *et al.*, 1999; Morris *et al.*, 1999 and Morris *et al.*, 2000) indicate that pre-lamb shearing during mid pregnancy (day 50 to day 100 of gestation) has a potential to increase lamb birthweights. Shearing ewes at this time can increase lamb birthweight by up to 1.0 kg (Morris *et al.*, 1999; Morris *et al.*, 2000), and decrease mortality rates of twin-born lambs by 3% when shorn-ewes at pregnancy day 67 compared with those unshorn-ewes (*i.e.*, mortality rates of 15 vs 18%) (Morris *et al.*, 1999). A further study has shown that the increase in lamb birth weights from shearing ewes between day 50 and 100 of pregnancy is associated with an increase in thyroid hormone concentrations in the maternal circulation (Morris *et al.*, 2000), and non-insulin dependent uptake of glucose by the placental-fetal unit (Revell, *et al.*, 2000). Furthermore, it was recommended that pre-lamb shearing offers real advantages to farmers wishing to increase lamb survival at birth (Morris *et al.*, 1999).

The effect of pre-lamb maternal nutrition on lamb birth weight

As lamb death is closely correlated to high and low birthweight, “narrowing the range” in birthweights, offers a partial solution to the problem (Morris, 1997). However, the ewe is resistant to attempts to alter the birthweight of

her lambs, by nutritional manipulation during late gestation, because of her ability to buffer the fetus against such changes by using her own body reserves. Furthermore, because neonates of both high and low birthweight are at risk, simply changing the mean birthweight is unlikely to reduce mortality (*e.g.*, increasing mean birthweight may increase the survival of the small twins but it will concurrently reduce the survival of large single-born lambs). Therefore, the solution lies in the developing methods which allow selective manipulation of the growth pattern of fetuses destined to be born as singles or twins (Morris, 1997).

Because placental development is especially important in ewes lambing 150% or more to ensure that lambs are sufficiently heavy at birth, a similar consideration to that of Morris (1997) is also suggested by Geenty (1997).

Inadequate placental development due to large litter sizes, or severe maternal undernutrition during mid pregnancy (Barlow *et al.*, 1987), can result in poor lamb birth weights. Furthermore, severe undernutrition during the last six weeks of pregnancy results in the birth of hypoglycaemic lambs with poor accumulations of liver glycogen and brown fat (Mellor and Murray, 1985a), and in poor udder development and colostrum production (Mellor and Murray, 1985b) that lead to death at the end.

Thus, an adequate maternal nutrition during pregnancy is essential to ensure appropriate lamb birthweights. The question is whether birthweight of singles can be reduced to the optimum birthweight range so as to avoid deaths caused by dystocia, while at the same time increasing birthweights of twins to the optimum birthweight range so that multiple-born lambs are protected from deaths caused by starvation/exposure.

The effect of maternal nutrition during early and mid-gestation has been studied by Cooper *et al.*(1998), who allocated 60 Romney ewes to three nutritional treatments, 0.5 maintenance (M), 1.0 M or 1.5 M from days 21 to 101 of gestation. The nutritional treatments generated a difference of 23 kg

live weight between the 1.5M and 0.5M groups. A 7.9 kg (16.8%) liveweight decrease in the 0.5M group did not significantly reduce fetal and placental weights at day 101 of gestation. However, increased fetal and placental weights were observed in the 1.5M group ewes, which gained 15.6 kg (21.4%) live weight over the trial period. Ewe liveweights for the three groups and their standard error of means at slaughter (day 101 of gestation) were 45.8 ± 1.4 , 56.8 ± 1.4 and 69.1 ± 1.4 kg respectively ($P < 0.001$). Fetal weights at slaughter were 1249.9 ± 40.6 , 1280.8 ± 38.0 and 1379.8 ± 35.2 g respectively ($P < 0.05$). It was concluded that, although the fetus places a relatively small nutritional demand on the dam in early gestation, fetal development in some situations be influenced by maternal nutrition, particularly when dam are over- rather than under-fed (Cooper *et al.*, 1998).

In summary, it has been shown that a high incidence of perinatal lamb deaths is associated with extremes of birthweight (*i.e.*, either lambs born too heavy or too light). The whole question of keeping the lambs alive after they are born becomes an essential consideration in sheep farming systems, as there is no point in having more twins and triplets if they fail to survive. Maternal undernutrition during mid-pregnancy can variably both to retard (McCraib *et al.*, 1986) and increase (Faichney and White, 1987) placental growth. In the first 100 days of gestation, fetuses require a small amount of nutrients for growth and development relative to that of the dam because of their small size. On the other hand, placental development increases to a maximum value by day 90 to 100 of pregnancy, which may require a higher level of maternal nutrition. If placental growth can be manipulated by changes in maternal nutrition during the first 100 days of gestation, it is not impossible that fetal growth can be subsequently influenced because of the close relationship between placental and fetal growth.

1.3. FACTORS AFFECTING PASTURE INTAKE OF GRAZING SHEEP

Feed intake is a result of three behavioural variables, grazing time, bite size and bite rate, all of which can vary amongst individuals. Maximum bite size is

related to the width of the mouth and the depth of the mouth cavity. Grazing time is composed of prehending, chewing and preparing the bolus to be swallowed. It also has a component of walking to a new grazing site, searching and selecting food (Lynch *et al.*, 1992). Because grazing is the most dominant behaviour of sheep each day, pasture intake has a major influence on animal performance (Poppi *et al.*, 1987). Therefore, it is important to study the factors that influence intake levels. The following sections discuss factors affecting pasture intake.

1.3.1. Plant Factors

Pasture characteristics composition affecting bite dimensions

The preference of sheep for various parts of the plant has a marked effect on intake per unit time (Hodgson, 1982), and most grazing animals select green herbage in preference to dead material. Dead material may be rejected because of low preference or its inaccessibility in the base of pasture (Clark *et al.*, 1982; Poppi *et al.*, 1987). On the other hand, a high proportion of green leaf in the diet selected may be due to its ease of prehension, as leaf has a lower structural strength and shear force than stem (Hendricksen and Minson, 1980).

It has been shown that plant height and density affect rate of intake. In the field there are great problems in achieving adequate experimental control over factors such as pasture height and bulk density. There has been some success with single forage species, but complex plant mixtures are far more difficult. Bite weight and hence bite volume are also affected by density of the pasture at the site of grazing (Lynch *et al.*, 1992). Allden and Whittaker (1970) have related the rate of herbage intake (g/min), size of bites and number of bites, and grazing times to herbage available and length of tillers. They found that bite size and rate increase with increases in pasture height and tiller length, while bite frequency declines when tillers are longer than 5 cm or there is more than 1000 kg/ha of pasture mass.

Sward structure can also influence herbage intake. In a study by Penning *et al.* (1991), a range of ryegrass swards were created by maintaining pastures at heights ranging from 30-120 mm for almost 6 months. Grazing time and prehension rate decreased with increasing height while mastication and rumination rate increased. Jaw movements of sheep in all pastures were constant at 150 per minute with the rate of prehension varying from 20,000-60,000 bites per day. Other research suggests that quality pasture at an optimum height of between 30 and 60 mm will maximise lamb growth rate and minimise ewe liveweight change (Lynch *et al.*, 1992). In addition, rate of intake will decline with very tall pastures although this varies considerably between plant types (*e.g.*, grass or clover) of the same height (Hodgson, 1982; Black and Kenney, 1984; Poppi *et al.*, 1987).

The breaking strength of plant material may also affect intake per bite where the size of a bite may be limited by the maximum force the animal is able to exert in prehending a bite (Evans, 1967; Poppi *et al.*, 1987). The choice by an animal of leaf or stem may be related to shearing strength and intake per bite would decrease as tensile strength of leaves increases (Evans, 1967; Hendricksen and Minson, 1980; Poppi *et al.*, 1987). Pseudostems of differing diameter and therefore breaking strength can be present in the pasture as a result of differing plant maturity and previous grazing management (Poppi *et al.*, 1987). Sheep can exhibit preferences about the depth they graze into the pasture. Using data provided by Bartham (1986), Lynch *et al.* (1992) found that when placed on a high-quality ryegrass pasture, sheep do not graze the area dominated by pseudostem and dead material but if the pasture is mixed and contains white clover, sheep will graze the pseudostem.

Therefore it is clear that pasture characteristics and pasture composition have a major influence on the intake of animals resulting from different animal preferences for plant components and their relative abundance and accessibility.

Digestibility

The major non-animal factor influencing intake is digestibility of the pasture being eaten. Poppi *et al.* (1987) noted that digestibility also has the major influence on the M/D value (MJ ME/kg DM) of pasture. This arises because the conversion of digestible energy to metabolisable energy shows little variation (0.79 - 0.84, assumed to be 0.82) whilst digestibility can vary from 0.4 (dead material) to 0.85 (grass or legume leaf). However, there is no consistent relationship between intake and digestibility. Intake of legumes is 40% greater than that of grass while leaf is 100% greater than stem when each is compared at the same digestibility (Ulyatt, 1971; Laredo and Minson, 1973; Rattray *et al.*, 1983; Cruickshank *et al.*, 1985; Poppi *et al.*, 1987).

It has been argued that intake is controlled by the amount of dry matter which can be contained in the rumen and the rate at which it disappears from the rumen. This disappearance of the material from the rumen occurs by digestion and by passage down the digestive tract. Factors such as chemical and structural composition of the forage and the adequacy of nutrients for microbial growth in the rumen, can influence digestion rate. For example, cell contents (largely soluble carbohydrates and protein) are rapidly digested whereas cell walls (the fibrous fraction of cellulose and hemicellulose) are slowly digested. The structural composition of the pasture can influence the digestion rate by influencing the ease with which microbes can attack various anatomical parts of a plant. Rate of passage of digesta out of the rumen is influenced by rate of the breakdown of large feed particles to small ones (*i.e.*, less than 1 mm in sheep and 2 mm in cattle, Waghorn and Barry, 1987) and rate of water flow from the rumen (Poppi *et al.*, 1987).

Water content of pasture may also influence intake (John and Ulyatt, 1987; Poppi *et al.*, 1987) although it is difficult to examine as plant intracellular water content would appear to have a different effect to intra-ruminal addition of water (Poppi *et al.*, 1987).

In summary, digestibility is a major determinant of pasture quality because of its influence on the M/D value of forage.

1.3.2. Animal Factors

Increased physiological drive, caused by high growth in young animals, poor body condition, pregnancy and lactation, increases grazing time and rate of intake (Arnold, 1981; Poppi *et al.*, 1987). In addition, behavioural studies have shown that young sheep accept new feeds more readily than adults do (Lynch *et al.*, 1992). Other factors, such as metabolic diseases and stress, can also affect feed intake (Baile and Forbes, 1974). For example, stress caused by dehydration results in decreased feeding in ruminants (Calder *et al.*, 1964; Baile and Forbes, 1974).

1.3.3. Regulation of Feed Intake during Pregnancy

In general, voluntary feed intake (VFI) in sheep remains constant during early pregnancy, increases (at a modest level) during mid-pregnancy, and often decreases in late pregnancy. Small increases in intake in mid-pregnancy, for example, have been noted in grazing ewes (Owen and Ingleton, 1963; Forbes, 1970). Hadjipieris and Holmes (1966) found increased intake in mid-pregnancy in single-bearing ewes but not in those carrying twins. In the last few weeks of pregnancy there is often a decline in intake, which starts earlier and is steeper with larger litter sizes (Reid and Hinks, 1962; Owen and Ingleton, 1963). This phenomenon suggests that there is a close relationship between intake and physiological states of animals which regulates the amount of feed eaten during pregnancy.

The regulation of feed intake in the sheep is complex involving many physiological processes, and is the subject of controversial discussion. In the roughage-fed sheep, it is clear that the digesta in the reticulo-rumen and the animal's energy transactions are important components of the regulation (Weston, 1985). However, a new theory suggests that feed intake is adjusted to maximise the efficiency of oxygen utilisation (Ketelaars and Tolkamp, 1992; Tolkamp and Ketelaars, 1992). This complexity in feed intake regulation is quite apparent, particularly when it is realised that this

control not only involves internal factors which have been shown to regulate feed intake but also acts together with the external factors such as stocking rate, herbage availability, grazing time, bite size and bite rate (Lynch *et al.*, 1992).

It has been suggested that there are short-term stimuli which control bouts of eating and long-term stimuli which regulate overall feed intake and hence body weight (Weston and Poppi, 1987; Lynch *et al.*, 1992). When eating occurs there are clearly many intrinsic factors producing stimuli, the net results of which is satiety or hunger. Figure 1 is a schematic diagram of the internal factors that have been shown to regulate feed intake.

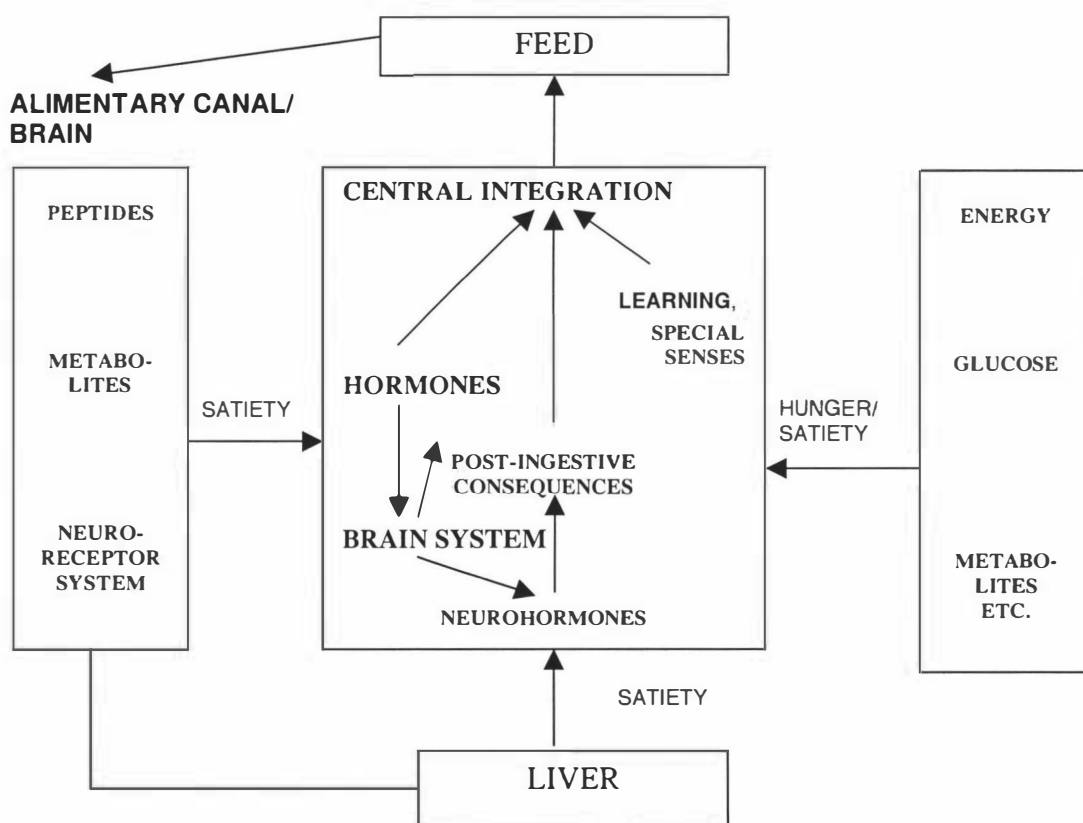


Figure 1. A model of animal factors affecting feed intake (From Lynch *et al.*, 1992).

The short-term stimuli arising from the chemical and physical properties of the feed operate to stop or start bouts of eating through stimuli in the oropharyngeal area to stimulate feed intake, while gut distension,

gastrointestinal and liver stimuli act to inhibit feeding. The role of hormonal factors is still uncertain, but exogenous cholecystokinin (CCK) decreases feed intake and is probably involved in eliciting satiety (Baile and McLaughlin, 1987; Lynch *et al.*, 1992). At least in rats, endogenous CCK, released from the upper part of intestine, induced satiety by stimulating receptors in the brain. In sheep, it has been shown that the longer the fasting period the greater the amount of CCK needed to suppress feeding. Also, neutralisation of CCK by antisera injected into the cerebrospinal fluid delayed satiety in sheep. Further, since CCK changes gut motility and affects secretion of insulin and glucagon, satiety could result directly from suppression of eating behaviour or indirectly from hormonally mediated metabolic changes (Lynch *et al.*, 1992). There is even less known about the long-term control of feed intake. It would appear that the overall decision about nutrient requirements has to be the integration of multiple inputs (Lynch *et al.*, 1992).

The physiological state of the animals may have a marked effect on VFI. In general, VFI is higher in physiological states that are associated with an enhanced energy demand by the animal even though the association is not universal. For example, the consumption of roughage diets increases in lactation when energy need is high but it generally fails to increase, and often decreases, in late pregnancy when extra energy is required for growth and maintenance of the utero-fetal complex and the mammary glands. In addition, as pregnancy progresses, there is competition for space in abdomen and remarkable changes in hormone concentrations in the blood. These phenomena suggest that either space limitation is the major factor decreasing VFI in late pregnancy or other factors, which are associated with late pregnancy, influence the VFI (Forbes, 1986a). Thus, the objective of the next section is to review the factors that the animal uses to increase its intake during a period of increased energy demand such as in the late pregnancy period. The words *food* and *feed* in this section are used interchangeably.

1.3.4. Possible Causes of Declining Intake in Late Pregnant Ewes

Physical factors

It is quite possible that the commonly observed decrease in intake during late pregnancy is due to the compression of the rumen by the growing uterus, exacerbated by abdominal fat. Forbes (1968), who studied sheep at several stages of pregnancy, found that there was a negative relationship between the volume of rumen contents at slaughter (RV, litres) and the volume of 'incompressible abdominal contents' (uterus plus abdominal fat, IAC, litres) in ewes which had been fed on hay ($RV = 10.4 - 0.39 IAC$ with $r = 0.67$; $P < 0.05$). Intake during the last 2 weeks before slaughter (I , kg day⁻¹) was positively related to volume of rumen contents at slaughter ($I = 0.50 + 0.03RV$ with $r = 0.41$; $P < 0.05$). This evidence suggests that the increase in volume of other abdominal organs such as abdominal fat or the pregnant uterus apparently causes compression of the rumen and reduction in feed intake. Forbes (1970) suggested that the decrease in intake, which also occurs in cattle when fed on roughage alone, was caused by compression of the rumen by the uterus. Thus, if this theory is correct, then the larger the uterus the greater the compression of the rumen and the more severe the decline in roughage intake.

The fact that forage intake usually falls in late pregnant ewes has prompted the practical use of concentrate supplements (in Northern Hemisphere countries), given at increasing levels in the last 6 weeks before parturition. This has often been practised in experiments as well, leading to uncertainty about the cause of the decline. Wylie and Chestnutt (1992) increased the rate of supplementation of silage from 400 to 600 to 800 g day⁻¹ over the last 6 weeks of pregnancy and observed that silage dry matter (DM) intake declined from 758 to 552 g day⁻¹ in those ewes which were fed concentrates once per day, from 853 to 782 g day⁻¹ in those given the supplement twice or thrice per day, and from 996 to 876 g day⁻¹ in those animals for which the supplement was mixed with the silage. Forbes (1970) who conducted experiments with 33 Speckledface Welsh ewes, provided further evidence for this. The ewes were divided into three groups and were fed on three different types of diet (*i.e.*, barley, hay, and straw). There was significant

difference in intake between weeks within any of the three dietary treatments. However, there was a tendency for intake to increase to the 15th week of pregnancy followed by a decline to parturition (during the last 5 weeks). The slopes of the declines were significantly different from zero (straw, $P < 0.001$; hay, $P < 0.01$; barley, $P < 0.01$).

The voluntary intake of barley in the Forbes (1970) experiment declined from approximately the same stage of pregnancy as the intake of hay and straw. There were large differences between the levels of intake of the three diets, as would be expected from their widely differing chemical and physical properties. It is most unlikely that a physical limitation to intake would occur at the same time in the three feeds, which differed so widely in composition. In a further study, pregnant ewes failed to choose an adequate diet when offered an *ad libitum* choice of protein concentrate, carbohydrate concentrate and hay (Gordon and Tribe, 1951). Coppock *et al.* (1972) found that the decline in intake by cows in the last 3 weeks of pregnancy was steeper for high concentrate than for high forage rations which does not support a purely physical theory for depression of intake. Thus, in summary, physical competition may not provide a complete explanation for the decline in intake in these circumstances.

Metabolic factors

In view of metabolic effects on voluntary feed intake, two common theories (*i.e.*, glucostatic and lipostatic theories) are usually used to explain how feed intake is controlled. Because of the scarcity of data on the effects of metabolites on feed intake in late-pregnant ewes, this review will focus on those theories that affect ruminants in general.

The glucostatic theory for the control of feeding, reviewed by Mayer and Thomas (1967), stated that a ruminant monitors its energy needs via glucoreceptive brain neurones that are responsive to their own rate of glucose utilisation. Accordingly, a fall in the level of glucose utilisation by these glucose-sensitive neurones is proposed to stimulate feed intake, whereas the effect of a meal to increase glucose utilisation, leading to satiety

by activating glucose-responsive neurones. These effects of glucose were proposed to take place in 'hunger centres' located in the lateral hypothalamic area (LHA), which when it has bilateral lesions causes anorexia and weight loss in rats, and in a 'satiety centre' located within the ventromedial hypothalamic nucleus (VMN), which, when it has bilateral lesions, produces obesity (Schwartz *et al.*, 1999).

Regarding the lipostatic theory, the suggestion that body fat content is held constant by changes in energy intake (and/or output) cannot now be regarded as sacrosanct. Incorporation of fat in the diet in a rat, for example, gives a reduction in feed intake. Ruminants, such as North European domesticated breeds of sheep and cattle, become very fat if offered free access to high quality feeds but eventually reach a plateau (Forbes, 1986a). Further experiments in ruminants showed that short-chain fatty acids do affect intake. However, a mixture of short-chain fatty acids in physiological proportions (0.55 acetate, 0.30 propionate, 0.15 butyrate), which was infused intraruminally during spontaneous meals, had a dose-related effect on intake (by sheep or goats, Baile and Mayer, 1969; by cattle, Simkins *et al.*, 1965). According to de Jong *et al.* (1981) the type of infusions used by Baile and others induces large, probably unphysiological, changes in rumen fluid and blood composition. As a result de Jong and co-workers could not reproduce their results with infusions made so that, according to Forbes (1986a), these unphysiological changes were avoided.

Although plasma levels of the short-chain fatty acids increase in ruminants after large meals, the changes in peripheral blood during and between small spontaneous meals are not clear-cut. Moreover, the infusions of long chain fatty acids intravenously in sheep does not stimulate feed intake because although intake was depressed by physiological amounts of stearate, the albumin to which the fatty acids were coupled was of bovine origin and might have caused an allergic response (Forbes, 1986b). However, 5g d⁻¹ of oleic acid esters, which were infused into the jugular vein, depressed the intake of sheep compared with albumin alone (Paquay and Vernailen, 1984), supporting the idea that the effect is physiological.

Fat ewes are particularly susceptible to declining voluntary intake in late pregnancy (Everitt, 1966; Foot and Greenhalgh, 1969) and there is a greater reduction in intake of a standard food in late pregnancy in ewes that have been fed at a high level, or on good quality silage, in mid pregnancy (Wilkinson and Chestnutt, 1988; Chestnutt, 1989). It was noted that progressive reduction in *ad libitum* intake in fat ewes during this period is due to metabolic changes associated with advancing pregnancy and exacerbated by the presence of more than one fetus and by the physiological processes of fattening.

According to Forbes (1986b), the metabolic changes in pregnancy and in fattening may be similar in nature and hence additive in effect. In this respect it is of interest to note the well-known fact that both pregnancy and obesity predispose to a diabetic state. The fat ewe is generally considered to be predisposed towards pregnancy toxaemia, which shares characteristic with some forms of diabetes.

Imbalance between the nutrients required by the ewe and fetus(es) might be expected to reduce food intake. To see whether a shortage of amino acids and/or glucose was limiting intake in grass-fed pregnant ewes, Barry and Manley (1985) infused glucose and casein into the abomasum. Glucose infusion in the absence of casein depressed herbage MEI by 1.6 times the ME infused as glucose. In the absence of glucose, casein infusion depressed herbage MEI by an amount equivalent (0.9) to the casein ME infused. Conversely, the combined glucose+casein infusion depressed herbage MEI by an amount equivalent to one third of the ME infused, with the result that total MEI (herbage+infusate) was increased by this treatment. The glucose x casein interaction was significant ($P < 0.001$) for herbage and total MEI and also for herbage total N intake. Total MEI was high and tended to decline over the last 4 weeks before parturition in the group infused with glucose+casein. In contrast, MEI in the other three groups tended to rise over this period, but the infusion x time interaction was not significant ($P > 0.05$). MEI in all groups decreased during the last 3 d before parturition. As the amount of ME infused

was maintained constant for each animal, these changes with time were due entirely to changes in herbage intake.

When amino acid absorption lay within the range to produce maximum voluntary intake (*i.e.*, glucose + casein infusion), MEI was either stable or fell with advancing pregnancy probably due to reduced rumen volume as a result of the expanding uterus. However, in the remaining three groups where amino acid absorption was not in the desired range and MEI therefore below maximum, increases in MEI towards the maximum were possible and did occur in late pregnancy. MEI in all groups declined during the last 3 days preceding parturition. This is probably as a result of a peak increase in oestrogen concentration on the onset of parturition (Oddy and Annison, 1979). Barry and Manley (1985) suggested that higher level of intake in the infused animals rendered them more prone to the intake-depressing effects of pregnancy whereas the uninfused controls were still suffering from the imbalanced diet.

Hormonal factors

Forbes (1970) suggested that a hormonal factor might be controlling feed intake in late gestation because the intake of three diets of varying digestibility (oat straw, grass hay or barley) began to decrease at similar times. Green *et al.* (1994) found maternal concentrations of oestradiol (E₂) in plasma increased (linear, $P < 0.01$) as gestation progressed. Increased oestrogen concentrations in plasma during late gestation have been observed in sheep (Bell *et al.*, 1989) and in cattle (Killen *et al.*, 1989).

The role of oestrogens during late gestation appears to be related to various physiological functions occurring at parturition (Jainudeen and Hafez, 1993). In the uterus, oestrogen increases myometrial activity and the number of oxytocin receptors that are involved in expulsion of the fetus at parturition. In the mammary gland, oestrogen increases ductal and alveolar size along with the induction of synthesis of prolactin receptors. The importance of oestrogens for parturition and lactogenesis is evident. However, the relationship of oestrogens to lowered feed intake during late gestation is

unclear, especially because the overall energy demand for fetal growth and lactogenesis is increasing (Green *et al.*, 1994).

In view of the effects of oestrogen on feed intake, Forbes (1974) injected oestradiol (E_2) (80 $\mu\text{g.}$) directly into the lateral cerebroventricle of sheep, which resulted in a decrease of feed intake. Gentry *et al.* (1976) determined that receptors for E_2 existed at the hypothalamus. The hypothalamus may be a site of action of the intake-decreasing effect of E_2 . The hypothalamus is thought to be the central site for control of feed intake. Specific areas in the hypothalamus recognise peripheral energy status and hormonal inputs (Baile and Forbes, 1974).

Muir *et al.* (1972) injected late gestating dairy cows with progesterone to antagonise the effects of oestrogens. Progesterone injections increased the length of gestation and increased intake relative to control cows, suggesting that oestrogen may influence feed intake during late gestation. The blockade effect of progesterone has also been investigated by Bargeloh *et al.* (1975), and the result was the increase of VFI in late pregnancy in cows. In this trial, progesterone ($0.25 \text{ mg kg}^{-1} \text{ day}^{-1}$) was given subcutaneously for 15 days before the expected date of calving and treated cows ate $17.1 \text{ kg DM day}^{-1}$ in the last six days of pregnancy compared with $11.7 \text{ kg DM day}^{-1}$ for untreated controls. Pregnancy was prolonged in two out of the five treated cows so this type of treatment is unsuitable for practical use.

Green *et al.* (1994) showed that ovariectomized ewes with sham implants maintained feed intake throughout the trial. Ewes implanted with E_2 had a transient decline (time X treatment, $P < 0.01$) of feed intake. Intake declined immediately after implantation, however, intake was similar between ewes receiving sham or E_2 implants 7 d after implantation. Ewes implanted with E_2 had higher ($P < 0.01$) concentrations of E_2 in plasma than ewes receiving sham implants at d 12 (16.6 ± 2.8 vs. $< 2 \text{ pg./ml.}$, respectively). Although the daily delivery of E_2 was not measured, Day (1982) used similar implants and found no differences in concentration of E_2 in plasma over a 161-d period.

The decline of feed intake after administration of E_2 has been observed in sheep (Forbes, 1972) and dairy cattle (Muir *et al.*, 1972). The satiety centre lies in the ventro-medial hypothalamus that has receptors for E_2 (Gentry *et al.*, 1976). Administration of E_2 increased hepatic triglyceride production (Abraham *et al.*, 1980; Gray and Greenwood, 1982), increased serum non-esterified fatty acids and triglyceride concentrations (Ramirez, 1981), decreased adipose tissue lipoprotein lipase (Ramirez, 1981; Wade *et al.*, 1985) and decreased adipose tissue lipogenesis (Faure *et al.*, 1984). The net effect of administration of E_2 increased lipid efflux in liver and adipose tissue. Increased availability of plasma lipids decreased feed intake in sheep. Wade and Gray (1979) suggested ovarian steroids acted on the liver and the adipose tissue to increase the availability of oxidizable substrates, which may alter feed intake. Thus, these tissues are involved in energy balance regulation by direct or indirect effects.

Recently, many researchers have studied effects of other hormones such as cholecystokinin (CCK) and leptin on the regulation of feed intake, for example: the role of endogenous CCK in regulation of interdigestive pancreatic exocrine secretion in sheep (Tachibana *et al.*, 1995); the effect of proglumide - (*i.e.*, a derivate of glutamic acid that is specific and fully competitive antagonist of the interaction of CCK with its cell surface receptors on dispersal acini) - on CCK-8 induced exocrine and endocrine pancreatic responses in conscious sheep (Mineo *et al.*, 1995; Mineo *et al.*, 1997); gene expression of ovine leptin (Dyer *et al.*, 1997); and leptin synthesis of ovariectomised ewes (Bocquier *et al.*, 1998). However, since available data on the effect of leptin or CCK on feed intake especially in pregnant ewes are limited, these hormones are not discussed in detail in this paper. As a growing general acceptance, however, these hormones are considered as potential regulators of voluntary feed intake in many species.

Other possible factors

Other possible reasons for the decreased intake in late pregnancy, especially the very low levels in the last few days, include discomfort, preoccupation

with seeking a suitable place for parturition, or other endocrine changes associated with parturition (e.g., corticosteroids, prostaglandin, oxytocin, relaxin). Whatever the cause, Forbes (1986a) suggested that mixed feeding with forages supplemented by concentrates might avoid too serious a decline in voluntary intake at a time when fetuses are very susceptible to undernutrition. This practice is however not practical under extensive low-cost grazing systems.

It can be concluded that physical constraints remain a factor (but not the major factor) limiting VFI during late pregnancy. Rather, a remarkable change in the uterus resulting from a rapidly growing fetus(es) during the late pregnancy period accompanied by the dramatic increase of oestrogen hormone concentrations in blood results in decreased feed intake. The glucostatic and lipostatic theories alone are not sufficient to explain the decrease in VFI during late pregnancy as there is limited evidence to support the glucostatic and lipostatic theories in ruminants. However, imbalance between the nutrients required by the ewe and fetus(es) might be expected to reduce feed intake. Furthermore, other factors such as discomfort, preoccupation with seeking a suitable place for parturition, or other endocrine changes associated with parturition are possible causes for the decline in VFI in the last few days of pregnancy.

1.4. PLACENTAL AND FETAL DEVELOPMENT

This section reviews the literature of how the placenta develops and then discusses fetal growth.

1.4.1. Placental Development

The placenta is a result from placentation processes that progress through the stages of apposition, adhesion and attachment to develop a physical union between the chorion or outer embryonic membrane and the uterine lining (King *et al.*, 1982; King and Thatcher, 1993). King and Thatcher (1993) further give an excellent description of how the placenta develops in sheep.

Placentation in sheep commences by day 14 or 15 of pregnancy when the chorionic vesicle becomes fixed and apposition is maintained. This is followed almost immediately by adhesion and attachment between the chorionic membrane near the embryonic disc and the associated uterine epithelium (Guillomot *et al.*, 1982; King and Thatcher, 1993). The process subsequently spreads, resulting in intimate attachment between most of the chorion and the apposed uterine surface during the first four weeks of gestation. Binucleate cells migrate and fuse with other binucleates or uterine epithelial cells, forming multinucleate cells that subsequently spread or fatten to produce the extensive syncytium on the maternal side of the interface. The formation of multinucleate cells and syncytium is accompanied by degeneration of maternal epithelial cells so that much of the uterine epithelium in sheep is replaced by migrant chorionic tissue. Eventually, this process ends with the production of an organ, the placenta, that brings embryonic and maternal capillaries into close proximity to facilitate exchange of nutrients from dam to conceptus and waste products in the reverse direction (King and Thatcher, 1993).

It has been suggested that the number of placentomes increases as gestation progresses but, since endometrial differentiation into intercaruncular and caruncular components occurs prenatally in cows (Atkinson *et al.*, 1984) and sheep (Wiley *et al.*, 1987), any increase would not be classical or true placentomes. In addition, the ruminant caruncles could be damaged or destroyed by metritis or during placental removal postpartum. If the destruction is extensive, according to King and Thatcher (1993), this might result in placental insufficiency with poor development or even death of fetuses in later gestations. However, substantial numbers of caruncles can be removed from the ovine uterus without affecting placentation and fetal growth in subsequent pregnancies (Alexander, 1964a).

The non-pregnant sheep uterus contains between 60 and 150 caruncles (Alexander, 1964a; King and Thatcher, 1993). Although numbers vary between species, breeds and strains, these sites are not completely occupied by the fetal cotyledons. The proportion of caruncle occupancy is

widely variable, with an approximate 70% occupancy rate if the ewe carries a single lamb, and over 80% with twin lambs (Davis, 1983). In addition, the proportion of caruncle occupancy varies between seasons with a 79% occupancy rate when ewes mated in December, and 88% with ewes mated in March (Jenkinson *et al.*, 1994). Similarly, occupancy was 91.2% and 76.0% when lambs were due to be born in spring and autumn respectively (McCoard *et al.*, 1997).

The maximum placentome size or placental weight in sheep is reported to be attained by 70-80 days of gestation (Kelly, 1992; Ehrhardt and Bell, 1995) or at about day 90 of pregnancy (Alexander, 1964a; Robinson *et al.*, 1977; Vatnick and Bell, 1992; Geenty, 1997). Placental growth rate far exceeds the growth rate of the fetus(es) during this period. From this time until the end of gestation, placental weight may decline by up to 50%, although the dry weight of the placenta remains constant (Vatnick and Bell, 1992; McCoard, 1998).

1.4.2. Factors Affecting Placental Development

Placental weight is commonly used as a measure of placental size and is assessed from placental tissue that consists of a maternal component (caruncle) and a fetal component (cotyledon). As the number of placental units decreases, the total placentome weight also decreases but the weight of individual placentomes increases. Further, the compensatory hypertrophy of individual placentomes occurs before about 90 days of gestation (Mellor, 1983). This explains why total placentome weight appears to be a better index of placental size than placentome number.

Placental growth and the development of functional ability are important because they are the means by which the fetus receives metabolic substrates for growth (McCraab *et al.*, 1992). However, the factors controlling placental growth and functional development are unknown (Bell *et al.*, 1989; McCraab *et al.*, 1992). Factors such as breed and strain of the sheep, litter size, age of ewe, sex of lamb, nutrition and environmental conditions have been indicated to have an effect on placental size (Alexander, 1964a).

Placental weight varies widely among uniformly treated animals (Alexander, 1964a; Alexander and Williams, 1971), although the basis of this variation is poorly understood (Bell, 1984; Cooper, 1998). Low placental weights are usually associated with small number of placentomes (Alexander, 1964a; Alexander, 1964b; Rhind *et al.*, 1980; McDonald *et al.*, 1981). The number of implantation sites may be restricted by the death of littermates soon after implantation, by apparently random imbalances in the area of the uterus occupied by the chorionic envelopes of littermates, or both (Rhind *et al.*, 1980; McDonald *et al.*, 1981; Mellor, 1983).

Hormonal factors might affect placental weight (Mellor, 1983). Maternal corticosteroids may affect implantation and placental development. Treating ewes with progesterone and/or oestrogens before 70 days of gestation can alter placental morphology, uterine blood flow and uterine oxygen consumption (Caton *et al.*, 1974; Mellor, 1983).

Ambient temperature may also be an important factor affecting placental growth. Exposing ewes to high temperatures reduces both placental growth below a level of c. 250 g (Alexander and Williams, 1971) and uterine blood flow (Brown and Harrison, 1981). In a recent study by McCrabb *et al.* (1993) ewes were exposed to 42°C ambient temperature for 9 hours per day (h day^{-1}) and 32°C for 15 h day^{-1} between day 30 and 80 of gestation with the sole objective of restricting placental weight. Results indicated that heat-exposed ewes had lower placental weight than control ewes at day 80 and 140 of gestation, and that their placentae exhibited a greater number of smaller placentomes, and a lower number of large placentomes. Maternal exercise, which is known to reduce uterine blood flow in pregnant ewes (Longo *et al.*, 1978), could have effects on placental development in grazing animals if there is a relationship between uterine blood flow and placental growth (Mellor, 1983).

Maternal undernutrition during mid-pregnancy has been variably reported both to retard (Everitt, 1964; Morris, 1973; Mellor, 1983; McCrabb *et al.*, 1986; McCrabb *et al.*, 1992) and increase (Faichney and White, 1987;

McCrabb *et al.*, 1992) placental growth. There is also some indication that underfeeding during late pregnancy reduces placental weight in twin-bearing but not single-bearing ewes (Thomson and Thomson, 1948; Mellor and Murray, 1981), but this requires verification (Mellor, 1983). Underfeeding throughout the entire gestational term has been shown to significantly reduce placental weight at, or near, term (Wallace, 1948; Alexander and Williams, 1971).

1.4.3. Fetal Growth and Development

Like other mammals, sheep are viviparous, *i.e.*, embryonic and fetal development occurs within the uterus. This intrauterine developmental period is termed “pregnancy or “gestation”, and spans the period from fertilisation through to parturition. The length of the gestation period is largely genetically determined (*e.g.*, species, breed and fetal genotype). However, maternal (*e.g.*, age of the dam), fetal (*e.g.*, litter size and sex) and environmental (*e.g.*, season, nutrition or temperature) factors can modify its duration (McCoard, 1998). Generally, the average length of gestation in sheep varies from 144 to 147 days and from 149 to 151 days representing the early-maturing improved meat breeds (*e.g.*, Southdown, Suffolk, Hampshire, Dorset Horn) and the slow-maturing fine-wool breeds (*e.g.*, Merino, Rambouillet) respectively. For crossbreed long-wool breeds (*e.g.*, Columbia, Corriedale) the period is in the intermediate range.

In sheep, three prenatal periods of development are generally recognised, and have been clearly classified by McCoard (1998). The first period is termed the ‘ovum period’ that spans the period from fertilisation until the initial attachment of the blastocyst to the uterine wall before the establishment of intraembryonic circulation, and overlaps the second “embryonic” period. The “embryonic” period extends from day 12 to about day 34. At about day 28-34 of gestation, the embryo implants to the uterus. Placentome number is thought to be fixed at implantation, and each fetus develops its own placenta and can grow independently from its littermates. Thus, embryo losses at this stage could have a detrimental effect on the surviving fetus(es), as the proportion of placentomes available for each fetus

declines. Since the remaining fetus(es) are unable to utilise the placentomes of the aborted fetus, the surviving embryos are restricted to a smaller proportion of placentomes resulting in subsequent reduction in fetal growth and birth weight (Rhind *et al.*, 1980; McCoard, 1998). The last intrauterine period is termed the 'fetal period'. This period spans from implantation (around day 34 of gestation) through to until birth (Jainudeen and Hafez, 1993), and is indicated by rapid growth and changes in the form of the fetus. Fetal weight increases slowly in early gestation, then increases rapidly towards term, with about 90% of the birth weight gained in the last 40% of gestation in sheep. Fetal growth usually follows an exponential growth curve and, at term, the weight of the fetus contributes approximately 60% of the total weight of the conceptus (McCoard, 1998).

1.4.4. Factors Affecting Fetal Growth

There are many factors involved in fetal growth and development. Factors such as sex, birth rank, breed or breed cross, dam age, heat or cold stress, placental size and maternal nutrition, are considered to contribute to the growth and development of the fetus. The importance of these factors and the degree to which they affect fetal growth differ between species (Ferrell, 1989). It has been suggested that fetal growth and development is primarily determined by the fetal genome, but superimposed on this genetic drive to grow are two opposing influences. One involves factors which impose constraint on fetal growth, such as the supply of nutrients to the fetus, determined mainly by maternal supply (Robinson and McDonald, 1989; Robinson and Symonds, 1995) and placental transfer capabilities (Carter *et al.*, 1991; Robinson *et al.*, 1995). The other involves factors which stimulate fetal growth such as hormones and growth factors, *e.g.*, the insulin-like growth factors (Owens *et al.*, 1994; Robinson *et al.*, 1994; Harding *et al.*, 1994; Anthony *et al.*, 1995; Kuhn-Sherlock *et al.*, 1995). Thus, fetal growth represents a balance between constraining and stimulating forces influencing the genetically determined drive to grow (McCoard, 1998). These factors affecting fetal growth will be discussed separately (*i.e.*, placental, fetal, maternal, and environmental factors).

Placental factors

The placenta exerts its effects on the growth of the fetus from the beginning of the pregnancy via metabolic and endocrine mechanisms. To achieve this, according to Robinson *et al.* (1995), the placenta exchanges a wide array of nutrients, endocrine signals, cytokines - (e.g., interleukin (IL)-1 and tumour-necrosis factor (TNF)) - and growth factors with dam and the fetus. These exchanges modulate or programme fetal growth and development.

Many methods are used to study the relationship between the placenta and fetal weight, such as ligation of the uterine horn and excision of caruncles (carunclectomy). Those methods are used to restrict placental growth in experimental animals as have been reviewed in more detail elsewhere (Owens *et al.*, 1989; Owens 1991). In early pregnancy, restriction of the area for implantation by ligation of one uterine horn effectively reduces the number of available caruncles and placentomes in sheep by about 50% (Caton *et al.*, 1984). Reduction of maternal uterine blood flow by either embolisation or ligation of the uterine artery can also be used to restrict fetal growth (Clapp *et al.*, 1981; Bennet and Hanson, 1994), and is associated with fetal hypoxaemia and altered O₂ metabolism (Robinson *et al.*, 1995).

Fetal growth restriction can be induced by removal of endometrial caruncles in non-pregnant ewes (Alexander 1964b). The majority of visible caruncles have to be excised (carunclectomy) to significantly restrict fetal growth. The placental compensation for the reduced number of implantation sites, according to Robinson *et al.* (1995), includes extension of the implanting conceptus to the extremes of the uterus, and increased size of the individual placentomes (particularly in the body of the uterus), a process that is apparent as early as day 77 of pregnancy.

The survival of the fetus is more likely to be jeopardised by a small placenta than by decreased maternal nutrition (Mellor, 1983) although, given that reduced placental size could be a result of decreased maternal nutrition, survival of the neonate may still be indirectly affected (Cooper, 1998). A possible mechanism is that small placentas have reduced functional

transport capacity that results in restricted placental transport of oxygen and nutrients from the maternal uterine to the fetal umbilical circulation. Since an adequate supply of oxygen and nutrients is essential to support anabolism in the fetus, a reduction in placental size with a subsequent decrease in uterine blood flow can lead to fetal hypoxaemia, hypoglycaemia and growth retardation (Owens *et al.*, 1987a; Owens *et al.*, 1987b).

Fetal factors

Factors affecting fetal development and growth are present from early gestation, before implantation of the embryo. These regulatory mechanisms are present and develop in the outer layer of the developing blastocyst, the trophoctoderm (or trophoblast), even before implantation occurs and before development of the membrane layers and the vascular system joining the fetal circulation with the maternal circulation (Bassett, 1991; Reynold and Redmer, 1995; McCoard, 1998). If these mechanisms are impaired in early life, subsequent fetal growth retardation may occur (Rivera *et al.* 1996; McCoard, 1998).

The placenta also affects the growth of the fetus from the beginning of pregnancy via metabolic and endocrine mechanisms and by the exchange of a wide array of nutrients, endocrine signals, cytokines and growth factors with the mother and the fetus. Evidence shows that the fetus may also regulate placental growth by altering concentrations of nutrients and hormones transported to the placenta via umbilical cord blood and/ or by alteration of the rate of the umbilical blood flow (Bassett, 1986). The fetus may also expand the vascular network of the endometrium around implantation (Bassett, 1991) and, in conjunction with the important role that the fetus plays in placental metabolism (Bassett, 1994), may dictate its own future growth (McCoard, 1998).

Maternal factors

Growth patterns of the fetus, the uterus, its membranes and the intra-uterine fluids during pregnancy are quite distinct. The placenta develops rapidly in the early stages of pregnancy whereas the fetus grows more quickly during

the final few weeks. By the end of the third month a fetus will weigh about 14% of its birthweight. By contrast, the placenta, through which the fetus receives nutrients from the ewe, makes its most rapid growth during mid-pregnancy, generally attaining at least 95% of its final weight of about 500 g per fetus by day 90 of gestation (Russel *et al.*, 1967).

It is clear that severe undernourishment during mid-pregnancy can reduce the weight of fetuses and of placental material at 90 days. However, except in situations where the undernourishment is exceptionally severe, adult ewes are generally able to compensate during late pregnancy for the effect of low levels of mid-pregnancy nutrition. Adult ewes which are in good body condition (condition score 3) at mating are likely to be able to withstand a moderate degree of undernourishment during the second and third months of pregnancy, without this unduly affecting their subsequent performance (Robinson, 1977).

High levels of feeding may also influence fetal growth. Recent study has shown that fetal weight at day 101 of gestation was increased as a result of high levels of nutrition from day 21 to 101 of gestation (Cooper *et al.*, 1998). This topic of maternal nutrition will be covered in more depth in section 1.5.

Environmental factors

Evidence shows that heat stress, especially during mid- and late pregnancy, is probably the major environmental factor affecting fetal growth. In heat-stressed ewes, for example, fetal growth is retarded as a result of a primary restriction in fetal growth (Bell *et al.*, 1987). Similarly McCrabb *et al.* (1993) found that fetal weights were reduced, not after the initial period of heat treatment from day 30 to 80 of pregnancy but at day 140 of pregnancy. These findings support the previous result of other authors (*e.g.*, Yeates, 1958; Alexander and Williams, 1971; Brown *et al.*, 1977) that maternal heat stress during mid- and late pregnancy influences fetal weight.

1.5. MATERNAL NUTRITION AND FETAL GROWTH

There are too few comparative slaughter studies involving undernourished ewes to draw any firm conclusions as to when and how nutrition affects fetal growth during gestation (Robinson, 1982). Studies by Harding and Johnston (1995) concluded that nutrition plays an important role for the development of fetus and correlates closely to lamb birth weight. The role in the regulation of fetal growth can be both direct and indirect. Direct supply of nutrients to provide building blocks for tissue growth is likely to be a minor component in this regulation. Indirect effects of nutrition on fetal endocrine and metabolic status and on the interaction between fetus, placenta, and mother must all be co-ordinated to allow fetal growth.

The effects of reduced maternal nutrition on fetal growth cannot be explained solely by a reduction in the supply of nutrients during the rapid growth phase of fetal development (direct mechanism). The processes of feto-placental metabolic and endocrine adaptation (indirect mechanism) that respond to reduced substrate supply are poorly understood, and their responses to refeeding are even less understood (Harding and Johnston, 1995).

During pregnancy, the mother makes metabolic and growth adjustments (*i.e.*, involving maternal body composition, feed intake, energy consumption, and metabolism) to provide an adequate supply of nutrients for the development of the fetus. Although the mechanism for alterations during pregnancy are not fully established (Jainudeen and Hafez, 1993), recent evidence indicates that the insulin-like growth factors (IGFs) and their binding proteins play important roles in maternal adaptations, to guarantee an adequate supply of substrates to the developing fetus (Owen, 1991).

In the face of inadequate or altered substrate supply, the fetus must alter its metabolic patterns and activity if it is to survive. Part of this adaptation in many instances involves slowing or cessation of growth. Growth is estimated to account for 20-40% of oxygen consumption in the normal fetal sheep (Clapp *et al.*, 1981), and reduction of this expenditure of energy and

substrates may be an important adaptation for survival. However, the mechanisms which mediate this change, are not well understood.

Furthermore, because approximately three-quarters of the ovine fetal growth takes place in the last 50 days of pregnancy and the fact that no reduction in fetal weight has been observed for ewes which lost 7% of their gross body weight during the first 90 days of pregnancy through underfeeding (Wallace, 1948), it was generally assumed that feeding level during this period did not influence fetal growth. The first evidence countering this assumption was the experiment of Everitt (1964). He found that there were 10% and 30% reductions in fetal and functional cotyledon weight, respectively, at day 90 of gestation in single-bearing Merino ewes which as a result of undernutrition, had lost 12% of their body weight in the first three months of pregnancy. Since then, many investigations (e.g., Parr *et al.*, 1982; Parr and Williams, 1982; Mellor, 1983; Parr *et al.*, 1986; McCrabb *et al.*, 1986; Faichney and White, 1987; McCrabb *et al.*, 1992; Harding and Johnston, 1995; Cooper *et al.*, 1998 and Smeaton *et al.*, 1999) have focused on the effects of maternal nutrition during early and/or mid-gestation on placental and fetal development and/ or lamb birth weight.

Rohloff (1984) speculated that placental development in high fertility sheep was quite clearly affected by nutrition in early pregnancy. In turn, this would affect both fetal development and lamb birthweight. Farm survey data, collected by Tarbotton and Webby (1999), have shown a wide variation in apparent fetal survival in high fertility ewes between farms and mobs within farms. This suggests nutrition in early to mid-pregnancy may have affected fetal development, lamb birth weight and survival in high fertility ewes (Smeaton *et al.*, 1999). Recent research by Cooper *et al.* (1998) has supported the speculation of Rohloff (1984), and found that improved maternal nutrition during early and mid-pregnancy enhanced placental weight at day 101 of pregnancy. Placental development between days 30 and 90 of pregnancy is linked to lamb birth weight (Geenty, 1997). By implication, this would therefore affect lamb survival (Dalton *et al.*, 1980; Smeaton *et al.*, 1999). Thus, although recent evidence (Smeaton *et al.*, 1999) has shown that

feeding high allowances to ewes has not influenced fetal development (Smeaton *et al.*, 1999), it is still logical to assume that improved maternal nutrition during early and mid-pregnancy could increase birth weight.

Another study has shown a reduction in weight and size of day-35 embryos removed from ewes that were undernourished (receiving 0.5 times maintenance requirements) (Parr *et al.*, 1986). Edey (1976) also demonstrated small but consistent reductions in birth weight after ewes were undernourished during early to mid-pregnancy.

These studies indicate that there are no firm conclusions which can be drawn as to the effect of maternal nutrition during pregnancy on placental and fetal development, and hence on lamb birth weight. This surely supports that further research in this area is needed to draw more solid conclusions and ultimately recommendations for farmers.

1.6. PURPOSE AND SCOPE OF THE STUDY

Many practitioners have proposed that greater fetal weights can arise from an enhancement of placental development due to improved maternal nutrition during the first 100 days of pregnancy. If maternal nutrition is important to enhance placental growth, insufficient nutrition in early and mid-pregnancy (up to day 100 of pregnancy) could retard fetal development in the later stages of pregnancy and hence decrease lamb weights at birth. Manipulating maternal nutrition (by altering pasture allowances) could be used as a method to move multiple-born lambs into the desired "optimum" birthweight range. This strategy could benefit the survival of multiple-born lambs. This experiment was therefore designed to examine the effect of different nutritional treatments during early and mid-gestation on placental and fetal development. It also investigated whether fetal growth during the last third of gestation (P100-140) could be affected by altering nutritional levels in either early or mid-pregnancy, *i.e.*, whether early pregnancy nutrition

could alter the potential for fetal growth in later pregnancy by changing the upper limit to placental development.

CHAPTER TWO

THE EFFECT OF MANIPULATION OF FEED INTAKE DURING PREGNANCY ON LAMB BIRTH WEIGHT

2.1. INTRODUCTION

Lamb birth weight is the dominant factor in survival of both singles and multiples (Hinch *et al.*, 1986). This is because excessively high or low birth weights are generally associated with an increase in neonatal mortality rates. Dams carrying large offspring are susceptible to dystocia, putting the life of both offspring and dam at risk (Laster and Gregory, 1973), while neonates with low birth weights suffer from exposure and starvation (McCutcheon *et al.*, 1981; Cooper *et al.*, 1998).

It has been established that lamb birth weight is partly associated with ewe nutrition during pregnancy. However, studies examining the correlation between maternal nutrition and fetal growth (and hence birth weight) are not consistent. For example, low nutrition during pregnancy reduced lamb birth weight up to 25% (Thomson and Thomson, 1948; Smeaton *et al.*, 1983), and restricted mammary growth and development (Russel *et al.*, 1977). Moreover, severe under-nutrition during early and mid-pregnancy (days 0 to 100) resulted in reduced placental weights (McCrabb *et al.*, 1992) and lamb birth weight (Everitt, 1967). Conversely, increased nutrition during mid- or late pregnancy did not significantly influence birth weight, or lamb survival between birth and two weeks of age (Kleemann *et al.*, 1993).

Productive performance of grazing ruminants is closely related to herbage intake (Rattray *et al.*, 1987). For grazing sheep, herbage intake and hence production are determined, in part, by how much herbage is offered and by the intensity of grazing (measured, for example, by the post grazing herbage mass) (Morris *et al.*, 1994). A recent study showed that feeding ewes above maintenance requirements during the first 101 days of pregnancy increased placental and fetal weight (Cooper *et al.*, 1998). If poor nutrition during early

and mid-gestation decreases development of the placenta in pregnant ewes leading to small lambs at birth, manipulation of ewe herbage intake during pregnancy, by offering different pasture allowances, could result in differences in ewe performance. Therefore, increased feed intake during early and mid-pregnancy could promote good placental growth and satisfactory lamb birth weights and hence survival of multiples.

There have been few studies carried out on the effect of manipulation of feed intake during the entire pregnancy period on lamb birth weight. The primary objective of this study was to investigate the effects of different nutritional treatments during early and mid-gestation on placental and fetal development. A secondary objective was to determine whether fetal growth during the last third of gestation could be affected by altering nutritional levels in either early or mid-pregnancy.

2.2. MATERIALS AND METHODS

2.2.1. Experimental Design and Animals

The objective of the study was to compare the effects of differential ewe feeding as illustrated in Figure 2. Three periods were involved: pregnancy days 19-47 (P19-P47) (mating to pregnancy diagnosis); P47-P102 (the latter date being equivalent to the time at which the study of Cooper *et al.* (1998) terminated); and P102-P136 (*i.e.*, until near term). One group of ewes was to be slaughtered at P102 (to test the effect of differential nutrition on fetal growth to that time) and the other at P136 (to test whether the effects of differential nutrition over P102-P136 were influenced by nutritional history to that point).

One hundred and sixty mixed-aged (three to six years) Romney ewes were mated at a synchronised oestrus (Eazi-breed CIDR Type G, Carter Holt Harvey Plastic Products, Hamilton, New Zealand) on 13 March 1998. Only ewes pregnant to the first synchronised oestrus were subsequently used in the study. At day 19 of pregnancy (P19), 136 ewes were randomly allocated to two nutritional treatments, either “high” (average initial live weight =

55.3±1.5 kg) or “medium” (average initial live weight 54.5±1.5 kg). These groups were fed from P19-47 at either 1.5 or 1.0 times maintenance for the high (H) and medium (M) treatments, respectively, the latter representing the “industry norm”. Maintenance requirements for a 55 kg ewe were assumed to be 11 MJ ME/day (Geenty and Rattray, 1987) and, at an energy concentration of 10 MJ ME/kg DM, the target levels of feed consumption were therefore 1.10 and 1.65 kg DM/ewe/day for the M and H treatment groups respectively.

At P47 the ewes were pregnancy diagnosed by using real-time ultrasound scanning (Carter, 1987), and single or multiple pregnancy was confirmed at this time. Initially it was the intention to use twin-bearing ewes but only 59 twin-bearing ewes were obtained from the first cycle of mating. Therefore, 30 single-bearing ewes were added to give a sufficient treatment number (n=89).

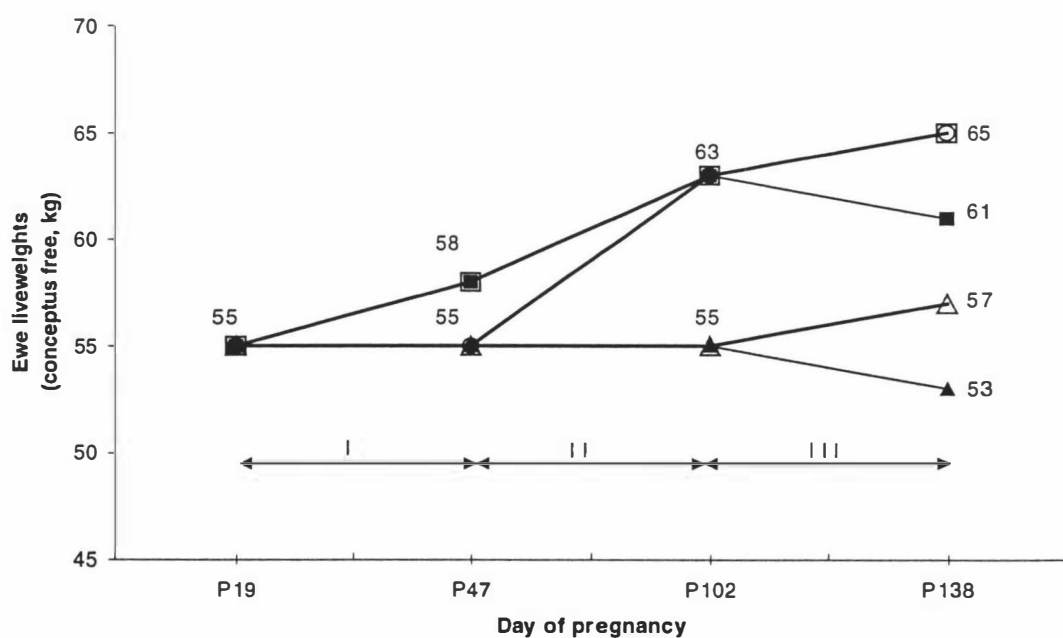


Figure 2. Diagram of target ewe live weights for each of the treatments.

▲ = MMM; △ = MMH; ● = MHM; ○ = MHH; ■ = HHM; and □ = HHH.

The 89 ewes were weighed and randomly allocated to three treatments (*i.e.*, MM = 21 twins, 10 singles; MH = 21 twins, 10 singles; and HH = 17 twins, 10 singles). At P102-104, 10 ewes from each treatment were slaughtered while the remaining ewes in each of the treatment groups (*i.e.*, MM=21; MH=21; and HH=17 ewes) were further subdivided into groups receiving either high or medium feeding levels for the next 34 days of pregnancy until P136, when they were slaughtered.

Two ewes died, three ewes were culled because they lost considerable live weight (presumably due to subclinical facial eczema), and one ewe was found to have quadruplets. Those animals were, therefore, subsequently excluded from the data analysis.

2.2.2. Pasture Preparation

The pastures used were composed principally of ryegrass (*Lolium perenne*) and white clover (*Trifolium repens*). Before P19, the pastures were prepared over the previous 6 weeks using a non-trial group of ewes. The two treatment groups were set stocked on pasture using electric fencing to subdivide the paddocks and maintain allowances at 1.0 and 1.5 times maintenance for the M and H group respectively. Five paddocks (comprising one each of 0.2, 0.6, 0.8 ha and two 1.0 ha paddocks with pasture masses ranging from 1000 to 1300 kg DM ha⁻¹) were rotationally grazed by the M group during the period P19-102. The M group was then moved to a 4.0 ha paddock with higher a pasture cover (average mass 1500 kg DM ha⁻¹) as herbage in the previous paddocks did not cover the increasing requirements of ewe maintenance and fetal growth over the period P102-136. Three paddocks (respective areas of 1.0, 1.8 and 2 ha paddocks with pasture masses ranging from 2600 to 3300 kg DM ha⁻¹) were rotationally grazed by the H group over the entire trial. Pasture cover was monitored by using an Ellinbank Pasture Meter, EPM (Earle and McGowan, 1979), where fifty readings were taken by the EPM on a diagonal path within each paddock (Morris *et al.*, 1994).

2.2.3. Animal Measurements

The ewes were weighed straight off pasture between 0800-1100 h at weekly intervals from day 19 to 131 of gestation. Organic matter intake of herbage of individual ewes was determined by the indirect method using the *in vitro* herbage digestibility (D) of organic matter (OM), and faecal output of grazing animals (FO, g/d OM). A single chromic oxide controlled release capsule (CRC) (3.0-cm core of pressed tablet, 65% Cr₂O₃ and 9 mm orifice, Captec (NZ) Ltd., Auckland) was inserted at P73. A second capsule was inserted at P108. For the first insertion, ewes were faecal sampled during P80-84 and P87-91. At the second insertion, faecal collection occurred from P115-119 and P128-132. The rate of chromic oxide release from the capsule was assumed to be 138 mg/d (Parker *et al.*, 1989). Faecal samples, bulked over five day periods, were oven-dried to a constant weight (at 70°C for 72h) and chromium concentration was assessed using atomic absorption spectrophotometry by dividing the rate of the chromium release from the CRC by the concentration of chromium in the faeces (mg Cr/g OM).

Four oesophageal-fistulated (OF) wethers were used to collect herbage samples on two occasions during each faecal collection period (*i.e.*, on P77-90 and P120-130) for determining the *in vitro* digestibility of herbage consumed. Extrusa samples taken from each OF sheep and each treatment were immediately placed on crushed ice and stored at -12°C (Morris *et al.*, 1993) until required for analysis. Samples were freeze-dried and then ground through a 1.0-mm sieve in preparation for *in vitro* digestibility determination using the cellulose incubation method described by Roughan and Holland (1977).

2.2.4. Slaughter Procedure

Final ewe live weights were determined approximately 12 hrs prior to the first day of slaughter and, from then on, each slaughter group was weighed immediately prior to slaughter (Cooper, 1998). At the first slaughter (P102-104), six single- and four twin-bearing ewes from each treatment group (*i.e.*, 18 single-bearing ewes and 12 twin-bearing ewes) were randomly allocated

to daily slaughter groups, thereby ensuring that the treatment groups were balanced across the slaughter dates. At the second slaughter (P136-140), 41 ewes slaughtered were carrying twins and other 12 were single-bearing ewes. The ewes were slaughtered by captive bolt pistol and exsanguination. Slaughter was conducted between 0800 and 1700 h, over a three-day period for the first slaughter group and over a five-day period for the second slaughter group (*i.e.*, on P102-104 and P136-140 respectively). Following exsanguination the procedure followed that of Cooper (1998), *i.e.* the abdominal cavity was opened and the gravid uterus removed. The cervix was trimmed off and the ovaries removed, following which the uterus was weighed, giving total gravid uterus weight. The allantoic and amniotic fluids were removed through an incision made along the greater curvature of the pregnant horn and discarded.

The fetus(es) were removed from the uterus and the umbilical cord ligated at the abdomen before being severed. Each fetus was gently squeezed by hand to remove amniotic fluid, and fetal number, weight and sex were then recorded. Any fetuses still alive were euthanased by intra-cardiac injection of sodium pentobarbitone (Pentobarb 500, Chemstock Animal Health, Christchurch, New Zealand). Fetal curved crown-rump length (with the fetus lying in a "relaxed" position) and chest circumference were then recorded (Cooper, 1998). Fetal volume was measured as the water displaced from a full bucket of water after the fetus was immersed and gently removed from the bucket. Selected fetal organs such as heart, liver, lungs, kidneys, adrenal glands and thyroid glands were also recorded.

Placentomes were dissected from the uterus using curved scissors, separated manually into their maternal (caruncle) and fetal (cotyledon) components, and their individual weights recorded. The myoendometrium and the fetal membranes were weighed. The total weight of the caruncles and cotyledons was used in this study as an index of placental size, and is subsequently referred to as 'placental weight' (Cooper, 1998). Ewe carcasses and pelt weights were also recorded.

2.2.5. Statistical Analysis

Analysis of variance was used to determine the effects of treatment during pregnancy on ewe live weights using liveweight at P19 as a covariate. Intake of dry and organic matter, weights of uterine components, numbers of placentomes, weights of fetal organs, fetal weights and size were adjusted to a common pregnancy rank (single vs. twin), to test for the effects of ewe pregnancy feeding on those parameters. Data are expressed as least square means and standard errors for treatment groups and fetuses. Statistical analyses were conducted using the computer package 'SAS' (SAS, 1988).

2.3. RESULTS

2.3.1. Ewe Intake during Early and Mid-Pregnancy

Ewe intakes give an indication as to the success of the pre-planned liveweight targets set for the nutritional treatments. Feed intakes for two periods (P80-84 and P87-91) were estimated to ensure pre-planned feeding levels were met. Table 1 shows the effect of ewe nutrition treatment from P47-102 and pregnancy rank on intake of dry matter (DMI) and digestible organic matter (DOMI) by ewes at the two indicated periods during pregnancy.

Ewes receiving a high (H) feeding allowance during P19-47 and P47-102 (HH) had significantly ($P < 0.05$) higher DMI and DOMI during P80-84 than ewes offered a maintenance (M) feeding level during P19-47 and P47-102 (MM) or ewes offered a maintenance (M) feeding level during P19-47 and offered a high (H) feeding level during P47-102 (MH). During the period P87-91 ewes fed MM had higher DMI than those ewes fed MH or HH levels of nutrition.

Table 1. The effect of pre-partum nutrition (from P47-102) and pregnancy rank on intake of dry (DM) and digestible organic matter (DOM) by ewes at two periods during pregnancy (mean±s.e.m.). Means within columns having superscripts with letters in common or no superscript are not significantly different ($P > 0.05$).

intake	Number	Dry matter intake		Digestible organic matter ¹	
		(kg DM ewe ⁻¹ day ⁻¹)		(kg DOM ewe ⁻¹ day ⁻¹)	
		P80-84	P87-91	P80-84	P87-91
Treatment					
MM ¹	16	0.98±0.07 ^a	1.24±0.06 ^b	0.46±0.04 ^a	0.47±0.03 ^a
MH	15	1.02±0.07 ^a	1.05±0.06 ^a	0.63±0.04 ^b	0.62±0.03 ^b
HH	11	1.22±0.06 ^b	1.10±0.07 ^a	0.75±0.04 ^c	0.61±0.03 ^b
Pregnancy Rank					
1	8	1.18±0.09 ^b	1.19±0.08	0.68±0.05 ^b	0.61±0.52
2	34	0.97±0.04 ^a	1.08±0.04	0.55±0.02 ^a	0.53±0.02

¹ MM ewes received a maintenance (M) feeding level during periods I (P19-47) and II (P47-102); MH ewes received M feeding level during period I and a high (H) feeding level during period II; HH ewes received H feeding level during periods I and II.

Ewes carrying a single fetus had significantly ($P < 0.05$) higher DMI and DOMI than ewes carrying twin fetuses (1.18±0.09 v. 0.97±0.04 kg DM ewe⁻¹ day⁻¹ or 0.68±0.05 v. 0.55±0.02 kg DOM ewe⁻¹ day⁻¹, respectively) during P80-84. Pregnancy rank had no effect on DMI and DOMI during P84-91 although intake of dry matter and DOM of ewes carrying a single fetus tended to be higher than that of ewes carrying twin fetuses (Table 1).

2.3.2. Ewe Intake during Late Pregnancy

Intake estimates from P115-119 and P128-132 indicated that ewe nutritional treatments had significant ($P < 0.05$) effects on DMI and DOMI. The exception was for DMI during P128-132, where there were no differences ($P > 0.05$). However, the pattern of DMI during this period was similar to the pattern of

DMI during P115-119. Ewes receiving M feeding levels during the period from P102 to P136 had significantly higher DMI during P115-119 than those ewes receiving H feeding levels. However this result was reversed for ewe intake of DOM. Ewes receiving M feeding levels during period P102 to P136 of gestation had significantly ($P < 0.05$) lower DOMI than those receiving H feeding levels during both P115-119 and P128-132 (Table 2).

Ewes carrying a single fetus tended to have higher DMI and DOMI compared with ewes carrying twin fetuses during P115-119 and P128-132. This effect of pregnancy rank on DMI and DOMI was significant only during the period P128-132 and then only for DOMI ($P < 0.05$, Table 2).

Table 2. The effect of pre-partum nutrition (from P102-136) and pregnancy rank on intake of dry and digestible organic matters by ewes at two periods during pregnancy (mean \pm s.e.m.). Means within columns having superscripts with letters in common or no superscript are not significantly different ($P > 0.05$).

	Number	Dry matter intake (kg DM ewe ⁻¹ day ⁻¹)		Digestible organic matter intake (kg DOM ewe ⁻¹ day ⁻¹)	
		P115-119	P128-132	P115-119	P128-132
Treatment					
MMM ¹	8	1.66 \pm 0.13 ^d	1.21 \pm 0.09	0.58 \pm 0.06 ^{ac}	0.50 \pm 0.05 ^a
MMH	9	1.00 \pm 0.12 ^a	1.03 \pm 0.09	0.66 \pm 0.06 ^{bc}	0.69 \pm 0.04 ^{cd}
MHM	9	1.40 \pm 0.13 ^{bcd}	1.08 \pm 0.09	0.49 \pm 0.06 ^a	0.47 \pm 0.04 ^a
MHH	9	1.30 \pm 0.12 ^{abc}	1.23 \pm 0.08	0.89 \pm 0.06 ^d	0.80 \pm 0.04 ^d
HHM	8	1.53 \pm 0.13 ^{cd}	1.21 \pm 0.10	0.52 \pm 0.07 ^{ab}	0.53 \pm 0.05 ^a
HHH	6	1.10 \pm 0.15 ^{ab}	0.97 \pm 0.10	0.72 \pm 0.07 ^{cd}	0.67 \pm 0.05 ^{bcd}
Pregnancy Rank					
1	11	1.36 \pm 0.11	1.19 \pm 0.08	0.67 \pm 0.05	0.66 \pm 0.04 ^b
2	38	1.31 \pm 0.06	1.05 \pm 0.04	0.62 \pm 0.03	0.55 \pm 0.02 ^a

¹Three letters indicate three periods of pregnancy (*i.e.*, the first letter = a period from day 19 to 47; the second letter = a period from day 47 to day 102; and the third letter = a period from day 102 to day 136); M and H represent feeding treatments of 1.0 and 1.5 times maintenance requirements.

2.3.3. Ewe Live Weight Changes, and Placental and Fetal Development during Early and Mid-Pregnancy.

Table 3 shows the changes in live weight for the first group of ewes from day 19 (end of mating) through to day 102 when this group was slaughtered. The change in ewe live weight from P19 to P47 for maintenance (M)-fed ewes was -1.3 kg while the high (H) ewes treatments increased by 3.2 kg (Table 3, note that MM and MH ewes are averaged as ewes were run together during this period). From P47, when the M ewes were further divided into MM and MH groups, ewe liveweight changes to P102 for MM, MH, and HH treatments were 3.8 , 5.8 , and 2.7 kg, respectively (Table 3).

Table 3. The effect of pre-partum nutrition on ewe live weights (kg) at different stages of pregnancy ¹ (means \pm s.e.m). P19 = day 19 of pregnancy; Means within rows having superscripts with letters in common or no superscript are not significantly different ($P > 0.05$).

Days of pregnancy	Treatment		
	MM ²	MH	HH
Number	10	10	10
P 19	53.8 \pm 2.6	55.1 \pm 2.6	55.6 \pm 2.6
P 26	54.5 \pm 0.7 ^a	54.6 \pm 0.7 ^a	59.0 \pm 0.7 ^b
P 33	55.5 \pm 0.6 ^{ab}	55.0 \pm 0.6 ^a	57.2 \pm 0.6 ^b
P 40	53.8 \pm 0.7 ^a	53.3 \pm 0.7 ^a	58.0 \pm 0.7 ^b
P 47	53.1 \pm 0.7 ^a	53.2 \pm 0.7 ^a	58.8 \pm 0.7 ^b
P 54	53.3 \pm 0.8 ^a	56.1 \pm 0.7 ^b	59.5 \pm 0.8 ^c
P 61	54.8 \pm 0.8 ^a	57.3 \pm 0.8 ^b	59.8 \pm 0.8 ^c
P 68	55.0 \pm 0.9 ^a	57.0 \pm 0.9 ^a	60.1 \pm 0.9 ^b
P 75	55.8 \pm 1.0 ^a	58.0 \pm 1.0 ^a	61.0 \pm 1.0 ^b
P 82	55.0 \pm 1.0 ^a	57.6 \pm 1.0 ^a	60.9 \pm 1.0 ^b
P 89	55.3 \pm 1.0 ^a	56.4 \pm 1.0 ^a	60.2 \pm 1.1 ^b
P 96	56.6 \pm 1.2 ^a	56.8 \pm 1.2 ^a	60.4 \pm 1.2 ^b
P 102	56.9 \pm 1.4 ^a	59.0 \pm 1.4 ^{ab}	61.5 \pm 1.3 ^b

¹ first slaughtered group of animals

² for definition see Table 1

Table 4 presents the changes in ewe live weights at different stages of pregnancy in respect to different litter sizes. Pregnancy rank (single and twin) had no effect on ewe live weights recorded from day 19 through to day 102 of pregnancy. Ewe live weights at the end of this period were 59.0 ± 1.0 and 59.2 ± 1.2 kg (*i.e.* increased by 5.0 and 3.6 kg from the initial ewe live weights at P19, Table 4) for ewes carrying single- and twin- fetus(es), respectively.

Table 4. The effect of pregnancy rank on ewe live weights (kg) at different stages of pregnancy ¹ (means \pm s.e.m). P19 = day 19 of pregnancy.

Days of pregnancy	Pregnancy rank	
	Single	Twin
Number	19	11
P19	54.0 \pm 1.9	55.6 \pm 2.5
P 26	56.1 \pm 0.5	56.0 \pm 0.7
P 33	56.1 \pm 0.4	55.7 \pm 0.6
P 40	55.0 \pm 0.5	55.1 \pm 0.6
P 47	55.2 \pm 0.5	54.9 \pm 0.7
P 54	56.3 \pm 0.5	56.3 \pm 0.7
P 61	57.4 \pm 0.6	57.2 \pm 0.7
P 68	57.6 \pm 0.6	57.1 \pm 0.8
P 75	58.3 \pm 0.7	58.2 \pm 0.9
P 82	57.5 \pm 0.7	58.1 \pm 1.0
P 89	57.2 \pm 0.7	57.4 \pm 1.0
P 96	57.8 \pm 0.9	58.1 \pm 1.2
P 102	59.0 \pm 1.0	59.2 \pm 1.2

¹ first slaughtered group of animals only.

Table 5 shows the effect of pre-partum nutrition (from P47 to P102) and pregnancy rank on final ewe live weights, weights of carcass, pelt and placental components, and placentome and caruncle numbers at day 102-104 (period slaughter date). The thirty ewes, which were slaughtered over a three-day period to compare with the previous study by Cooper *et al.* (1998),

carried 19 singles and 11 sets of twins. H feeding levels significantly increased ($P<0.05$) both final live weights and carcass weights but not pelt weights. None of placental parameters was affected by pre-partum nutrition from day 19 to day 102 of gestation.

Pelt weights were significantly ($P<0.05$) affected by pregnancy rank although final live weights and carcass weights were not. Other components, such as weights of the gravid uterus, uterus, myoendometrium, fetal membranes, total placentome and total cotyledon were significantly ($P<0.05$) heavier in twins than in singles. Ewes carrying twin fetuses had significantly ($P<0.05$) more placentomes and tended to have more caruncles than single-bearing ewes. Caruncle occupancy (*i.e.*, number of placentomes/number of caruncles) was significantly ($P<0.05$) higher in twins than in singles (87% v. 80%, respectively).

