

# Nutrition - What You Feed Really Does Matter

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## Introduction

Cattle have an inherent growth pattern dictated by genetics. We manipulate the expression of growth by controlling energy intake, using implants, and by imposing stress. Low energy diets are used to suppress fat deposition while allowing skeletal growth to progress. We use high energy diets to maximize muscle growth, but do so with a concomitant increase in body fatness.

During growth, all tissues are not progressing at constantly proportional rates. In humans, it is obvious that the eyes of a newborn are quite large in proportion to their face. In cattle, we recognize visible examples of disproportionate growth when reproductive and mammary tissues begin to mature. There is evidence that this disproportionate growth, although not visible, is occurring in many tissue systems and perhaps also with the immune system.

Recently we have come to realize how this applies to development of intramuscular fat (IMF). This tissue develops earlier, rather than later, in growth. We still have much to learn, but we do know enough today to understand that management at the ranch and the transition to the feedlot will have a significant effect on Quality Grades in carcasses many months later. The goal here is to touch on some empirical observations from research (and my biases) about how early management affects the quality of beef we produce.

## Development of Marbling

It is important to start by noting that there is no evidence that we can induce an increase in IMF deposition. Genetics will dictate the maximum marbling potential. What we need to do is learn how not to impede that genetic potential.

Bruns et al. (2004) demonstrated the importance of early IMF deposition. Their results depicted in Figure 1 show that marbling increased linearly with carcass weight across a wide spectrum of the growth curve. The line for marbling is not parallel to the line for total fatness (depicted as ribfat). These data indicate that relatively early in growth, Quality Grade is increasing more rapidly than Yield Grade increases. Later in the growth curve, Yield Grade is increasing more rapidly than Quality Grade. The interpretation then is if we are going to alter the Percent Choice in a set of cattle, it will be done by early management.

Bruns et al. (2005) followed the first experiment with a study that confirmed the importance and sensitivity of early IMF deposition. Steers were implanted at nine months of age at 55% of mature size or implanted after reaching 75% of mature size. Implanting one time early (155 days before slaughter) increased carcass weight but caused a significant grade reduction from non-implanted controls. Delaying implanting until cattle reached 75% of mature size (85 days before slaughter) eliminated the grade reduction but provided the favorable carcass weight response of the implants (Table 1).

The time, age, or bodyweight window for this response is not yet defined. Suess et al. (1969) reported reductions in marbling due to early growth restriction. Meyers et al. (1999) reported increases in marbling in calves weaned early. One of their studies implies a narrow window of time that is critical. In that study, marbling at slaughter appeared related to growth rate at 177 to 231 days of age (Table 2). We have seen similar responses in the same age window (Pritchard et al., 1988).

## Managing Nutrition at Critical Times

There is no single perfect nutrition formula. We have two primary objectives: 1) to allow normal growth; and 2) provide nutrition necessary to support health. For growth, the principle factor is energy intake for the type of calf that you are producing. For our Frame Score 5+ cattle in the northern plains, it appears that average daily gain (ADG) from 5 to 9 months of age should exceed 2 lb. If these calves are implanted, or if Frame Score is 6, then ADG must be increased. If pasture and milk won't support that growth rate, it might be wise to wean early. Alternatively, creep feed can be used to achieve an IMF result that is better than if unsupplemented, but not as good as if calves are early weaned (Meyers, et al., 1999). When feeding calves, gains are always cheaper when ADG is higher. We need to take care to allow normal growth but to not produce calves with excess flesh. We all realize that buyers discount fleshy calves. If you are retaining ownership of the fleshy calves, performance to slaughter will be very good. Despite the early body flesh, the early weaned calves are very efficient. As young cattle at harvest they grade well. The drawback is that they may reach Yield Grade 3.0 at substantially lighter body weights.

How and what you feed is important for health, and health will make a difference in carcass value in the end. Gardner et al. (1999) reported the association between lung lesions and reductions in ADG and Quality Grades. The first thought here is that we need a good vaccination program, and I agree that pre-weaning vaccinations are a powerful tool. What I oftentimes see is that nutrition is overlooked when vaccinating suckling calves. The protein, energy, and trace minerals that are essential to get a good vaccine response in the feedlot are also essential on the ranch. Low CP pastures and inadequate mineral supplements can lead to vaccinated calves that are not immunized calves.

When calves are weaned and bunk broke at home, nutrition is just as important as if they were shipped directly to the feedlot. Last fall we fed 156 steer calves purchased directly from one ranch. There were 71 calves in this population that were weaned

on the truck to the feedlot. The other 85 calves were weaned and drylotted at the ranch 35 to 40 days prior to shipping. A balanced, total mixed diet was provided to the drylotted calves. Some had been weaned off two-year old dams. Others were weaned to spare drought-stressed pastures. Fed together during the first 35 days in our feedlot, we pulled 4.2% of the bawling calves and 15.3% of the bunk broke calves for bovine respiratory disease (BRD) or coccidiosis. All of the calves had been similarly vaccinated at the ranch prior to weaning. There was no co-mingling or sale barn travel confounding the outcomes. I interpret this as an example of a nutrition failure, by virtue of either a poor start at birth (calves on two-year olds) or poor range conditions at initial vaccination, or inadequate nutrient intake while drylotted.

