ABSTRACT: It is well documented that grain feeding stimulates adipogenesis in beef cattle, whereas pasture feeding depresses the development of adipose tissues, including intramuscular (i.m.) adipose tissue. Additionally, production practices that depress adipocyte differentiation also limit the synthesis of MUFA. Marbling scores and MUFA increase in parallel suggesting that stearoyl-coenzyme A desaturase (SCD) gene expression is closely associated with and necessary for marbling adipocyte differentiation. Similarly, marbling scores and fatty acid indices of SCD activity are depressed in response to dietary vitamin A restriction. In bovine preadipocytes, vitamins A and D both decrease glycerol-3-phosphate dehydrogenase (GPDH) activity, an index of adipocyte differentiation, whereas incubation of bovine preadipocytes with L-ascorbic acid-2-phosphate increases GPDH activity. Exposing bovine preadipocytes to zinc also stimulates adipogenesis, putatively by inhibiting nitric oxide (NO) production. However, incubation of bovine preadipocytes with arginine, a biological precursor of NO, strongly promotes differentiation in concert with increased SCD expression. This suggests that the effect of either arginine or zinc on adipogenesis is independent of NO synthesis in bovine preadipocytes. Enhanced expression of SCD is associated with a greater accumulation of MUFA both in bovine preadipocyte cultures and during development in growing steers. In bovine preadipocytes, trans-10, cis-12 CLA strongly depresses adipocyte differentiation and SCD gene expression, thereby reducing MUFA concentrations. The bovine preadipocyte culture studies suggest that any production practice that elevates vitamins A or D or trans-10, cis-12 CLA in bovine adipose tissue will reduce i.m. adipose tissue development. Conversely, supplementation with vitamin C or zinc may promote the development of i.m. adipose tissue.

Key words: adipose tissue, bovine, fatty acid, intramuscular, stearoyl coenzyme A desaturase, vitamin

INTRODUCTION

The United States may lose its competitive advantage over Australia, South America, and other countries that have specifically targeted Asian countries for the export of high-quality beef. This would incur an economic loss of billions of dollars to the United States. In Japan, US beef was considered superior to Australian beef because 1) Australian producers could not produce beef as highly marbled as that from the United States, even in long-fed cattle; and 2) Australian cattle have harder fat than US cattle. Japanese and Korean consumers highly value both marbling (but not excess fat trim) and soft fat, but the distinction between US and Australian beef is disappearing.

There may be a downward trend in cattle that grade USDA Choice (i.e., a decline in the deposition of marbling adipose tissue) because of the loss of corn and other grains to the production of ethanol. Feeding distillers grain byproducts to feedlot cattle may cause an increase in fat hardness because of a reduction in the endogenous synthesis of MUFA in adipose tissues, although this could be offset by the increased concentration of PUFA in distillers grain by-products. Moreover, as the cost of grains for feedlot rations increases, more producers may delay the time at which cattle go into the feedlot. Marbling adipose tissue development appears to be especially sensitive to grain finishing early after weaning, and this window of opportunity may be

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lost. Also, hay- or pasture-feeding may lead to the absorption of fatty acids or other nutrients that specifically depress marbling adipogenesis. This review will address 1) the biology of marbling; 2) a comparison of the effects of hay/pasture and grain feeding on carcass and fat quality; and 3) vitamin supplementation strategies to promote marbling development in beef cattle.

**BIOLOGY OF MARBLING**

Marbling adipose tissue, also known as interfascicular or intramuscular (i.m.) adipose tissue, represents a unique depot. It can be distinguished from other fat depots by its location within perimysial connective tissues alongside myofibers (Moody and Cassens, 1968; Figure 1). Although some scientists have provided evidence for transdifferentiation of satellite cells to preadipocytes (e.g., Teboul et al., 1997; Singh et al., 2007), the localization of marbling adipocytes to the perimysium in most breed types would support the argument that marbling arises primarily from fibroblasts associated with perimysial connective tissue.