Table 5. The effect of pre-partum nutrition and pregnancy rank on: final ewe live weights; weights of carcass, pelt and placental components; and placentome and caruncle numbers at day 102-104 of pregnancy (mean±s.e.m.). Means within rows and main effects having superscripts with letters in common or no superscript are not significantly different ($P > 0.05$).

	Treatment			Pregnancy Rank	
	MM ¹	MH	HH	1	2
Number	10	10	10	19	11
Weights:					
Final live weight (kg)	56.0 ± 1.9 ^a	56.6 ± 1.8 ^{ab}	60.0 ± 1.2 ^b	57.3 ± 0.9	57.8 ± 1.1
Carcass (kg)	24.8 ± 0.8 ^{ab}	23.3 ± 0.8 ^a	26.7 ± 0.9 ^b	25.1 ± 0.6	24.7 ± 0.8
Pelt (kg)	5.4 ± 0.4	6.4 ± 0.4	6.0 ± 0.4	6.5 ± 0.3 ^b	5.4 ± 0.3 ^a
Mammary gland (g)	352.6 ± 33.0	346.9 ± 33.0	384.0 ± 33.7	350.8 ± 23.8	371.8 ± 31.3
Gravid uterus (g)	4404.2 ± 192.4	4252.2 ± 192.4	4244.4 ± 196.5	3272.0 ± 138.8 ^a	5086.5 ± 182.7 ^b
Uterus (g)	1357.7 ± 77.6	1280.2 ± 77.6	1466.9 ± 79.3	1186.8 ± 56.0 ^a	1549.8 ± 73.7 ^b
Myoendometrium (minus caruncles) (g)	385.6 ± 18.3	379.9 ± 18.3	426.4 ± 18.7	340.4 ± 13.2 ^a	454.2 ± 17.4 ^b
Fetal membrane (minus cotyledons) (g)	221.0 ± 16.8	234.7 ± 16.8	220.6 ± 17.1	181.9 ± 12.1 ^a	268.9 ± 15.9 ^b
Total placentome (g)	658.0 ± 49.5	612.1 ± 49.5	676.7 ± 50.6	577.2 ± 35.7 ^a	720.6 ± 47.0 ^b
Average placentome (g)	6.2 ± 0.5	5.9 ± 0.5	6.7 ± 0.5	6.2 ± 0.4	6.4 ± 0.5
Total caruncle (g)	132.0 ± 17.7	130.6 ± 17.7	130.8 ± 18.1	134.9 ± 12.8	127.3 ± 16.8
Average caruncle (g)	1.1 ± 0.1	1.0 ± 0.1	1.1 ± 0.1	1.1 ± 0.1	1.0 ± 0.1
Total cotyledon (g)	526.4 ± 37.6	482.8 ± 37.6	547.1 ± 38.4	443.6 ± 27.1 ^a	593.9 ± 35.7 ^b
Average cotyledon (g)	4.9 ± 0.4	4.6 ± 0.4	5.4 ± 0.4	4.8 ± 0.3	5.2 ± 0.4
Number:					
Total placentomes	107.2 ± 5.9	105.3 ± 5.9	101.7 ± 6.0	97.3 ± 4.3 ^a	112.2 ± 5.6 ^b
Total caruncles	124.3 ± 7.7	130.0 ± 7.7	124.8 ± 7.9	122.4 ± 5.6	130.3 ± 7.3
Caruncle occupancy ²	0.87 ± 0.03	0.81 ± 0.03	0.82 ± 0.03	0.80 ± 0.02 ^a	0.87 ± 0.03 ^b

¹ for definition see Table 1.

² Number of placentomes/number of caruncles.

The fetal weights, crown rump length, girth, fetal volume, and the weights of fetal heart, liver, lungs, kidneys, adrenal and thyroid glands at day 102-104 of gestation (period slaughter date) are presented in Table 6. None of these parameters was affected by pre-partum nutrition during early and mid pregnancy (from day 19 to day 102 of gestation).

Table 6. The effect of pre-partum nutrition on fetal weight, crown rump length, girth, volume and organ weights at day 102-104¹ of pregnancy (means±s.e.m).

Treatment	MM ²	MH	HH
	Number	14	14
Fetal weight (g)	1281.7 ± 50.4	1296.0 ± 50.8	1258.2 ± 53.4
Crown rump length (CRL) (cm)	35.9 ± 0.5	35.7 ± 0.5	36.3 ± 0.5
Girth (cm)	23.3 ± 0.4	23.5 ± 0.4	23.3 ± 0.4
Fetal volume (g)	1038.4 ± 60.2	1172.1 ± 63.5	1074.4 ± 63.5
Fetal organ weights:			
• Heart (g)	10.6 ± 0.5	10.2 ± 0.5	10.0 ± 0.6
• Liver (g)	63.8 ± 3.3	63.5 ± 3.3	60.2 ± 3.5
• Lungs (g)	48.0 ± 2.4	46.6 ± 2.4	46.8 ± 2.6
• Kidneys (g)	11.9 ± 0.4	11.4 ± 0.4	11.5 ± 0.5
• Adrenal glands (g)	0.2 ± 0.0	0.2 ± 0.0	0.2 ± 0.0
• Thyroid glands (g)	0.8 ± 0.2	1.3 ± 0.2	1.4 ± 0.3

¹ first slaughtered group of animals.

² for definition see Table 1.

2.3.4. Ewe Live Weight Changes, and Placental and Fetal Development during Late Pregnancy.

Table 7 shows results for the second slaughter group of ewes from day 19 through to day 131 of gestation. Liveweight decreased by 0.9 kg for the M (*i.e.*, MMM, MMH, MHM and MHH) groups, and increased by 5.3 kg for the H (*i.e.*, HHM and HHH) groups over the period P19-47.

From P47 to P102, the MM (*i.e.*, MMM and MMH) groups increased by 4.3 kg (from average 54.1 kg at P47 to average 58.4 kg at P102), the MH (*i.e.*, MHM and MHH) groups increased by 7.1 kg (from average 54.2 kg at P47 to average 61.3 kg at P102), and the HH (*i.e.*, HHM and HHH) groups increased by 1.9 kg (from average 61.4 kg at P47 to average 63.3 kg at P102), respectively.

Over the period P102 to P131, there was an increase in ewe live weight of 3.3, 6.0, 2.4, 7.3, 1.4 and 6.9 kg for the MMM, MMH, MHM, MHH, HHM and HHH groups, respectively.

Table 7. The effect of pre-partum nutrition on ewe live weights (kg) at different stages of pregnancy ¹ (means±s.e.m). P19 = day 19 of pregnancy; Means within rows and main effects having superscripts with letters in common or no superscript are not significantly different ($P > 0.05$).

Days of pregnancy	Treatment					
	MMM ²	MMH	MHM	MHH	HHM	HHH
Number	8	10	10	10	8	7
P 19	57.5±2.8	52.9±2.5	54.1±2.7	55.5±2.5	54.6±2.9	57.7±3.0
P 26	55.0±0.6 ^a	56.0±0.5 ^a	55.8±0.6 ^a	55.2±0.5 ^a	59.7±0.6 ^b	60.3±0.6 ^b
P 33	56.1±0.5 ^a	56.4±0.5 ^a	55.8±0.5 ^a	55.8±0.5 ^a	57.9±0.5 ^b	58.7±0.6 ^b
P 40	53.8±0.6 ^a	54.7±0.6 ^a	54.3±0.6 ^a	54.0±0.6 ^a	59.0±0.7 ^b	58.9±0.7 ^b
P 47	54.0±0.6 ^a	54.2±0.6 ^a	54.3±0.6 ^a	54.1±0.6 ^a	63.3±0.7 ^b	59.6±0.7 ^b
P 54	54.1±0.7 ^a	55.4±0.6 ^{ab}	57.1±0.7 ^b	57.1±0.6 ^b	60.2±0.7 ^c	60.7±0.7 ^c
P 61	55.2±0.7 ^a	55.9±0.6 ^a	58.4±0.7 ^b	57.8±0.6 ^b	61.2±0.7 ^c	60.8±0.8 ^c
P 68	55.6±0.8 ^a	56.5±0.7 ^a	58.6±0.7 ^{bc}	58.4±0.7 ^b	60.5±0.8 ^{cd}	60.9±0.8 ^d
P 75	57.0±0.8 ^a	57.2±0.8 ^{ab}	58.7±0.8 ^{ab}	59.3±0.8 ^b	61.9±0.9 ^c	61.5±0.9 ^{bc}
P 82	55.9±0.9 ^a	56.7±0.8 ^{ab}	58.7±0.9 ^{bc}	58.4±0.8 ^{bd}	61.1±1.0 ^c	60.9±1.0 ^{cd}
P 89	56.1±1.0 ^a	56.2±0.9 ^a	58.5±0.9 ^{ab}	58.6±0.9 ^{ab}	61.1±1.0 ^{bc}	61.6±1.1 ^c
P 96	57.2±1.1 ^a	57.2±1.0 ^a	58.8±1.1 ^{ab}	59.4±1.0 ^{ab}	61.4±1.1 ^b	60.8±1.3 ^b
P 102	58.4±1.2 ^a	58.5±1.1 ^a	61.5±1.1 ^{ab}	61.2±1.1 ^{ab}	63.3±1.2 ^b	63.4±1.3 ^b
P 110	59.5±1.3 ^a	60.4±1.2 ^a	60.3±1.2 ^a	62.7±1.1 ^{ab}	62.0±1.3 ^{ab}	64.6±1.4 ^b
P 117	58.9±1.2 ^a	62.1±1.1 ^a	59.8±1.2 ^a	65.2±1.1 ^b	61.3±1.3 ^a	66.6±1.3 ^b
P 124	59.8±1.4 ^a	64.2±1.2 ^b	62.2±1.3 ^{ab}	67.8±1.2 ^c	63.0±1.4 ^{ab}	69.2±1.5 ^c
P 131	61.7±1.5 ^a	64.5±1.3 ^a	63.9±1.4 ^a	69.5±1.3 ^b	64.7±1.5 ^a	70.3±1.5 ^b

¹ second slaughtered group of animals.

² for definition see Table 2.

Pregnancy rank had no effect on ewe live weights. The changes in ewe live weights resulting from the effect of pregnancy rank (*i.e.*, 31 and 52 ewes carrying single and twin fetus(es), respectively) are presented in Table 8.

Table 8. The effect of pregnancy rank on ewe live weights (kg) at different stages of pregnancy (means \pm s.e.m). P19 = day 19 of pregnancy.

Days of pregnancy	Pregnancy rank	
	Single	Twin
Number	31	52
P 19	54.4 \pm 1.4	55.7 \pm 1.1
P 47	55.9 \pm 0.3	55.4 \pm 0.3
P 102	60.0 \pm 0.7 (19) ¹	61.0 \pm 0.5 (11) ¹
P 131	65.2 \pm 1.2 (12) ¹	66.3 \pm 0.6 (41) ¹

¹ number of animals slaughtered.

Final ewe live weights, weights of carcass, pelt, placental components, and numbers of placentomes and caruncles were observed at day 136 of gestation, and are presented in Table 9. Pre-partum nutritional treatments significantly ($P < 0.05$) affected final ewe live weights, carcass weights and pelt weights. There were no significant effects of pre-partum nutritional treatments on placental weights and placental numbers except for fetal membrane weights. The data in Table 9 do indicate a slight trend whereby ewes fed the H feeding level at any period of gestation tended to have greater weights of mammary glands, gravid uterus, uterus, and myoendometrium. However, there was also a trend that ewes fed the H feeding level at any period of gestation tended to have lower placentome weights and cotyledon weights.

Pregnancy rank had no effects on final ewe live weights or pelt weights but did affect carcass weights. Carcasses of ewes carrying a single fetus were significantly ($P<0.05$) heavier than those of ewes carrying twins. However, single ewes had significantly ($P<0.05$) lower weights of mammary glands, uterus, myoendometrium, fetal membrane, total placentomes, and total cotyledons, and had fewer placentomes compared to ewes carrying twins. Weights of gravid uterus, total caruncle weights and total caruncle number were not affected by pregnancy rank although they showed a tendency to be increased in weight or number in twins as observed at day 136 of gestation (Table 9).

Table 9. The effect of pre-partum nutrition and pregnancy rank on final ewe live weights and weights of carcass, pelt and placental components, and placentome and caruncle numbers at day 136-140 of pregnancy (mean±s.e.m.). Means within rows and main effects having superscripts with letters in common or no superscript are not significantly different ($P > 0.05$)

	Treatment						Pregnancy Rank	
	MMM ¹	MMH	MHM	MHH	HHM	HHH	1	2
Number	8	10	10	10	8	7	12	41
Weights:								
Final live weight (kg)	61.3 ± 1.7 ^a	63.3 ± 1.5 ^{abc}	62.0 ± 1.6 ^a	67.1 ± 1.5 ^{bc}	63.0 ± 1.5 ^{abc}	67.3 ± 1.8 ^c	63.1 ± 1.4	64.8 ± 0.7
Carcass (kg)	22.5 ± 0.9 ^a	23.4 ± 0.8 ^{ab}	23.6 ± 0.8 ^{ab}	25.9 ± 0.8 ^{cd}	25.3 ± 0.9 ^{bc}	28.0 ± 1.0 ^d	25.9 ± 0.7 ^b	23.7 ± 0.4 ^a
Pelt (kg)	7.0 ± 0.5 ^{ab}	6.5 ± 0.5 ^{ab}	5.9 ± 0.5 ^a	7.4 ± 0.5 ^b	6.3 ± 0.5 ^{ab}	7.1 ± 0.6 ^{ab}	7.0 ± 0.4	6.4 ± 0.2
Mammary gland (g)	932.0 ± 130.7	904.8 ± 116.3	952.5 ± 123.4	1113.3 ± 116.3	869.3 ± 135.0	1057.1 ± 138.4	798.4 ± 105.7 ^a	1144.7 ± 56.9 ^b
Gravid uterus (g)	10501.8 ± 724.1	10908.7 ± 644.2	11291.8 ± 684.4	11223.8 ± 644.2	9617.6 ± 747.7	10995.9 ± 766.7	8453.5 ± 585.7	13059.8 ± 315.2
Uterus (g)	1867.1 ± 152.9	1840.5 ± 136.0	1931.2 ± 144.6	1955.8 ± 136.0	1779.4 ± 157.9	1992.2 ± 161.9	1625.4 ± 123.7 ^a	2163.3 ± 66.6 ^b
Myoendometrium (minus caruncles) (g)	767.0 ± 56.9	728.6 ± 50.6	710.0 ± 53.7	786.1 ± 50.6	751.2 ± 58.7	757.4 ± 60.2	619.2 ± 48.0 ^a	880.8 ± 24.8 ^b
Fetal membrane (minus cotyledons) (g)	365.0 ± 39.2 ^a	395.0 ± 34.9 ^{ab}	463.5 ± 37.1 ^{bc}	387.5 ± 34.9 ^a	358.3 ± 40.5 ^a	509.3 ± 41.5 ^c	371.4 ± 31.7 ^a	455.1 ± 17.1 ^b
Total placentome (g)	583.2 ± 81.9	545.8 ± 72.8	602.3 ± 77.4	551.5 ± 72.8	622.5 ± 84.6	547.3 ± 86.7	490.6 ± 66.2 ^a	660.2 ± 35.6 ^b
Average placentome (g)	6.4 ± 0.7	5.2 ± 0.6	5.2 ± 0.7	5.6 ± 0.6	6.5 ± 0.7	5.4 ± 0.8	5.3 ± 0.6	6.1 ± 0.3
Total caruncle (g)	92.6 ± 43.5	91.6 ± 38.7	104.9 ± 41.1	102.3 ± 38.7	175.8 ± 44.9	102.8 ± 46.0	97.4 ± 35.2	125.9 ± 8.9
Average caruncle (g)	0.8 ± 0.3	0.7 ± 0.3	0.8 ± 0.3	0.9 ± 0.3	1.4 ± 0.3	0.8 ± 0.3	0.8 ± 0.3	1.0 ± 0.1
Total cotyledon (g)	491.9 ± 53.6	455.2 ± 47.7	498.3 ± 50.6	450.6 ± 47.7	447.7 ± 55.3	445.4 ± 56.7	395.0 ± 43.3 ^a	534.7 ± 23.3 ^b
Average cotyledon (g)	5.4 ± 0.5	4.4 ± 0.4	4.3 ± 0.5	4.5 ± 0.4	4.8 ± 0.5	4.4 ± 0.5	4.3 ± 0.4	5.0 ± 0.2
Number:								
Total placentomes	92.1 ± 7.4	106.4 ± 6.6	115.7 ± 7.0	99.8 ± 6.6	94.3 ± 7.6	102.8 ± 7.8	93.7 ± 6.0 ^a	110.0 ± 3.2 ^b
Total caruncles	114.2 ± 8.4	128.6 ± 7.5	133.3 ± 8.0	120.4 ± 7.5	115.9 ± 8.7	123.0 ± 8.9	122.3 ± 6.8	122.9 ± 3.7
Caruncle occupancy ²	0.82 ± 0.03	0.83 ± 0.03	0.87 ± 0.03	0.83 ± 0.03	0.83 ± 0.03	0.85 ± 0.03	0.78 ± 0.02 ^a	0.89 ± 0.01 ^b

¹ for definition see Table 2.