In an unrelated experiment, we compared providing unlimited access to a receiving diet (46 Mcal  $NE_G$ /cwt) with managing intake at what we consider appropriate levels for adapting healthy, bawling calves ( $n = 191$ ) to feed. During the initial 45 days in the feedlot, the pull rate was 2.2% for managed calves and 17.8% for calves fed ad libitum. If each pull resulted in a decline in IMF deposition by 0.5 marbling score, the postweaning feeding program would have represented a 10% point swing in Choice carcasses. Our intake schedule is for 550 lb calves to be offered 3.5 lb of dry matter on day 1, 9 lb of dry matter on day 7 and 14 lb of dry matter on day 14. Our historical records indicate that intakes below this scale reflect the purchase of unhealthy cattle. We think we create sick cattle if intakes are allowed to get too high too soon. This concern with initial intake levels began with an evaluation of the receiving diet  $NE_G$  for fully preconditioned (PC) and bawling calves. All calves were allowed to express maximum feed intake. The morbidity scores in Table 3 show that PC calves on low energy diets were healthiest, but PC calves on the higher energy diet were the least healthy even though they had the greatest dry matter intake (DMI). Performance was improved only during this receiving period and would not have been of sufficient value to withstand a reduction in Percent Choice carcasses brought on by BRD.

There is still a great deal to learn about early nutrition effects on overall outcomes in beef production.

Vitamin E presents an interesting example. We know it is important for maintaining the health of the respiratory tract. Even so, outcomes from studies evaluating the role of Vitamin E on controlling BRD are variable. Is this because body stores masked responses? Alternatively, could it be that respiratory tissue responds like muscle tissue in that a large dosage of Vitamin E delivered in a short time span does not provide the tissue protection seen by feeding lower dosages for longer periods of time? If so, then Vitamin E nutrition to abate BRD starts at the ranch of origin. Similar concerns may apply to other nutrients as well.

## Summary

The composition of growth while calves are still at the ranch, and the immunocompetence developed at the ranch, impact feedlot production costs and carcass value. Growth and health are dictated by the plane of nutrition of the calf. Critical time windows for nutrition are late gestation-early lactation, and from five to nine months of age. Feedlots that feed for carcass specifications are already beginning to recognize that green calves (sought for compensatory growth) are subject to grade depression. In the future, they will probably become as concerned about calf nutrition during preconditioning as they currently are about which vaccines were used. What you feed your calves at home really does matter.

## References

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Table 1. Stage of growth response to E<sub>2</sub>-TBA implants<sup>a</sup> (Bruns et al., 2005).

	No Implant		BW at Implant	
	Control	650 lb	850 lb	
HCW	752 <sup>b</sup>	777 <sup>c</sup>	781 <sup>c</sup>	
Average Choice, %	24	8	23	
Low Choice, %	45	53	40	
Select, %	31	37	38	
Standard, %	0	2	0	

<sup>a</sup>Distributions effect (P<0.11).<sup>b,c</sup>Means differ (P<0.05).

Table 2. Early calf growth and marbling (Myers et al., 1999).

	Weaning Management			SEM
	Early	Creep	Normal	
ADG, lb				
177 – 231 d <sup>ab</sup>	3.17	1.81	1.37	0.11
231 – 443 d <sup>a</sup>	2.82	3.04	3.04	0.04
Marbling <sup>a</sup>	1,198*	1,144	1,120	18

<sup>a</sup>Early versus rest (P<0.01).<sup>b</sup>Creep vs. normal (P<0.05).

\*1,100 = Modest.

Table 3. Initial 28 day feedlot performance (Pritchard et al., 1998).

	Management			
	Control		Preconditioned	
Diet NE <sub>G</sub>	53	46	53	46
ADG <sup>a</sup>	3.28	3.37	3.87	3.74
DMI <sup>b</sup>	12.91	11.46	14.28	13.20
F/G	3.94	3.41	3.69	3.53
Morbidity pts <sup>c</sup>	170	148	224	122

<sup>a</sup>Management effect (P<0.05).<sup>b</sup>Diet effect (P<0.05).<sup>c</sup>Management x diet (P<0.05).

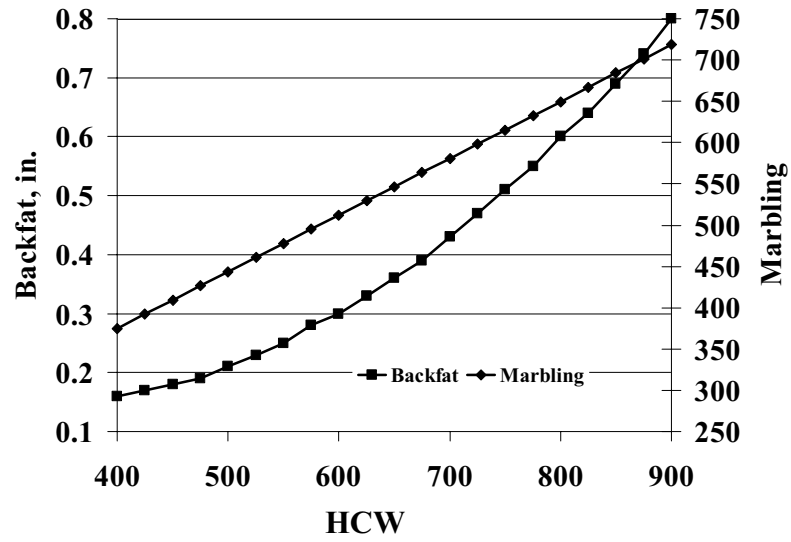


Figure 1. Backfat and marbling regressed against hot carcass weight (HCW).

## Notes: