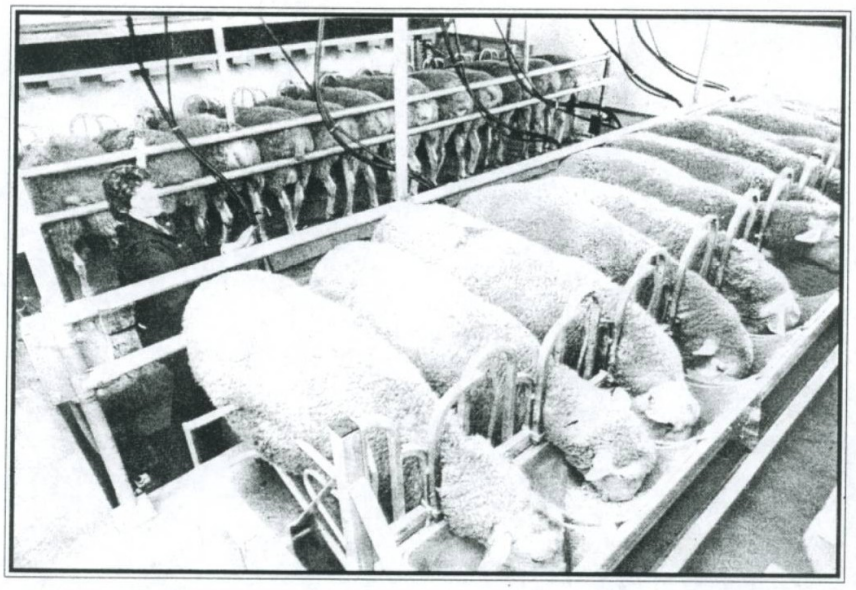
PAPER IN SYMPOSIUM PROCEEDINGS

BEE TOLMAN

Proceedings of the 7th Great Lakes Dairy Sheep Symposium



November 1-3, 2001

Eau Claire, Wisconsin

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THE EFFECT OF GROWTH RATE ON MAMMARY GLAND DEVELOPMENT IN EWE LAMBS: A REVIEW

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Summary

Over the past twenty years, research has established that there is a negative relationship between prepubertal growth rate and lifetime milk production in sheep and cattle. In cattle, reduced mammary development as a result of high growth rate can result in 10 to 17% less daily milk production during the 1st, 2nd, and 3rd lactation, compared to animals raised at slower growth rates. We as dairy sheep producers place great emphasis on growing our replacement ewe lambs quickly, to ensure that they will be of adequate size to reach puberty and conceive as lambs, thereby giving birth and lactating for the first time as yearlings. By doing this, we may be producing ewes with less capacity for milk production and, in effect, shooting ourselves in the foot: spending money on feed supplements for ewe lambs who then become less-productive milkers precisely because they grew so well as lambs. It is therefore paramount that attention be given to determining the proper growth rate in dairy ewe lambs.

The negative effect of high gain occurs during a critical period: from about 1 month to 5 months of age in the ewe lamb. In this period the lamb's mammary gland is producing great masses of "ductules", the small ducts that in pregnancy will grow into the milk-secreting alveoli and the milk-transporting ductal network. Any process that limits the development of these ductules will thus also limit the mature gland's capacity to produce and transport milk.

The effect of nutrition on prepubertal mammary growth is largely due to the inverse relationship between feeding level and growth hormone (GH) concentrations. When animals are fed at lower levels of energy, GH release is relatively higher, compared to animals on high-energy, ad-lib diets. GH acts via other hormones to stimulate cell division, provoking growth and proliferation of the ductules, and it makes energy available for the rapidly-dividing cells in the ductules. If nutrition is too restricted, however, there will be inadequate growth of the mammary fat pad, which is essential for mammogenesis. The fat pad provides the framework for mammary gland development and is also a source of local hormones critical to the growing gland. If energy intake is too low, and the fat pad too small, mammary growth can be inhibited because the proliferating system of ducts will literally run out of fat pad area in which to expand.

Recent trials in dairy heifers suggest that optimal gain -- that which results in the greatest milk yield -- is about 65 to 75% of maximum daily gain. Although there have been no equivalent trials with sheep, research has shown that growth rates in the young ewe lamb must not be too low, limiting fat pad area and seriously delaying puberty (thus lowering reproductive efficiency), nor too high, resulting in lower milk production. The objectives of this paper are to review the current state of available knowledge concerning the effect of nutrition on mammary growth in the young ruminant female, and the subsequent effects on lactation performance.