² Number of placentomes/number of caruncles.

Table 10. The effect of pre-partum nutrition on fetal weight, crown rump length, girth, and fetal volume, and organ weights at day 136-140 ¹ of pregnancy (means±s.e.m). Means within rows having superscripts with letters in common or no superscript are not significantly different ($P > 0.05$).

	Treatment					
	MMM ²	MMH	MHM	MHH	HHM	HHH
Number	14	17	19	17	13	12
Fetal weight (g)	4535.9±175.4	4640.5±162.7	4836.6±166.3	4651.5±159.3	4408.5±186.1	4389.2±189.1
Crown rump length (CRL) (cm)	53.1± 0.8	53.3± 0.7	54.6± 0.7	53.7± 0.7	52.7± 0.8	53.2± 0.9
Girth (cm)	34.8± 0.6	35.4± 0.5	35.9± 0.5	35.2± 0.5	34.5± 0.6	34.4± 0.7
Fetal volume (g)	4468.1±241.1	4157.6±223.6	4500.1±228.6	3955.9±218.9	3899.2±255.8	3889.5±259.9
Fetal organ weights:						
• Heart (g)	34.8± 2.1	37.1± 2.0	37.9± 2.0	34.7± 1.9	35.8± 2.2	34.0± 2.3
• Liver (g)	107.0± 5.2	102.7± 4.9	109.9± 5.0	103.3± 4.8	105.5± 5.6	95.0± 5.6
• Lungs (g)	123.4± 5.4	124.1± 5.0	134.1± 5.1	128.2± 4.9	117.8± 5.7	118.7± 5.8
• Kidneys (g)	21.0± 0.9	21.6± 0.9	22.5± 0.9	22.1± 0.8	20.7± 1.0	20.7± 1.0
• Adrenal glands (g)	0.5± 0.0	0.5± 0.0	0.5± 0.0	0.5± 0.0	0.5± 0.0	0.5± 0.0
• Thyroid glands (g)	1.7± 0.2 ^a	1.6± 0.2 ^a	2.1± 0.2 ^{ab}	1.6± 0.2 ^a	1.6± 0.2 ^a	2.6± 0.3 ^b

¹ second slaughtered group of animals.

² for definition see Table 2.

Table 10 shows the effect of pre-partum nutrition from P19 to P136 on fetal weights, crown rump length, girth and fetal volume, and weights of selected fetal organs at day 136 of gestation. None of these growth parameters (except for the thyroid gland weights) was significantly affected by pre-partum nutrition. The data in Table 10 does indicates a slight trend whereby ewes fed MM or MH throughout the first 102 days of pregnancy tended to have heavier fetuses at day 136 of pregnancy. The weights of thyroid glands were affected by pre-partum nutritional changes during pregnancy. Fetuses born to ewes receiving the H feeding levels over the whole trial (the HHH group) had significantly heavier thyroid glands compared with the other groups.

No significant interactions were found between the effects of pre-partum nutrition and pregnancy rank in any of the parameters measured.

2.4. DISCUSSION AND CONCLUSIONS

This study was designed to examine the effect of maternal nutrition (from day 19 to day 136 of gestation) on lamb birth weight in Romney ewes. On days 102-104 of pregnancy a group of ewes were slaughtered to test the hypothesis that maternal nutrition from day 19 to 102 of gestation can influence early placental growth and development. A second group of animals were slaughtered on days 136-140 to test the following hypotheses. Firstly, that fetal growth during the last third of gestation can be affected by altering different nutritional levels in either early (P19) or mid (P47) pregnancy. Secondly, that increasing or decreasing maternal nutrition from day 102 to 136 of gestation can increase or reduce lamb birth weight.

The comparison between target ewe live weights and actual ewe live weights reached over the trial, is presented in Figure 3 and Figure 4. Target ewe live weights in Figure 2 were projected to live weight at day 138 of gestation. Data on actual live weights were only available up to day 131 and for the comparison between target and actual ewe live weight the target live weights

are adjusted to day 131 of gestation. Each of the six different nutritional treatments is presented in Figure 4.

The actual pattern of ewe liveweight changes generating followed the target pattern. However, the actual live weights did not reach the targets set. Target live weights for the MM, MH and HH groups at day 102 of gestation were 58.7, 66.7 and 66.7 kg, respectively while the actual live weights reached were 56.9, 59.0 and 61.5 kg for each of those groups (Figure 3). Target live weights for MMM, MMH, MHM.MHH, HHM and HHH groups were 63.1, 67.1, 71.1, 75.1, 71.1 and 75.1 kg, respectively while the actual live weights reached were 61.7, 64.5, 63.9, 69.5, 64.7 and 70.3 kg for each of those groups at day 131 of gestation (Figure 4).

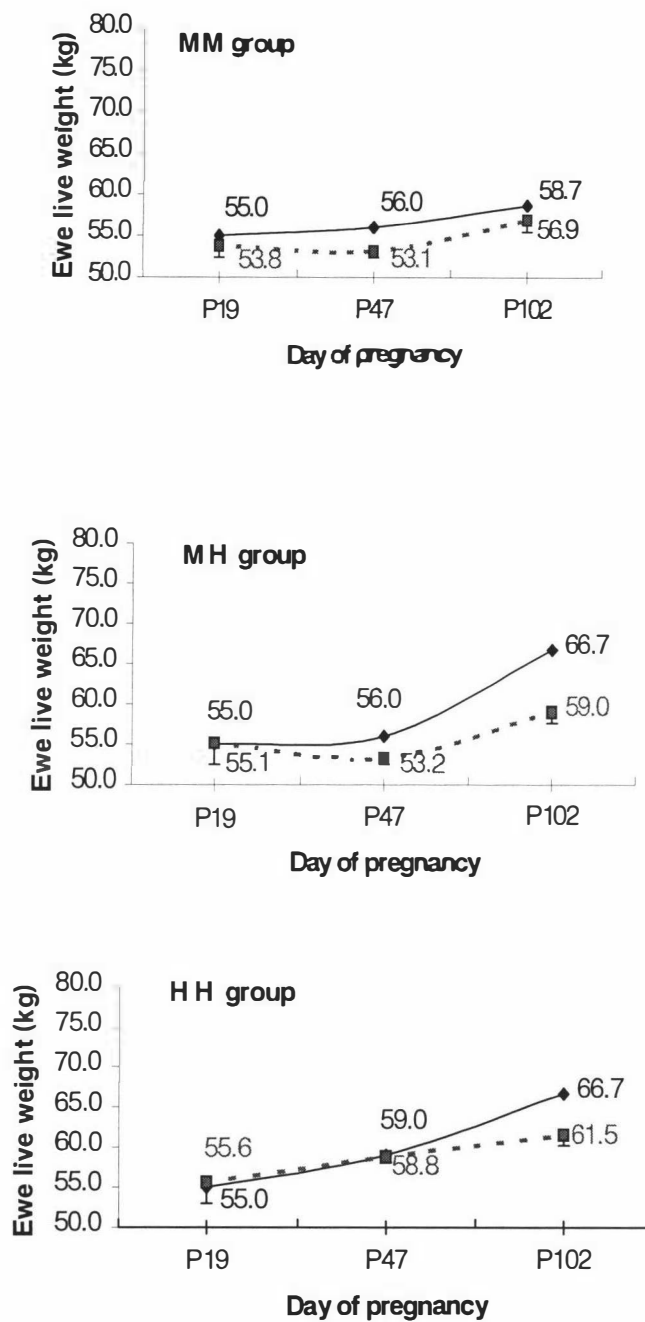


Figure 3. The comparison between target and actual live weights from day 19 to day 102 of gestation for three nutritional treatments. Solid lines (—◆—) indicate target live weights while dotted lines(----■----) indicate actual live weights reached.

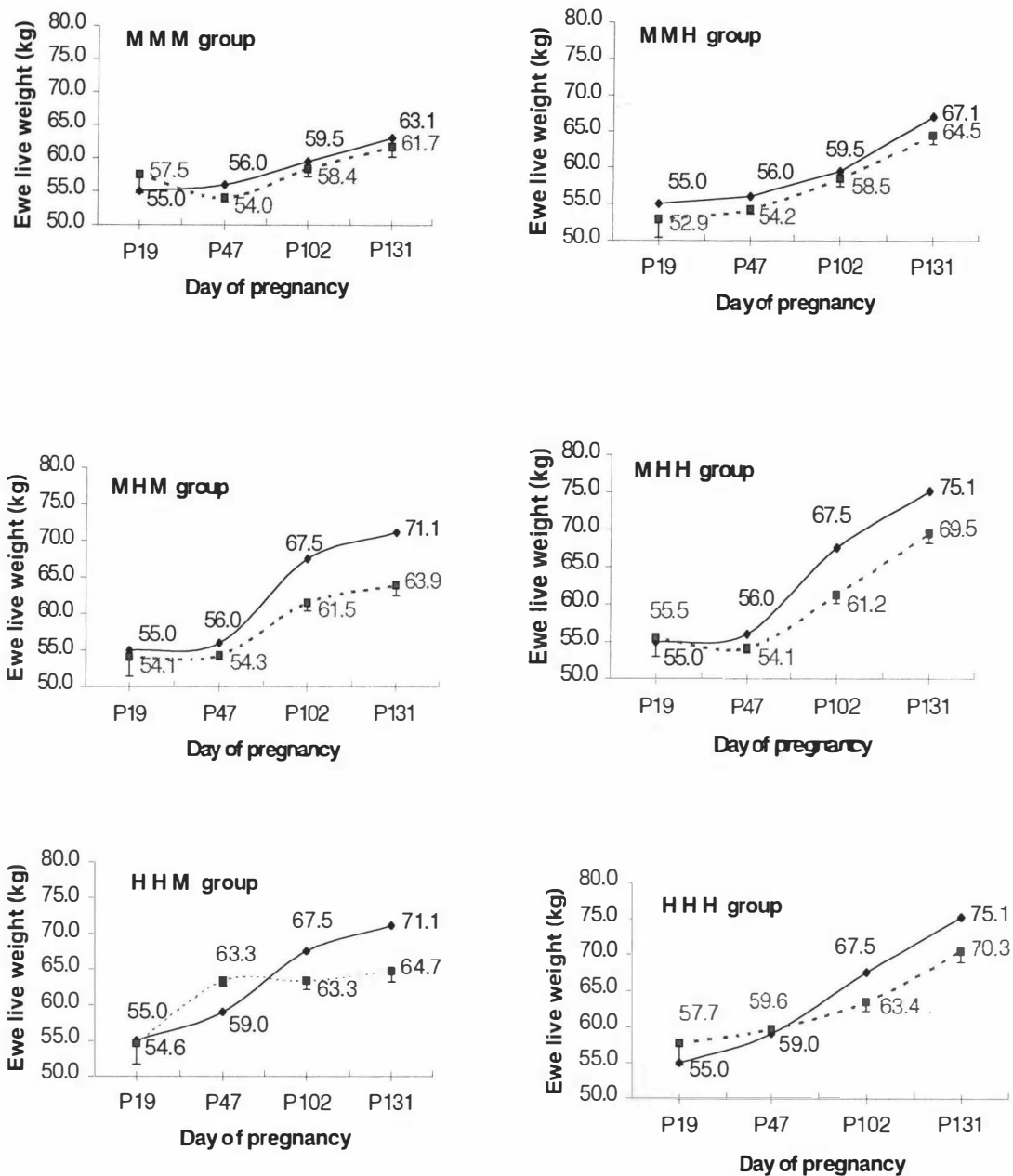


Figure 4. The comparison between target and actual live weights from day 19 to day 131 of gestation for six nutritional treatments. Solid lines (—◆—) indicate target live weights while dotted lines(----■----) indicate actual live weights reached.

These significant effects of pre-partum nutrition on ewe live weight were also reflected in significant ($P < 0.05$) differences in ewe carcass weight at P102 and P136.

The smaller ewe live weight gains than expected in the present study were primarily due to low ewe intakes. Intake of dry matter (DMI) during two sampling periods (P80-84 and P87-91) in this study ranged from 0.98 to 1.24 kg DM ewe⁻¹ day⁻¹ while intake of digestible organic matter (DOMI) ranged from 0.46 to 0.75 kg DOM ewe⁻¹ day⁻¹ (Table 1). For two other sampling periods (P115-119 and P128-132), DMI and DOMI ranged from 0.97 to 1.66 kg DM ewe⁻¹ day⁻¹ and from 0.47 to 0.89 kg DOM ewe⁻¹ day⁻¹, respectively (Table 2). DOMI of Border Leicester X Romney ewes at two periods during pregnancy (P122-125 and P135-139) was 1.1 to 1.2, 1.3 to 1.5, 1.3 to 1.5 and 1.4 to 1.6 kg for pastures of 2.0, 4.0, 6.0 and 8.0 cm sward surface height (SSH) treatment, respectively (Morris *et al.*, 1993). The figures for the 2.0 cm and 8.0 cm SSH treatments approximate the M and H feeding level in this experiment, and suggest that the intake figures in the present study are low for pregnant ewes. Based on feed tables (Geenty and Rattray, 1987), feed requirements at P82 – P89 for pregnant ewes with live weights ranging from 52.9 to 58.6 kg (conceptus free live weight) carrying singles and twins range from 1.09 to 1.16 and from 1.17 to 1.26 kg DM day⁻¹ for singles and twins, respectively. Feed requirements P117 – P131 for pregnant ewes with live weights ranging from 50.5 to 58.5 kg (conceptus free live weight) carrying singles and twins range from 1.41 to 1.45 and from 1.41 to 1.74 kg DM day⁻¹ for singles and twins, respectively.

A possible reason for the low ewe intakes was the poor pasture quality available throughout the experiment. Although dry matter intake (DMI) significantly increased with an increase in feed allowance (*e.g.*, H vs M), average digestible organic matter intakes (DOMI) over the two periods (P80-84 and P87-91) were only 0.46, 0.62 and 0.68 kg DOM ewe⁻¹ day⁻¹ for the MM, MH and HH feeding levels, respectively. This means that only a small amount of OM was available and could be used by the animals for their tissue growth. Therefore, there would be a relatively small amount of

nutrients (*e.g.*, protein, fatty acids and carbohydrate) provided by the dams from their diet for fetal and placental development. Results from laboratory analyses support this conclusion. Herbage OF samples from M paddocks (obtained during P80-84 and P87-91) contained 23.9% ash and those (obtained during P115-119 and P128-132) contained 31.2% ash, compared with ash contents of 14.9% and 16.5% from the H treatment paddocks. This large difference in ash content between M and H treatment was probably related to a high frequency of rainfall during the trial thereby influencing pasture conditions, particularly for the M group where there was a noticeable increase in dirty and muddy pastures. The consequence of this is that ewes were forced to consume herbage that was mixed with soil, hence increasing the ash content of the diet.

Although animal ME requirements were calculated according to pre-planned herbage covers, it is common to experience difficulties in meeting requirements while following pre-planned pasture covers during a grazing experiment. In the present study, the availability of feed supply was determined by measuring herbage cover using an EPM. The measurement of herbage mass using the EPM should be calibrated by other pasture measurements such as using the Hill Farming Research Organization (HFRO) sward stick (Bartham, 1986) and the cutting of quadrates. However, all these methods can give inaccurate readings, for example when the ground is uneven, soil is pugged and when rainfall results in muddy pasture conditions. A high proportion of dead material in the pasture can also cause errors in the calculation. This condition leads to overestimation in the calculation of feed supply as dead material is not usually eaten by grazing animals and, even when eaten, is of little nutritional value.

The ability of dams to buffer a lack of nutrients during pregnancy by mobilizing their body reserves is well recognised. This ability leads to difficulty in determining feeding sufficiency for fetal growth. Final ewe live weights (*i.e.*, at P102) for MM, MH, and HH feeding treatment were 56.0 ± 1.9 , 56.6 ± 1.8 and 60.0 ± 1.2 kg (Table 5). If the gravid uterus weights in Table 5

were subtracted from the actual ewe liveweights giving conceptus free weights of 51.6; 52.3 and 55.8 kg, respectively). These ewes, therefore, lost 2.2 and 2.8 kg for the MM and MH groups, respectively, and gained 0.2 kg for the HH group from P19 to P102 of gestation.

The present study found that there was no impact of nutritional treatment on placental and fetal development. This is in agreement with results of a recent study by Smeaton *et al.* (1999) using half Finn and quarter Finn x Romney ewes offered five different pasture allowances (1.14, 1.58, 2.28, 5.09 and 6.84 kg DM ewe⁻¹ day⁻¹) from day 20 until day 70 of pregnancy. Over the treatment period, corresponding mean-herbage intakes were 0.69, 0.87, 1.14, 1.87 and 2.04 kg DM ewe⁻¹ day⁻¹ resulting in significant ($P < 0.001$) ewe liveweight changes of -1.8, 0.4, 1.5, 3.0 and 3.0 kg respectively (the initial ewe liveweight was 51.7 kg). These differences in ewe liveweight changes, however, resulted in no significant differences in lamb birth weight.

Likewise, in a study using Booroola x South Australian Merino ewes, Kleemann *et al.* (1993) found that the effects of nutritional treatments (low and high) during mid- and late-pregnancy resulted in the treatment groups varying by 7.5 kg and 5.0 kg live weight at day 100 and 135 of pregnancy ($P < 0.05$). Nutrition during mid- or late pregnancy did not influence birth weight (2.96 v. 3.02 kg and 2.98 v. 3.00 kg representing low and high treatments for mid- and late pregnancy respectively). In addition, Davies (1980) stated that a moderate level of under-nutrition in early pregnancy has been shown to have no effect on lamb birth weight. However, Cooper *et al.* (1998) found that a significant difference of 12.3 kg (56.8 ± 1.4 v. 69.1 ± 1.4 kg) in ewe live weight between control (1.0 M) and high (1.5 M) feeding treatments resulted in a significant ($P < 0.05$) difference of 99.0 g (1280.8 ± 38.0 v. 1379.8 ± 35.2 g), and 71.9 g (631.0 ± 30.7 v. 702.9 ± 29.7 g) for placental and fetal weight respectively at day 101 of gestation. In addition, Parr *et al.* (1986) found that fetuses at day 90 of pregnancy, were significantly heavier in ewes fed 1.5 M compared to those fed at a 0.5 M level. These differences in fetal weight are understandable, as the ewe live

weight differences generated were considerably larger than in the present study. The difference between M and H feeding treatment over 83 days (from P19 to P102) of the present experiment was 4.0 kg (Table 5) compared to a 12.3 kg difference between control and high treatment over 81 days (from P20 to P101) in the experiment of Cooper *et al.* (1998).

Increasing or decreasing maternal nutrition during the last period of pregnancy (P102-P136) did not increase or decrease fetal weight regardless of whether the ewes had been fed at a high or medium level earlier in pregnancy. However, placental weights at P136-140 were similar to weights at P102-104 (0.6 kg) indicating that the placenta did not grow beyond P102. This is consistent with the work of many authors (*e.g.*, Alexander, 1964a; Robinson *et al.*, 1977; Vatnick and Bell, 1992; Geenty, 1997) who observed that the placenta reaches its maximum size at about day 90 of pregnancy. On the other hand, fetal weights (absolute growth) increase exponentially, reaching a maximum during late gestation (Jainudeen and Hafez, 1993). This study showed that during the period P102-136, fetal weights increased by over 250% from 1.3 kg at P102-104 to 4.6 kg at P136-140.

Crown rump length (CRL), girth, fetal volume and weights of selected fetal organs such as heart, liver, lungs, kidneys, adrenal glands and thyroid glands at P102-104 and P136-140 were not affected by pre-partum nutrition. The only significant ($P < 0.05$) difference measured occurred in thyroid glands at P136-140. This result contradicts with the fact that there was no significant effect on fetal weight at P136-140. The real cause of these differences is unknown. Sensitivity differences in growth responses between fetus and thyroid glands may be the cause of the contradiction effect. The fetus is possibly less sensitive when responding to differences in maternal nutrition compared with those thyroid glands.

The reduction in total weights of mammary glands, gravid uterus, myoendometrium, fetal membranes, placentomes, caruncles, and cotyledons, in total numbers of placentomes and caruncles, and in caruncle

occupancy in single-bearing ewes compared with twin-bearing ewes is in agreement with other authors (Alexander, 1964b; McCoard *et al.*, 1997). Differences between single-bearing ewes and twin-bearing ewes in placental weights were more likely affected by differences in cotyledon weights and caruncle occupancy. As the placentome is the organ controlling the supply of the nutrition from the dam to the fetus (Jenkinson *et al.*, 1994; McCoard *et al.*, 1996), any increase in placental weight could increase the passage of nutrients to the fetus to meet the high demand of twins (assuming, of course, that the transfer capacity of the placenta was unchanged). The present study found that cotyledon weight (not caruncle weight) of twin-bearing ewes was significantly ($P < 0.05$) greater than in single-bearing ewes. The fact that caruncle occupancy was significantly ($P < 0.05$) greater in twins than in singles is presumably associated with the increased nutrient demand of two fetuses rather than a single fetus. In order to obtain more nutrients from dams, fetuses adapt by increasing cotyledon numbers together with an increase total cotyledon weight.

In this study there was no interaction between pre-partum nutritional treatments and pregnancy rank in any of the parameters observed, suggesting that these effects were independent of another.

In summary, the manipulation of maternal feed intake during pregnancy resulted in no significant effects on placental and fetal weight. It can be concluded (relative to the study of Cooper *et al.*, 1998) that the nutritional levels were not extreme enough to change placental and fetal weight. Some questions remain unanswered such as:

- (1) What effect would applying a similar pattern (but with more extreme nutritional treatments) have on placental and fetal weight?
- (2) What effect would applying the similar pattern (but with more extreme nutritional treatments) have on the concentrations of metabolic hormones?
- (3) What effect would applying the similar pattern (but with more extreme nutritional treatments) have on neonatal survival until weaning time (a critical time period when the lamb is at significant risk)?

- (4) What is the subsequent productive and reproductive performance of the offspring generated from ewes subjected to these nutritional treatments (but with more extreme nutritional levels)?

All of the above offer new areas of potential study. A suggested experimental design could be as follows. A first experiment would be run using a similar design to the present study but with more extreme nutritional treatments. These might give significant effects on placental and fetal weights at day 100 of gestation, and therefore might increase lamb birth weights (particularly for lambs destined to be born to low birth weight). A second experiment would be run in parallel, and with a similar design, to the first experiment but no ewes would be slaughtered. The survival parameters of neonatal or offspring would be tested as well as productive and reproductive performance of the offspring.

It is often suggested by farmers that maternal nutrition during pregnancy is important for placental and fetal development and therefore promotes an optimal lamb birth weight and ultimately lamb survival. In practice, however, this is difficult to achieve. Optimum birth weights for lamb survival have been estimated as about 3.9-5.0 kg for single lambs and 3.2-4.5 kg for twin lambs (Hight and Jury, 1970; Geenty, 1997) or 3.5-5.5 kg for both singles and twins with an optimal birth weight of 4.7 ± 0.2 kg for maximum lamb survival (Dalton *et al.*, 1980; Geenty, 1997). Lambs weighing less than about 3.0 kg or more than 6.5 kg at birth have a very low survival rate (Hight and Jury, 1970; Dalton *et al.*, 1980; Geenty, 1997).

Referring to those optimal figures for lamb birth weights, this study indicated that average fetal weights at day 136-140 were in the optimum birthweight range for maximum lamb survival. For example, ewes receiving maintenance feeding level throughout pregnancy (*ie.*, MMM, from day 19 to day 136) produced fetuses with a mean weight of 4535.9 ± 175.5 g. Average conceptus free liveweights of these ewes at P136-140 was 51.0 kg so that these ewes lose a 6.5 kg of their initial liveweights (*ie.*, 57.5 kg) over the

entire trial period (P19 to P140). This means that about 11.3% of their body reserves were used to support pregnancy. This suggests that a maternal feeding level of 1 times maintenance was, in this trial, sufficient to support good developments of placentas and fetuses.

There was no increase or decrease in fetal weights in this trial when ewes offered a high or maintenance feeding level from P102 to P136. These results reinforce the importance of adequate feeding in early and mid-pregnancy to ensure lamb birth weight and lamb survival are optimum (Geenty, 1997). Attempts to increase lamb birth weight through high levels of ewe nutrition in late pregnancy are limited by placental development which has been determined by day 90 of pregnancy (Davis *et al.*, 1981; Geenty, 1997). On the other hand, severe reduction in ewe feeding in the last 40-50 days of pregnancy can decrease fetal growth by 30-70% (Mellor, 1983; Geenty, 1997). Farmers should, therefore, be concerned about maternal nutrition during mid-pregnancy.

To ensure adequate feeding during early and mid pregnancy, it is essential that ewes are mated in good body condition. During early pregnancy, the ewe's body condition should be maintained. Underfeeding during this period may result in the death of some embryos in multiple pregnancies. The development of the placenta occurs between days 30 and 90 of pregnancy and during this period ewes mated in good body condition (condition score 3.5 or better) can afford to lose up to 10% of body weight. Over feeding may also have an adverse effect on placental development as well as contributing to excessive body condition at parturition and subsequent dystocia or vaginal prolapse. Underfeeding results in ewes entering the final six weeks of pregnancy, and ultimately lambing, in poor body condition. The maximum period of fetal growth is during the final six weeks of gestation. Nutrition during this period has a large effect on lamb birth weights, their subsequent viability and growth rates. However, this research indicates that a moderate level of feeding (maintenance plus allowance for fetal growth) is sufficient to ensure optimum lamb birth weights.

It is recommended that weighing and body condition scoring during pregnancy are used to ensure feeding levels meet pre-planned targets.

In conclusion, it seems that a moderate level of nutrition throughout the entire pregnancy is advisable rather than severe under- or over-feeding during specific periods of pregnancy. While the results of Cooper *et al.* (1998) suggest that fetal growth may be enhanced by high levels of maternal nutrition during the first 100 days of gestation, the present study indicates that this response is unlikely to occur at the more modest feeding levels achievable on most New Zealand farms.

APPENDIX

CALENDAR OF EVENTS FOR AUGUST-LAMBING EWES

THE EFFECT OF MANIPULATION OF FEED INTAKE DURING PREGNANCY ON LAMB BIRTH WEIGHT

Number of Ewes	Day	Date	Week Day	Task
200		13/03/98	Friday	Tag sheep - insert CIDR's
200	P-2	25/03/98	Wednesday	Weigh ewes, introduce rams & remove CIDR's
200	P0	27/03/98	Friday	Assumed conception date
200	P6	02/04/98	Thursday	Change crayon tup red
200	P12	08/04/98	Wednesday	Tup red
160	P19	15/04/98	Wednesday	Weigh and allocate ewes to treatments
160	P26	22/04/98	Wednesday	Weigh ewes
160	P33	29/04/98	Wednesday	Weigh ewes
160	P40	06/05/98	Wednesday	Weigh ewes
90	P47	13/05/98	Wednesday	Pregnancy diagnosis, weigh, select and allocate ewes to treatments
90	P54	20/05/98	Wednesday	Weigh ewes
90	P61	27/05/98	Wednesday	Weigh ewes
90	P68	03/06/98	Wednesday	Weigh ewes
90	P73	08/06/98	Monday	Insert CRC (I)
90	P75	10/06/98	Wednesday	Weigh ewes
90	P80	15/06/98	Monday	Collect faeces
90	P81	16/06/98	Tuesday	Collect faeces
90	P82	17/06/98	Wednesday	Collect faeces and weigh ewes
90	P83	18/06/98	Thursday	Collect faeces
90	P87	22/06/98	Monday	Collect faeces
90	P88	23/06/98	Tuesday	Collect faeces
90	P89	24/06/98	Wednesday	Collect faeces and weigh ewes
90	P90	25/06/98	Thursday	Collect faeces
90	P96	01/07/98	Wednesday	Weigh ewes
60	P101	06/07/98	Monday	Start slaughter (10 ewes each treatment)
60	P103	08/07/98	Wednesday	Weigh ewes
60	P108	13/07/98	Monday	Insert CRC (II)
60	P110	15/07/98	Wednesday	Weigh ewes
60	P115	20/07/98	Monday	Collect faeces
60	P116	21/07/98	Tuesday	Collect faeces
60	P117	22/07/98	Wednesday	Collect faeces and weigh ewes
60	P118	23/07/98	Thursday	Collect faeces
60	P124	29/07/98	Wednesday	Weigh ewes
60	P129	03/08/98	Monday	Collect faeces
60	P130	04/08/98	Tuesday	Collect faeces
60	P131	05/08/98	Wednesday	Collect faeces and weigh ewes
60	P132	06/08/98	Thursday	Collect faeces
60	P136	10/08/98	Monday	Start slaughter ewes
60	P137	11/08/98	Tuesday	Slaughter ewes
60	P138	12/08/98	Wednesday	Slaughter ewes
60	P139	13/08/98	Thursday	Slaughter ewes
60	P140	14/08/98	Friday	Slaughter ewes

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