Several years ago, it was demonstrated that glucose contributes a greater proportion of acetyl units to fatty acid biosynthesis in i.m. adipose tissue than in subcutaneous (s.c.) adipose tissue (Smith and Crouse, 1984). In i.m. adipose tissue, acetate and lactate contributed less than 20% of the acetyl units to fatty acid biosynthesis, whereas glucose contributed approximately 70% of the acetyl units. The reverse was seen for s.c. adipose tissue; under these conditions, glucose contributed less than 5% to total acetyl units. In a more recent investigation (Lunt et al., 2005; Chung et al., 2007), a group of steers was fed a corn-based finishing diet to achieve a growth rate of 1.36 kg/d for 8 mo (short-fed, corn-fed), or were fed a hay-based diet supplemented with the same finishing ration to achieve a growth rate of 0.9 kg/d for 12 mo, with a targeted final BW of approximately 525 kg (short-fed, hay-fed). The corn-based diet contained 48% ground corn, 20% ground milo, 6% cottonseed meal, 15% cottonseed hulls, 7.5% molasses, 0.96% limestone, 0.56% trace mineral salt, and 0.08% vitamin premix on a DM basis. A second set of steers was fed the corn- or hay-based diets for 16 or 20 mo, respectively, to approximately 650 kg (long-fed, corn-fed and long-fed, hay-fed, respectively). In both s.c. and i.m. adipose tissue of the long-fed, corn-fed steers, fatty acid biosynthesis from acetate declined over 75% between 8 and 16 mo on diet in the corn-fed steers (Figure 2; Chung et al., 2007). Conversely, fatty acid synthesis from acetate increased slightly in both adipose tissue depots between 12 and 20 mo on diet in the hay-fed steers. Thus, in long-fed corn-fed steers, fatty acid biosynthesis from acetate provided a much smaller proportion of the substrate for lipid accumulation in s.c. and i.m. adipose tissues. Additionally, in both short-fed and long-fed hay-fed steers, fatty acid biosynthesis from acetate never contributed greatly to lipid accumulation in i.m. adipose tissue. Subcutaneous fat thickness and marbling scores increased over time in both the corn- and hay-fed steers (Chung et al., 2007); if lipid synthesis from acetate was depressed in long-fed steers, then what was the primary carbon source for de novo fatty acid biosynthesis in these steers?

It is likely that glucose replaced acetate as the primary substrate for fatty acid biosynthesis as the contribution of acetate declined in the long-fed steers. This is especially true for i.m. adipose tissue. In s.c. adipose tissue from long-fed corn- or hay-fed steers, rates of glucose and acetate incorporation into glyceride-fatty acids were similar (Figure 3; Rhoades et al., 2007). However, in i.m. adipose tissue of long-fed, corn- and hay-fed steers, the rate of glucose incorporation into fatty acids was more than twice as high as the rate of acetate incorporation. Thus, under conditions in which overall capacity for fatty acid biosynthesis is diminished (e.g., in long-fed cattle or in i.m. adipose tissue at virtually all stages of development), glucose provides a greater proportion of the acetyl units to lipid synthesis than acetate.

**PASTURE VS. GRAIN FEEDING**

In another recent study, steers were adapted at 8 mo of age to a corn-based finishing diet (fed as calves, CF), or were allowed to graze native pasture until 12 mo of age (fed as yearlings, YF) (M. A. Brooks and S. B. Smith, unpublished data). The corn-based diet was the same as described above. At 10 mo of age the YF steers were supplemented with the same concentrate ration to provide ADG of 0.9 kg/d. Subcutaneous and i.m. adipose tissue were dissected from the fifth to eighth thoracic rib section obtained at slaughter at 8, 12, and 16 mo of age. Adipose tissue samples were also obtained from the YF steers at 17.5 mo of age, at which time they had achieved the same BW as the CF steers (530 kg). The fat thickness measured at the 12th

![Figure 1. Marbling adipocytes located in perimysial seams of connective tissue. Samples were from the eighth to tenth rib section of the I.M. A large blood vessel is seen centrally located in the perimysium. M. A. Brooks and S. B. Smith (unpublished).](image-url)
thoracic rib increased at a greater rate between 8 and 12 mo in the CF steers than in the YF steers, but the YF steers had the same 12th-rib fat thickness by the final sampling time because of the rapid increase in the rate of fat gain between 12 and 17.5 mo of age (i.e., after being full-fed the high-corn finishing diet; Figure 4). Marbling scores were also similar between the CF and YF steers, primarily because there was no further increase in marbling scores in the CF steers between 12 and 16 mo of age (Figure 5).