Prepubertal Mammogenesis

The portion of the mammary gland that contains the secretory alveoli and the ducts that transport the milk secretions is called the "parenchyma"; parenchymal tissues are composed of epithelial cells. In the ewe lamb, development of the mammary epithelium is restricted to undifferentiated cell production: the proliferation of a network of ducts through the mammary fat pad. During pregnancy these epithelial cells will further proliferate and differentiate into either ducts or the specialized milk-producing cells of the alveoli. The parenchyma is surrounded and supported by the "stroma", also called the fat pad. The fat pad is composed primarily of connective tissue and adipose tissue, as well as vascular and lymphatic systems, and nerves and myoepithelial cells.

In the ruminant, postnatal development of the mammary parenchyma proceeds through specific proliferative stages. At birth the basic glandular structures have been formed, with a single primary duct arising from the teat. In the calf/baby lamb period (until 2 to 3 months in the calf, and probably until 1 to 2 months in the lamb), mammary epithelial growth is *isometric* -- growing at a rate similar to that of the whole body -- and is limited to the development of secondary and tertiary ducts in the zone adjoining the gland cistern, and to growth of non-epithelial connective and adipose tissues (Sejrsen and Purup, 1997).

At some point in the second month of life, the lamb's mammary gland enters an *allometric* phase of growth -- epithelial cell numbers are increasing at a rate faster than that of the whole body. During this time, extensive outpocketing of epithelial tissue arises from the secondary and tertiary ducts around the gland cistern. Hovey et al. (1999) has described these outpockets as clusters of ductules arising from the termini of more sizable ducts (Figure 1). The ductules advance as dense masses, replacing surrounding adipose tissue as they progress. DNA is being actively synthesized at the periphery of these masses of ductules, indicating rapid cell proliferation. During this period the fat pad is also growing, adding adipose tissue and the structurally-supporting connective tissues (Sejrsen et al., 2000).

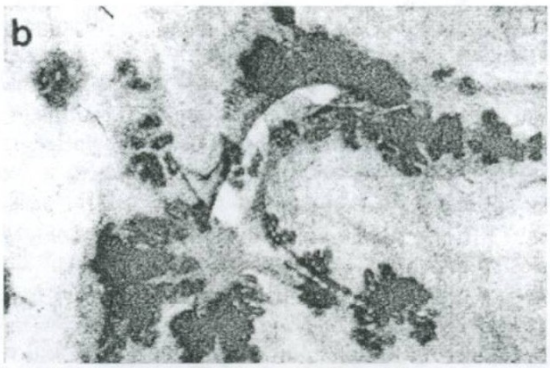

Figure 1. Whole mount of a terminal ductal unit from the mammary gland of a prepubertal ewe lamb. Dense clusters of epithelial ductules can be seen arising from hollow ducts, particularly at their terminus. From Hovey et al., 1999.

After puberty, the mammary gland reverts to isometric growth, with growth again paralleling that of the whole body.

Pregnancy causes the mammary gland to undergo another period of extensive development and allometric growth (Sejrsen et al., 2000). In the ewe, mammary growth in early pregnancy consists of rapid ductal growth. In late pregnancy, differentiated epithelial cells form alveoli, which are anchored to collagen. DNA analysis of cell numbers of epithelial and connective tissue in Romney sheep showed that 20% of gland growth occurred between birth and puberty, 78% during pregnancy, and 2% during lactation (Anderson, 1975).

Total milk yield is proportional to total epithelial cell numbers (Hovey et al., 1999). As revealed by the Romney ewe study above, 98% of epithelial cell numbers are established by parturition. Between parturition and peak lactation, secretory cells hypertrophy and become more fully differentiated. Forsyth (1995) studied the relationship between milk yield and alveolar cell activity and persistence in dairy cattle. She found that after peak lactation, activity per individual cell is maintained, and that alveolar cell loss is the primary cause of milk yield decline. In the modern dairy cow, peak yield accounts for 66 to 80% of the variance in total yield, and persistency (days in milk) accounts for only 8 to 12 % of the variance. Peak yield is primarily determined by secretory cell numbers. Therefore any process that negatively impacts epithelial cell numbers will negatively impact total milk yield.

The Fat Pad

The mammary fat pad is a matrix of connective and adipose tissue that also houses the gland's vascular and lymphatic systems. It has three major functions critical to mammogenesis: first, its connective tissue network provides the structural support for the parenchyma; second, it serves as an active lipid store for the growing and/or differentiating epithelia; and third, in a function only now being understood, it locally regulates mammary development by mediating hormone action and synthesizing growth factors.

As shown in the recent work by Hovey et al. (1999), the fat pad's adipose tissue is extensively interlaced with rope-like cords of connective tissue fibroblasts. In the lactating adult, these cords will give structural support to the fluid-filled udder. In the developing animal, the connective tissue meshwork, interspersed between adipocytes, directs the spread of parenchymal growth: ductules are seen lying embedded within veins of connective tissue (Figure 2). As the growing ducts advance through the adipose/collagen matrix, fibroblasts proliferate in response to the oncoming ductules, continuously ensheathing the ductules in multiple layers of fibrous tissue.

Although the proliferating epithelial cells do not directly abut the fat pad's adipocytes, the adipose tissue acts as a lipid depot for the dividing cells. Lipid-depleted adipocytes are seen in the area of ductal infiltration. Furthermore, Hovey et al. (1999) found that adipocyte-derived fatty acids markedly increased the response of mammary epithelial cells to growth factors in vitro.

The actions of many hormones are mediated by the mammary fat pad, and the fat pad is also a major site for the synthesis of local growth factors. Growth hormone (GH), a dominant mammogenic hormone, has binding sites in adipose tissue in the prepubertal ewe lamb. Insulin-like growth factor-1 is stroma-derived in the mammary gland, and there appear to be epidermal growth factor receptors within the fat pad as well as on epithelial cells (Hovey et al., 1999).

The mammary fat pad is therefore not simply an inert supporting material. It plays an integral and critical role in directing, stimulating, and regulating mammogenesis.

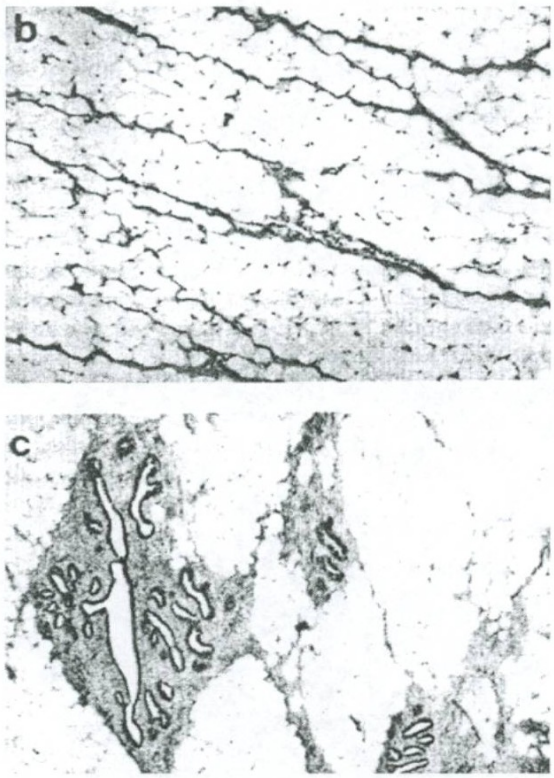

Figure 2. Sections of mammary fat pad from a prepubertal ewe lamb. (a) A meshwork of fibrous connective tissue cords can be seen throughout the adipose tissue. (b) As the epithelial tissue expands into the fat pad, ductules advance ensheathed within cords of connective tissue. From Hovey et al., 1999.

Level of Nutrition and Milk Yield

A large number of trials have shown that high levels of nutrition in the prepubertal ruminant can limit mammary gland development, resulting in reduced milk yield in subsequent lactations.

Umberger et al. (1985) reared Suffolk- and Dorset-cross ewe lambs from early weaning (20kg) to puberty on either pasture plus ad-lib grain ("F" ewes; 0.20 kg/d gain) or on pasture plus limited grain ("T" ewes; 0.12 kg/d gain). After breeding at 9 months, treatments were reversed for half of each group. Milk was collected at 20, 40, and 60 days of the ewes' first lactation, and

daily milk yields were estimated for the 60-day period. Alveoli were also counted from ewes sacrificed in each group (alveoli per 4.23mm^2). Results showed that slower-growing ewe lambs had more alveoli (402 vs 334 alveoli per unit area) and produced more milk (1.56 vs 1.29 kg/d). Reversing the feeding level after the start of breeding did not affect milk yield or alveoli numbers.

In dairy heifers, the negative relationship between prepubertal daily gain and milk yield has been well documented. In a recent study, Hohenboken et al. (1995) reported that as prepubertal daily gain was reduced from a high level (H: 0.79kg/d) to a moderate level (M: 0.67 kg/d) to a low level (L: 0.52 kg/d), first-lactation milk yield increased: compared with H heifers, those on M and L rearing intensities had 9% and 14% more milk, respectively. Also, consistent with their lower milk production, H heifers were less efficient in nutrition utilization during lactation. Compared with L and M groups, H heifers lost less weight during early lactation, passed into positive energy balance sooner after parturition, and gained more weight to the end of lactation.

Many trials have examined the mammary parenchymal tissue of animals reared on different levels of food intake, and related observed mammary development to the subsequent milk yield of similarly-raised adults.