Stearoyl-CoA desaturase (SCD) gene expression was barely detectable in s.c. adipose tissue of 8-mo-old weaned calves (Figure 6; not measured in i.m. adipose tissue at 8 mo of age). In both s.c. and i.m. adipose tissues, SCD gene expression was approximately 8-fold greater in CF steers than in YF steers, which was due to the greater concentrate intake in the CF steers between 8 and 12 mo of age. There was a sharp decline in SCD gene expression between 12 and 16 mo of age in s.c. and i.m. adipose tissues of the CF steers, whereas SCD gene expression in the YF steers increased linearly between 12 and 17.5 mo of age.

The SCD gene encodes Δ9-desaturase, which is responsible for synthesizing all MUFA from their respective SFA. The MUFA:SFA ratio increased with time on the corn-based diet in CF steers, but there was not a significant increase in the MUFA:SFA ratio in adipose tissues from the YF steers until after they were placed on the finishing diet (at 12 mo of age). The MUFA:SFA ratio reached a plateau by 12 mo of age in i.m. adipose tissue of the CF steers, and by 16 mo of age in i.m. adipose tissue of the YF steers. These data suggest that, although the MUFA:SFA appears to increase indefinitely in s.c. adipose tissue of feedlot steers, there is a limit to the concentration of MUFA that can accumulate in marbling adipose tissue. This may be related to the ability of cattle to marble, which is addressed below. These data are consistent with our previous reports that i.m. adipose tissue represents a more saturated adipose tissue depot than s.c. adipose tissue in both short- and long-fed, corn-fed cattle (Sturdivant et al., 1992; May et al., 1993).

**Relationship of Amount of i.m. Lipid to Fatty Acid Composition**

A final caveat of feeding corn to finishing cattle is that any increase in i.m. lipid (or marbling scores) typically is positively correlated with an accumulation of MUFA (Chung et al., 2006b; reviewed in Smith et al., 2006). An unusual aspect of the data indicated in Figure 5 is that marbling did not increase further between 12 and 16 mo of age in the CF steers. We do not know the reason for this, but the plateau in marbling scores in the CF steers is consistent with the decline in i.m. adipose tissue SCD gene expression (Figure 6) and the plateau in the MUFA:SFA ratio (Figure 7) seen during the same time in the CF steers. Previous research with porcine stromal-vascular preadipocytes (Ding and Mersmann, 2001) and porcine i.m. preadipocytes (Sanosaka et al., 2008) demonstrated that increasing the concentration of oleic acid in the culture medium promoted lipid filling and PPARγ gene expression. Taken together, the data suggest that grain feeding increases SCD gene expression, which elevates intracellular MUFA (and especially oleic acid), which subsequently promotes adipocyte differentiation and increases the concentration of MUFA.

These studies have demonstrated several factors that are important in producing high quality beef that contains high quality fat (i.e., soft fat enriched in MUFA): 1) corn feeding consistently produces high quality grades and adipose tissue enriched with MUFA; 2) feeding pasture and hay depresses SCD gene expres-
VITAMIN SUPPLEMENTATION IN LONG-FED CATTLE

Under some production conditions (i.e., in long-fed, grain-fed cattle) restricting or supplementing vitamin intake can enhance marbling deposition. This has been studied in detail in Japanese Black cattle, which typically are fed to at least 28 mo of age. Vitamin A

Farmers in Japan are likely to feed low-vitamin-A diets to beef cattle in Japan because of the improvement of carcass quality, although this may lead to increased incidence of blindness and muscular edema (Oka, 1999). Therefore, studies were conducted to scientifically elucidate this effect of vitamin A. Torii et al. (1996) and Adachi et al. (1999) reported that serum retinol concentration at the end of the finishing period was negatively correlated with beef marbling performance (r = −0.71, P < 0.001). Similarly, Gorocica-Buenfil et al. (2007) reported that mild dietary vitamin A restriction increased marbling scores of beef cattle, which was consistent with an elevation in apparent ∆9-desaturase activity (Siebert et al., 2006). However, the effects of vitamin A restriction on marbling scores and fatty acid ∆9-desaturation have been difficult to reproduce (Gorocica-Buenfil et al., 2007, 2008), possibly because of interaction with other dietary feedstuffs.

All-trans retinoic acid, the metabolite of retinol, is known to suppress adipocyte differentiation of mouse preadipocyte cell line through its repressive action on the expression of PPARγ (e.g., Ribot et al., 2001; Brandebourg and Hu, 2005). Bovine preadipocytes also express PPARγ, and the addition to 400 mg/L of retinol decreases glycerol-3-phosphate dehydrogenase (GPDH)
activity (an index of adipocyte differentiation) and the number of lipid-laden cells in a dose-dependent manner (Ohyama et al., 1998). These results suggest that all-trans retinoic acid, metabolized from retinol during culture, suppresses the differentiation of bovine adipocytes. The plasma retinol concentration in Japanese finishing cattle is about 300 mg/L, and a vitamin A-deficient diet decreased its plasma concentration to less than 90 mg/L. Therefore, retinol present in the blood circulation may act as the inhibitor of adipocyte differentiation in normal fattening beef cattle, and decreasing plasma vitamin A may remove its antiadipogenic action. The enhanced development of marbling by vitamin A restriction was thought to result from the effects of vitamin A on adipocyte differentiation. Oka et al. (1998) reported that marbling scores were significantly greater in the cattle given a vitamin A-deficient diet from 15 mo of age to final finishing (29 mo of age) than in the cattle given a vitamin A-sufficient diet. However, there was no significant difference in carcass marbling between cattle fed the vitamin A-deficient diet from 23 to 29 mo and the cattle fed the vitamin A-sufficient diet. An increase in adipocyte number within muscle occurs between 13 and 19 mo of age (Cianzio et al., 1985), and feeding a vitamin A-deficient diet during in this age range stimulated marbling (Oka et al., 1998).

Vitamin C

Unlike vitamin A, vitamin C has been shown to promote adipocyte differentiation of 3T3-L1 cells (Kawada et al., 1990). Domestic animals have not been considered to require dietary vitamin C because they can synthesize a sufficient amount of ascorbic acid in the liver (McDowell, 1989). However, Takahashi et al. (1999) reported that plasma concentrations of vitamin C decreased with advanced fattening in long-fed Japanese Black cattle. This result suggests that there may be a dietary requirement for vitamin C concentrations in long-fed cattle.

Torii et al. (1998) reported that ascorbic acid enhanced adipocyte differentiation of bovine adipocytes in vitro. The addition of l-ascorbic acid-2-phosphate into the culture medium increased GPDH activity of the cells at concentrations of 50 to 500 mM in a dose-dependent manner. These concentrations are equivalent to 0.9 to 9 mg/L. Plasma vitamin C concentrations of healthy beef cattle are within the range of 2.4 to 4.7 mg/L, but plasma concentrations of vitamin C can decrease to less than 2 mg/L in terminal fattening periods. In Japanese long-fed cattle, the greatest concentration of grains is fed during the last 3 to 4 mo of feeding (Zembayashi, 1994), which may affect ruminal production of vitamin C. These results raise the possibility that physiological vitamin C concentrations regulate bovine adipocyte differentiation. Ohashi et al. (2000) reported that in Japanese Black cattle, dietary supplementation of L-ascorbic acid-2-phosphate during the terminal fattening stage produced greater marbling scores but also fatter carcasses. Further investigation is needed to clarify the availability of vitamin C preparations for beef cattle and the most suitable supplementation periods for beef marbling, particularly in cattle receiving high-concentrate diets for extended periods.

Vitamin D

The liver and plasma concentrations of 1,25-dihydroxyvitamin D₃ were substantially less in Bos taurus English breed type cattle than in Bos indicus breed cattle. An increase in adipocyte number within muscle occurs between 13 and 19 mo of age (Cianzio et al., 1985), and feeding a vitamin A-deficient diet during in this age range stimulated marbling (Oka et al., 1998). This suggests that the effect of vitamin A removal on the marbling development depends on adipocyte differentiation; removing vitamin A improves beef marbling by stimulating i.m. adipocyte differentiation. Because reducing the dietary concentration of vitamin A also increases fatty acid indices of Δ⁹-desaturase activity (Siebert et al., 2006; Gorocica-Buenfil et al., 2008), this provides additional evidence that SCD gene expression is closely associated with and necessary for maximal adipocyte differentiation.

Figure 5. Marbling scores of steers adapted to a corn-based finishing diet as calves (calf-fed) or fed native pasture until 12 mo of age (yearling-fed). Steers were produced as described in Figure 3. 100 = Practically Devoid; 200 = Trace; 300 = Slight; 400 = Small; 500 = Modest. Each data point is the mean of 4 or 5 steers; pooled SEM are affixed to the symbols. M. A. Brooks and S. B. Smith (unpublished).
In the same study, marbling scores were greater in *Bos taurus* English cattle than in *Bos indicus* cattle. This suggests that elevated plasma concentrations of 1,25-dihydroxyvitamin D₃ may depress marbling scores. Similarly, Torii et al. (1995) reported that 1,25-dihydroxyvitamin D₃ suppressed ovine adipocyte differentiation. The action of 1,25-dihydroxyvitamin D₃ in 3T3-L1 cells is caused by repression of PPARγ gene expression (Hida et al., 1998). 1,25-Dihydroxyvitamin D₃ plays an important role in maintaining calcium homeostasis and reacts sharply to dietary calcium level. Thus, there is a possibility that an increase in plasma 1,25-dihydroxyvitamin D₃ production caused by decreased calcium intake could repress bovine preadipocyte differentiation, thereby decreasing marbling scores. Reiling and Johnson (2003) indicated no effect of supplemental vitamin D₃ on carcass-quality YF steers, but they did not report what form of vitamin D₃ was administered, nor did they report plasma or liver (i.e., storage) concentrations of 1,25-dihydroxyvitamin D₃.

In general, studies published to date suggest that water-soluble vitamins generally stimulate preadipocyte differentiation, whereas fat-soluble vitamins depress preadipocyte differentiation. This has been confirmed for marbling in animal studies, but only in long-fed cattle raised in Japan. Furthermore, it may be possible to demonstrate effects of vitamins A, C, and D₃ in cattle only after true deficiencies are established. Therefore, it may not be practical to use vitamin supplementation or withholding as a means for increasing the development of marbling in beef cattle.

**NITRIC OXIDE, ARGININE, AND ADIPOSE TISSUE DEVELOPMENT**

There is a growing paradox with respect to the potential impact of NO on adipose tissue differentiation. Nitric oxide is synthesized from L-arginine and O₂ by NO synthase (NOS) in almost all mammalian cells. Three
isoforms of NOS have been identified: nNOS, iNOS, and eNOS; nNOS was first identified in neuronal tissue, iNOS was shown to be inducible by cytokines, and eNOS was first identified in vascular endothelial cells. Only small amounts of NO are synthesized by nNOS or eNOS, whereas iNOS can be induced by cytokines to produce large amounts of NO (Meininger et al., 2000; Wu et al., 2001).