From a histological perspective, prepubertal daily gain is positively correlated with total mammary gland weight and the percentage of adipose tissue in the gland, but negatively correlated with measures of epithelial cells and connective tissue. Sejrsen et al. (1982) found that heifers on a restricted diet (0.64 kg/d gain) had less total gland weight than heifers on a high-energy, ad-lib diet (1.3 kg/d). Restricted heifers, however, had 61% less extra-parenchymal adipose tissue and 30% more absolute parenchyma, which occupied a greater proportion of the available fat pad area. Within the parenchyma itself, the restricted heifers had more DNA content (reflecting epithelial cell numbers: 1562 mg vs 1061 mg), more hydroxyproline (reflecting connective tissue mass: 2288 mg vs 1466 mg), and more parenchymal lipid (3140 mg vs 2340 mg).

This effect on mammogenesis appears to be permanent, and has been shown to persist over several lactations in both beef cattle and dairy cattle. After four lactations, dairy cows fed a moderately-restricted diet (0.75 kg/d) as heifers still had 68% more secretory tissue and 16% less adipose tissue than cows that had been fed to near-maximum gain (1.1 kg/d) as heifers (Harrison et al., 1988). Similarly, beef cows reared at a low daily gain had significantly higher 30-day milk yields and weaned significantly heavier calves in their 1st, 2nd, and 3rd lactations, compared to contemporaries reared at a high daily gain (Johnsson and Obst, 1984).

The negative effect of high plane of nutrition on mammary gland development appears to cease at or around puberty. In the non-pregnant dairy animal, there is no consistent growth of mammary secretory tissue after the 2nd estrous cycle (Sejrsen et al., 1982), and there is no correlation between rate of gain and secretory tissue mass in postpubertal dairy cattle (Sejrsen et al., 1982; Harrison et al., 1983), in beef cattle (Johnsson and Obst, 1984), or in sheep (Umberger et al., 1985; Johnsson and Hart, 1985; Anderson, 1975).

Allometric Mammary Growth

It is important to identify just when, between birth and puberty, nutritional level and daily gain have the greatest impact on mammogenesis and therefore lactation. Sinha and Tucker (1969) reported that in the heifer's mammary gland, major allometric growth occurs, but within a specific timeframe: relative to increases in liveweight, mammary DNA increased 1.6 times faster from birth to 2 months, 3.5 times faster from 3 to 9 months, and then 1.5 times faster from 10 to 12 months. Accordingly, Sejrsen et al. (2000) found that in calves of up to 2 - 3 months of age, there was very little epithelial growth, and raising or lowering the plane of nutrition in this early period had no significant effect on mammary gland development.

Although some researchers have assumed that the equivalent first isometric growth phase in lambs is from birth to 4-6 weeks of age (Johnsson and Hart, 1985; McCann et al., 1989), no work has been done to specify the end point of this first period in sheep.

Two major trials conducted with sheep and beef heifers identified the main period when the mammary gland is most sensitive to changes in nutritional level. Using Hereford heifers, Johnsson and Obst (1984) looked at the effects of high (H), moderate (M), and low (L) energy intakes in two prepubertal periods: before 8 months of age, and between 8 months and puberty (Table 1). They found that LM heifers (i.e., low energy intakes 2-8 months and moderate energy intakes 8-14 months) produced 50% more milk and weaned 40-kg heavier calves in the first lactation than either HM or MM heifers, despite weighing significantly less at first calving. They concluded that nutritional level had more impact on mammogenesis in the early prepubertal period than in the late prepubertal period. In support of this, crossover treatments after 8 months had limited effect: HL heifers (severely restricted growth after 8 months) only marginally overcame the detrimental effects of early rapid growth, while increasing to ad-lib feeding after 8 months (LH) improved liveweight at breeding but did not depress milk yield.

Table 1. Milk yield, estimated at 30 days of lactation, over 3 lactations from beef heifers fed to grow at high (H), moderate (M), or low (L) rates.

		Rearing treatments					
		HM	*HL*	*MM*	*LH*	*LM*	Stat.signif
Daily gain, kg	2-8 months	0.91	.91	0.67	0.55	0.55	***
	8-14 months	0.57	.14	0.58	0.97	0.55	***
Liveweight, kg	8 months	235	235	185	164	164	***
	14 months	331	259	284	323	258	***
Puberty	Live weight, kg	244	237	238	234	233	NS
	Age, mo	9.0	9.1	11.6	11.0	12.1	***
Relative milk yield	1st lactation	100	120	105	139	154	**
	2nd lactation	100	131	135	130	135	*
	3rd lactation	100	127	141	145	149	**

After Johnsson and Obst, 1984

Using February-born Hampshire Down-cross ewe lambs, Johnsson and Hart (1985) were able to quantify the effects of nutrition on allometric growth rates. With these slower-maturing, highly-seasonal lambs, the trial revealed two distinct periods of allometric parenchymal growth before the lambs reached puberty in October and November. Between 4 and 20 weeks of age, lambs were fed for either low or high gain (L or H; Table 2). Then, between 20 and 36 weeks, lambs were either maintained at low gain, switched to high gain, or switched to low gain (LL, LH, or HL). As with the Hereford heifers, mammogenesis in lambs was greater in the early prepubertal period: overall, total parenchymal DNA increased 19-fold between 4 and 20 weeks, whereas total parenchymal DNA increased only 3-fold between 20 and 36 weeks.

The trial also showed that a high plane of nutrition reduces the *rate* of allometric growth in early puberty. Between 4 and 20 weeks, mammary growth was rapid and significantly affected by liveweight gain: mammary parenchyma increased 3.7 times faster than liveweight in L lambs, but only 2.4 times faster in H lambs. Between 20 and 36 weeks, however, the response to feeding level appeared to be determined by nutrition in the previous period. In ad-lib fed lambs (H), restriction of diet after 20 weeks (HL) promoted allometric growth: 2.6 times that of liveweight. In restricted lambs (L), mammary development after 20 weeks occurred in proportion to liveweight gain: 1.4 times that of liveweight for LH lambs, 1.6 for LL lambs.

Table 2. Mammary fat pad and parenchymal tissue development in ewe lambs fed to grow at low or high rates at 4 to 20 weeks and 20 to 36 weeks

	Rearing treatment					
	L	*H*	*LL*	*LH*	*HL*	Stat.
	4 to 20 weeks		*20 to 36 weeks*			Signif.
Daily gain, kg	0.12	0.22	0.11	0.21	0.11	
Age at slaughter, wks	20	20	36	36	36	
Live wt, kg	23.7	33.2	36.1	48.9	47.7	*
Trimmed fat pad, g	14.7	30.0	46.0	86.7	70.3	*
% Fat pad occupied	65	27	53	46	44	*
Parenchyma (dried, fat-free tissue), mg	844	623	1580	2496	1883	*
Total epithelial DNA, mg	32	26	61	91	73	*
Live weight at puberty, kg			33.4	43.3	45.7	***
Age at puberty, days			233	240	235	NS

After Johnsson and Hart, 1985

The exception to this was the early puberty reached by a few lambs in the H group, which ended allometric mammary development at 18 weeks and resulted in reduced mammary gland size.

There was also a significant difference in the proportion of fat pad occupied by parenchyma. At 20 weeks, parenchyma in L lambs had occupied 65% of available fat pad area, while H lambs had 27% occupied. And although LH lambs had 25% more parenchymal DNA than HL lambs at

similar liveweights (48 kg at 36 weeks), the final proportion of fat pad occupied at 36 weeks was similar (46 and 44%, respectively).

From these results the authors concluded that 1) the allometric phase of prepubertal mammogenesis is primarily a function of age and largely reverts to isometric growth before normal puberty; 2) after 20 weeks, the size of available fat pad may limit further lateral expansion in restricted-gain lambs; and 3) precocious puberty attained within the major phase of allometric growth can lead to marked decrease in mammary gland size.

The effect of nutritional plane on prepubertal mammogenesis is similar across breeds in sheep and cattle. Hohenboken et al. (1995) reported that there was no difference in sensitivity of subsequent milk yield to high prepubertal gain across four Danish dairy or dual-purpose cattle breeds. In sheep, this relationship has been demonstrated in trials with Dorset-, Suffolk-, Hampshire-, and Finn-cross ewe lambs (Umberger et al., 1985; McCann et al., 1989; Johnsson and Hart, 1985; McFadden et al., 1990).

Hormonal Influences on Prepubertal Mammogenesis

Researchers now recognize that the impaired mammary development of rapidly-reared prepubertal ruminants is caused by altered secretions of hormones, growth factors, and binding proteins, all of which regulate mammary development (Sejrsen et al., 1997). Although the exact physiological mechanisms are not understood, it is agreed that high feeding levels inhibit circulating growth hormone (GH) concentrations in the blood, and that there is a positive correlation between serum GH and prepubertal mammary growth (Sejrsen et al., 1983, Johnsson et al., 1985). This has also been confirmed by trials showing that exogenous administration of GH produces increased parenchymal tissue growth in lambs (McFadden et al., 1990; Johnsson et al., 1986) and heifers (Sejrsen et al., 1997).

Johnsson et al. (1985) examined the effects of daily nutrition on GH plasma concentrations at various ages before puberty and the associated mammogenesis. Blood samples were taken from ewe lambs at 10, 14, 18, 26, and 36 weeks of age. As described earlier, lambs were fed to either a high (H) or low (L) daily gain from 4 to 36 weeks. Results showed that GH response to fresh daily feed was significantly higher in L lambs than H lambs at 10, 14, and 18 weeks, and that GH concentrations and mammary development were positively correlated. As the lambs' ages increased after 20 weeks, both the mean level of serum GH and the sensitivity of GH concentrations to feed intake declined (Figure 3).

Growth hormone levels have also been positively correlated with ductal growth during the allometric period in heifers (Sejrsen et al., 1983). Serum GH concentrations were depressed in prepubertal heifers on ad-lib feed, but were not affected by rearing rate after puberty. And, as with lambs, serum GH concentration was negatively correlated with extraparenchmal adipose tissue mass, indicating a lipolytic role of GH.

It is believed that the mammogenic actions of GH may be mediated by the stromal cells of the fat pad. In the prepubertal ewe, adipose tissue binds GH, and probably stimulates the production and secretion of insulin-like growth factor-1 (Hovey et al., 1999). IGF-1 is a direct

and potent mitogen for undifferentiated ruminant epithelial cells (Weber et al., 1999), and also, in gestation, for differentiated cells, stimulating DNA synthesis in ductal epithelial cells, secretory alveolar cells, and myoepithelial cells (Forsyth, 1995).

It is important to note that IGF-1 is highly mitogenic for undifferentiated mammary epithelial cells *at low concentrations*. Weber et al. (1999) reported that in vitro, additional IGF-1 caused epithelial cells to secrete binding proteins (IGFBP-2 and IGFBP -3) that bind IGF-1 with high affinity and regulate its bioactivity. It is suspected, therefore, that high feed levels in the prepubertal ruminant result in high concentrations of IGF-1, which in turn trigger production of binding proteins that render the mammary tissue insensitive to circulating IGF-1 (Sejrsen et al., 2000).

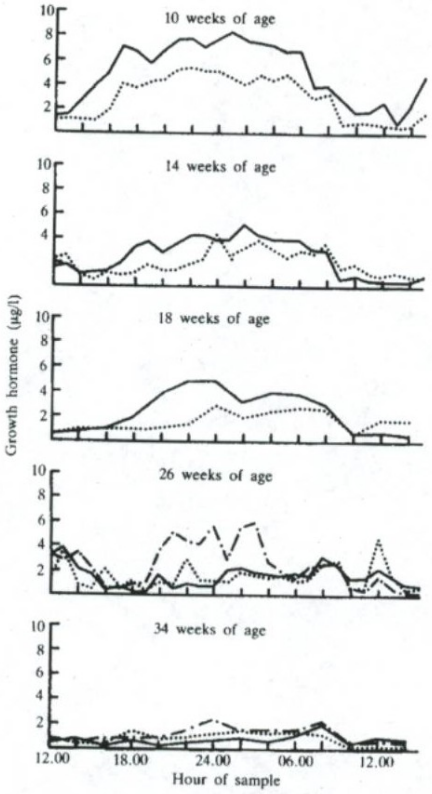

Figure 3. 24-hour plasma GH response to daily feeding of lambs on either HL (.....), LH (__), or LL (_._) feed levels. For all treatments, feeding occurred between 8.00 and 9.00 am. From Johnsson et al., 1985.

A large number of other growth factors are involved in the regulation of mammogenesis, as either stimulators or inhibitors. Normal cells require more than one growth factor for mitotic cell cycle progression (Forsyth, 1995), and may require a sequential series of growth factor actions. Growth hormone is a dominant player in mammogenesis. How this hormone controls the growth factors, which in turn mediate hormonal actions, is still largely unknown.

In summary, restricted energy intake in the prepubertal ewe lamb allows full release of GH to 1) stimulate IGF-1 production and consequent mitogenic activity in mammary epithelia, and, through the lipolytic role of GH, to 2) make energy from adipocytes available to the rapidly-dividing ductal epithelia. Conversely, unrestricted energy intake in the prepubertal ewe inhibits GH release which, via an unknown mechanism, allows high concentrations of IGF-1 to trigger production of IGF binding proteins, thereby rendering the mammary gland less sensitive to mitogenic stimuli.

Onset of Puberty and Reproductive Performance

Puberty is the culmination of a gradual maturation process that begins before birth and is primarily determined by body weight and photoperiod (season). Within breeds of sheep or cattle, the main source of variation in age at the onset of puberty is level of feeding (Sejrsen and Purup, 1997).

Within seasonal limits, attainment of puberty appears to be determined by body weight rather than age. In both sheep and cattle trials, the restricted planes of nutrition were sufficient for the skeletal and body development required for sexual maturity (Stelwagen and Grieve, 1990; Johnsson and Hart, 1985)

While studying the effects of daily gain on mammary development, researchers have also observed effects of daily gain, particularly restricted gain, on reproductive performance. Overall, gain restricted to improve mammary development had no clear adverse impact on conception or lambing rate. Umberger et al. (1985), for instance, reported no difference in conception rate between prebreeding treatments, but found an increased lambing rate (1.41 vs 1.11) in ewe lambs on the higher level of feed. Conversely, McCann et al. (1989) found that ewe lambs on the higher feed level had improved conception (92% vs 88%) but no better lambing rate.