Supplementation of zinc to feedlot diets can increase marbling scores (e.g., Spears and Kegley, 2002), but the mechanism of action of zinc on adiposity is unknown. Zinc stimulates GPDH activity in preadipocytes from i.m. adipose tissue of mature Hanwoo cattle (Oh et al., 2004; Figure 8). Earlier, Hino et al. (2001) demonstrated an increase in lipid filling in Japanese Black perirenal preadipocytes treated with zinc during the differentiation period. Hino et al. (2001) also reported that the iNOS inhibitor, N-nitro-l-arginine methyl ester stimulated lipid filling of bovine preadipocytes. Because treatment of Hanwoo preadipocytes with zinc also caused a small reduction in NO (Figure 8), it is possible that zinc promotes preadipocyte differentiation by inhibiting iNOS activity.

Fu et al. (2005) demonstrated that dietary supplementation with arginine, which increased plasma NO concentrations, reduced adiposity in Zucker diabetic fat rats. After 10 wk of dietary arginine supplementation to the Zucker rats, those rats fed arginine (1.5% in drinking water vs. an alanine control group) had 30% less epididymal fat ($P < 0.05$) and 45% less abdominal fat. Dietary arginine supplementation increased the expression of several genes in rat epididymal adipose tissue, including PPAR$_\gamma$ (1.7-fold), phosphofructokinase (1.6-fold), and carnitine palmitoyltransferase I (1.5-fold). Paradoxically, increased expression of the first 2 genes should have increased lipid filling or glucose uptake in adipocytes, although carnitine palmitoyltransferase I (the rate-limiting enzyme in fatty acid oxidation) would have promoted lipid depletion of adipocytes. These findings support the hypothesis that supplementation with arginine depresses adiposity by increasing the production of NO.

However, whereas NO donors and dietary arginine supplementation to rats depressed adipocyte differentiation, treating rat preadipocytes (Yan et al., 2002) or bovine perirenal adipocytes (Chung et al., 2006a) with arginine strongly stimulated lipid filling and adipogenic gene expression (Figure 9). This raises the very real paradox that arginine per se may promote adipogenesis, and that the depression in adiposity caused by dietary arginine in whole-animal studies may not due to direct effects of NO on adipocytes. A recent article (Tan et al., 2008) reported that supplemental dietary arginine had no effect on s.c. adipose tissue amount in pigs, but increased the amount of i.m. lipid in the LM. Similar findings have not yet been reported for beef cattle, probably because of the inherent difficulties associated with ruminal metabolism of supplemental arginine. However, the report by Yan et al. (2002) suggests that i.m. adipocyte differentiation is enhanced by supplemental dietary arginine, similar to the response seen in rat and bovine preadipocytes exposed to arginine during culture.

**GOOD VS. BAD CLA**

No current discussion of adipose tissue differentiation would be complete without some mention of CLA. The 2 primary isomers of CLA, cis-9,trans-11 (c9,t11 CLA) and trans-10,cis-12 (t10,c12 CLA) have very different biological effects. Whereas c9,t11 CLA strongly depresses mutagenesis in cell lines (Pariza et al., 2000), t10,c12 CLA reduces adipocyte differentiation (e.g., Brown et al., 2003; Chung et al., 2006a).

Dietary CLA, especially t10,c12 CLA, has been demonstrated to reduce carcass adiposity in several studies. Pariza et al. (1996) first reported that mice, rats, and chicks fed diets containing 0.5% CLA for 4 to 8 wk experienced body fat reductions of 57 to 70, 23, and 22%, respectively. We subsequently demonstrated that

![Figure 8. Effect of zinc on glycerol-3-phosphate dehydrogenase (GPDH) activity (filled circles) and nitric oxide (NO$_2$ + NO$_3$; open circles) in adipocytes isolated from intramuscular adipose tissue of Korean (Hanwoo) cattle and induced to differentiate with insulin, dexamethasone, and 1-methyl-3-isobutyl-xanthine for 10 d. Zinc chloride (ZnCl$_2$) was supplemented at the concentrations of 0, 5, 25, 50, and 100 $\mu$M. Nitric oxide was measured after conversion to NO$_2$ and NO$_3$. Each data point is the mean of 3 preadipocyte culture replications; pooled SEM are affixed to the symbols. Adapted from Oh et al. (2004).](image-url)
mixed isomers of CLA depressed 3T3-L1 preadipocyte proliferation and [3H]thymidine incorporation into DNA (Satory and Smith, 1999). Dietary supplementation of pigs with CLA depressed [3H]thymidine incorporation into DNA of stromal-vascular cells of weanling pigs fed 3% mixed isomers of CLA in diets containing 15% total added fat (corn oil or beef tallow; Adams et al., 2005).

Although CLA caused only small reductions in adipocyte volume, it strongly depressed SCD gene expression and catalytic activity (Smith et al., 2002). The t10,c12 CLA isomer prevents lipid filling by decreasing PPARγ gene expression in rodent preadipocytes (Brown et al., 2003). Differentiation of bovine preadipocytes can be stimulated powerfully by the addition of PPARγ agonists (such as pioglitizone), insulin, and dexamethasone (Figures 9 and 10). Early differentiation is characterized by the expression of genes such as those encoding SCD. The t10,c12 isomer of CLA strongly depresses SCD gene expression, and thereby decreases the synthesis of MUFA (Figure 11). This is unusual in light of the fact that t10,c12 CLA is a product of rumen fermentation, and its accumulation would effectively block the conversion of trans-vaccenic acid (a primary product of ruminal fermentation) to c9,t11 CLA.

The strong depression in hepatic SCD gene expression caused by t10,c12 CLA may be responsible for its

![Figure 9](image)

**Figure 9.** Stearoyl-coenzyme A desaturase (SCD) and PPARγ gene expression in bovine stromal-vascular preadipocytes incubated in the absence (control) and presence of 5 mM Arg with and without 40 μM CLA. Preadipocytes were differentiated for 5 d in the presence of 5 μM pioglitizone, 10 μg/mL insulin, and Dulbecco’s modified essential medium. Samples were obtained from 3 replicate culture plates. Reprinted with permission from Chung et al. (2006a).

![Figure 10](image)

**Figure 10.** Stearoyl-coenzyme A desaturase (SCD) gene expression in bovine perirenal preadipocytes. Preadipocytes were differentiated in the presence of 5 μM pioglitizone, 10 μg/mL of insulin, and Dulbecco’s modified essential medium. The RNA from differentiated adipocytes was extracted after 7 d of treatment with differentiation medium, followed by 3 d of treatment with CLA. Reprinted with permission from Chung et al. (2006a).
“antiobesity” effect. Ntambi et al. (2002) demonstrated that SCD1 knockout mice do not express SCD in their livers and do not demonstrate the obese phenotype. However, reducing SCD activity may cause hepatic triglyceride accumulation (Miyazaki et al., 2001), leading to fatty liver. It may be prudent to consider c9, t11 CLA as “good” CLA but t10, c12 CLA as “bad” CLA. From a production standpoint, clearly any production practice that profoundly increases the formation and absorption of t10, c12 CLA from the digestive system will depress adipogenesis (including i.m. adipose tissue development), and will cause adipose tissue lipids to become more saturated.

**SUMMARY**

Producing well-marbled beef necessitates the feeding of grains to calves ideally shortly after weaning. The longer cattle are fed grain-based diets, the more marbling they accumulate and the greater the concentration of MUFA in their adipose tissues; these 2 responses to grain feeding appear to be tightly linked. Unfortunately, extended feeding of grain-based finishing diets may become impractical in light of the rising costs and limited availability of corn as grain production is diverted to ethanol production. Under some production conditions, and especially during extended feeding of grain-based diets, supplementation with vitamins A or D may depress the development of i.m. adipose tissue, whereas supplementation with vitamin C may actually enhance i.m. adipose tissue development. Although arginine supplementation has been shown to depress carcass adiposity, it may actually enhance the differentiation of preadipocytes within i.m. adipose tissue beds. Finally, any production practice that elevates t10, c12 CLA in bovine adipose tissue likely will reduce i.m. adipose tissue development.

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