Effect of Diet Formulation

It appears to be dietary energy level, rather than ration formulation or total intake, that influences growth hormone release and subsequent mammary development. In heifers, the negative impact of feeding level is not affected by roughage type (corn silage vs grass silage vs barley straw; Hohenboken et al., 1995); by energy source (fiber vs starch; Houseknecht et al., 1988); by protein level or protein source (Sejrsen and Purup, 1997); or by concentrate/roughage ratio (Sejrsen et al., 2000).

Beef heifer weanlings were fed either high-energy-fiber (HEF), high-energy-starch (HES), or low-energy-fiber (LE) diets (Houseknecht et al., 1988; Table 3). Protein levels were similar across treatments; daily gain, as intended, was lower with the low-energy diet. The LE diet produced significantly higher levels of GH, while both HE diets produced higher levels of IGF-1, showing that serum GH and IGF-1 concentrations are influenced by dietary energy level rather than energy source.

Table 3. Growth hormone (GH) and insulin-like growth factor (IGF)-1 response to differing energy intakes and sources in beef heifers

	Rearing treatments			
	High energy - starch *(corn/soybean meal)*	High energy - fiber *(soy hulls/soybean meal)*	Low energy - fiber *(soy hulls/soybean meal)*	*Statistical significance*
ME, Mcal/kg	18.84	17.16	10.17	
CP, %	13.3	13.1	14.1	
NDF, %	34.7	63.8	61.4	
Daily gain, kg	0.92	0.88	0.42	*
GH, ng/ml	6.4	8.3	13.8	**
IGF-1, ng/ml	92.6	92.2	63.3	**

From Houseknecht et al., 1988

Specific effects of some nutrients cannot be ruled out, however, particularly the addition of polyunsaturated fats (PUF) to the diet. At five months of age, lambs on an ad-lib diet with a PUF supplement (sunflower seeds) had more parenchymal and fat pad development than either the non-supplemented ad-lib or the restricted-diet lambs (McFadden et al., 1990). Furthermore, Hovey et al. (1999) noted that a diet deficient in essential fatty acids impaired ductal growth, and that unsaturated fatty acids have been found to enhance the proliferative effects of mammary growth factors in vitro.

Optimum Growth Rate and the Influence of Genetics

What is the "optimal" growth rate in a prepubertal ruminant? What growth rate will be large enough not to limit necessary fat pad development but not so large as to depress growth hormone release and consequent mammary ductal proliferation?

Numerous trials with dairy heifers have revealed a curvilinear relationship between prepubertal growth rate and first lactation milk yield (see Sejrsen et al., 2000; Johnsson, 1988). Hohenboken, et al. (1995) found that when groups of Danish Friesian heifers were fed to achieve gains ranging between 0.50 kg/d and 0.90 kg/d, maximum milk yield was produced in groups gaining between 0.60 and 0.70 kg/d, or about 70-75% of maximum gain (Figure 4). By comparison, a group of British Friesians achieved 1.18 kg/d gain on an ad-lib diet (Harrison et al., 1988), and optimal parenchymal growth occurred at 0.57 kg/d, or about 50% of maximal gain. In trials with ewe lambs, improved mammary development occurred when lambs on restricted diets gained 74%, 53%, and 60% of the gain achieved on less-restricted diets (McCann et al., 1989, Johnsson and Hart, 1985, Umberger et al., 1985, respectively).

The genetic capacity for growth and milk yield has a large influence on determining the optimum gain for an individual or group. Hohenboken, et al. (1995), for instance, found that although the negative effect of feeding level existed and was similar across three dairy breeds, the optimum or injurious feeding level differed between breeds. Negative effects occurred when daily gain exceeded 0.35 kg/d in Jerseys, 0.55 kg/d in Danish Reds, and 0.65 kg/d in Danish

Friesians. Heifers from the larger breeds ate more, grew faster, were heavier at calving, and produced more milk than Jerseys.

In a trial involving Hampshire Down-cross ewe lambs (Johnsson et al., 1985), large variation in GH secretory patterns and mammary development were observed within the same treatment. They suggested that during prepubertal allometric growth, restricted feeding will not affect mammary development of genetically poor animals, but will allow better development of glands in animals with higher genetic potential. This implies that there may be a separate, independent, mechanism for the genetic expression of mammary parenchymal growth.

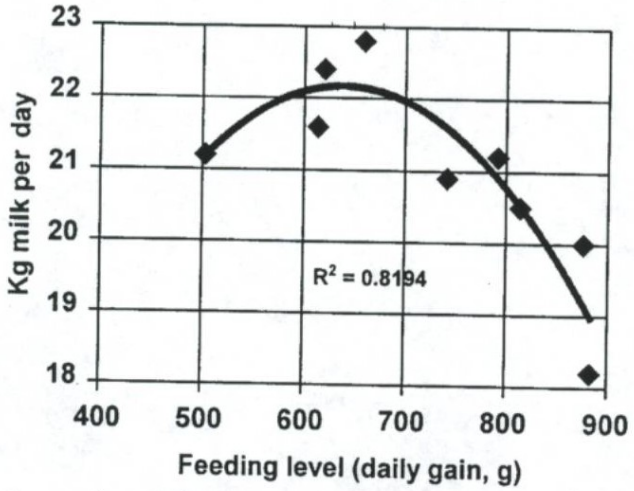

Figure 4. Effect of prepubertal feeding level on subsequent milk yield in Danish Friesian heifers. Each point represents the mean of 17-18 heifers. Data from Hohenboken et al., 1995; figure from Sejrsen et al., 2000.

Similarly, calves, heifers, and cows sired by bulls with high genetic potential for milk production were found to have higher circulating levels of blood GH than animals sired by bulls from a random population (Akers, 1985).

Sejrsen et al. (2000) noted that at the same level of feeding, the genetic capacity for higher milk production is positively correlated with the genetic capacity for higher growth. Therefore, for groups with greater milk yield potential, optimum daily gain would be higher, and for an individual with higher milk yield potential, gain above the optimum would have less negative impact on lifetime milk production. And, within a prepubertal group or breed, at an optimum feed level, a higher genetically-based growth rate will be reflected in higher milk yield (Figure 5), whereas a higher growth rate due to increased feeding will be reflected in reduced milk yield potential.

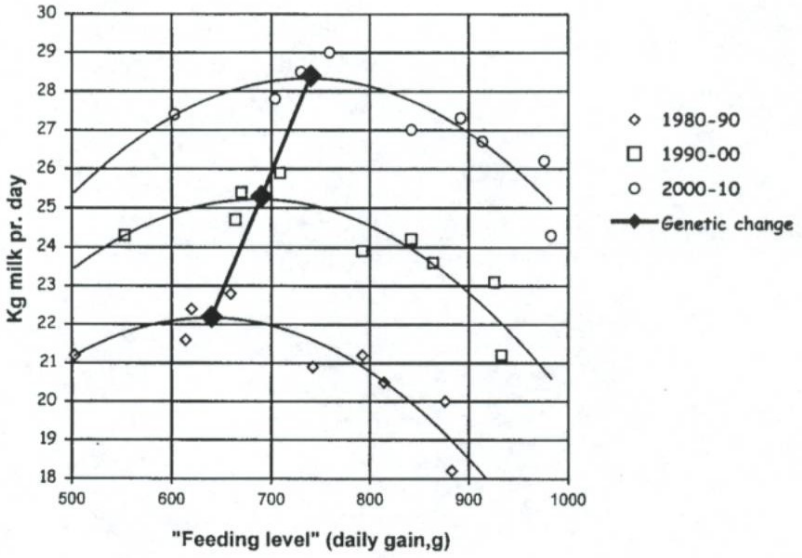

Figure 5. Illustration of the expected change in optimal daily gain with the increased genetic potential in milk yield in Danish dairy cattle from the 1980's to the 2000's. The relationship between feeding level and milk yield is unchanged and the higher optimal daily gain is achieved at the same feeding level. From Sejrsen et al., 2000.

Conclusions

Milk production is limited by the number of secretory cells in the udder. Diets excessive in energy, prior to puberty, inhibit the growth of the mammary epithelial tissue that will later sprout the milk-producing alveolar cells, and thus also limit lifetime milk yield. It is therefore recommended that dairy sheep producers should restrict the energy intake of replacement ewe lambs to about 65 to 75% of their ad-libitum intake. This will increase the rate of mammary growth and increase the total amount of epithelial tissue that will later develop into milk-secreting alveolar cells.

The negative impact of high-energy diets on ewe lambs is greatest starting at about 4 to 6 weeks of age and declines over the next few months of age, due to both the reduced concentration of circulating growth hormone and the lessening influence of growth hormone on mammary tissue. At the end of this phase of rapid duct extension, the absolute amount of ductal tissue, the degree of ductal penetration into the mammary fat pad, and the final size of the fat pad, will be primary determinants of the ultimate size of the adult lactating mammary gland.

Early puberty, especially before 20 weeks of age, may substantially reduce mammary gland development in rapidly-reared ewe lambs by curtailing both the rate and the duration of the allometric growth phase. Increased feed levels after 20 weeks have less influence on mammary development and can improve liveweight at breeding.

Acknowledgements

This study was funded by a grant from the Northeast Sustainable Agriculture Research & Education (SARE) Program. The authors are also grateful for the technical assistance provided by Mr Bruce Clement, UNH Cooperative Extension, Keene, NH, and by Dr. Michael Akers, Department of Dairy Science, Virginia Tech, Blacksburg, VA.

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