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# Postnatal development of stearoyl coenzyme A desaturase gene expression and adiposity in bovine subcutaneous adipose tissue

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# Postnatal Development of Stearoyl Coenzyme A Desaturase Gene Expression and Adiposity in Bovine Subcutaneous Adipose Tissue[1]

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**ABSTRACT:** This investigation addressed the hypothesis that stearoyl coenzyme A desaturase (SCD) gene expression would serve as a postnatal marker of adipocyte differentiation in bovine s.c. adipose tissue. Samples of tailhead s.c. adipose tissue were obtained by biopsy from preweaning steer calves 2.5 wk, 5 mo, and 7.5 mo of age and from yearling steers 12 mo of age. Samples also were obtained at slaughter when the steers were 18 mo of age. The steers sampled as yearlings were fed native pasture from weaning until 12 mo of age, and the steers sampled at slaughter were fed a high-concentrate diet from 12 to 18 mo of age. Major peak adipocyte volumes for the 2.5-wk-, 5-mo-, and 7.5-mo-old steers were 14, 270, and 700 pL, respectively ($P < .001$). The steers did not gain weight during pasture feeding, and at 12 mo of age peak adipocyte volume had decreased ($P = .009$) to 270 pL. At this time, a second, smaller population of adipocytes had appeared with a peak volume of 115 pL. At slaughter, adjusted fat thickness of the steers was 1.60 ± .13 cm, the USDA yield grade of the carcasses was 3.51 ± .31, and peak adipocyte volume had increased ($P = .01$) to over 2,500 pL. The number of adipocytes per 100 mg of adipose tissue doubled ($P = .006$) between 2.5 wk and 5 mo of age, concurrent with the nearly 20-fold increase in peak adipocyte volume, indicating that this was a period of apparent adipocyte hyperplasia. Uncoupling protein mRNA was undetectable at all stages of postnatal growth, indicating that differentiating tailhead s.c. adipocytes do not acquire brown adipocyte characteristics postnatally. Lipogenesis expressed on a cellular basis was low in all preweaning samples and increased significantly above preweaning values only in the 18-mo-old steers. Stearoyl coenzyme A desaturase mRNA concentration also was low in all preweaning samples, but it peaked ($P = .07$) at 12 mo of age. Because the peak in SCD mRNA concentration preceded a significant rise in lipogenesis and lipid filling, we conclude that the level SCD gene expression may be indicative of the extent of terminal differentiation in bovine tailhead s.c. adipose tissue.

Key Words: Adipocytes, Differentiation, Acyl-CoA Desaturase, Protein, Genes, Cattle

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## Introduction

As preadipocyte cell lines such as 3T3-L1 preadipocytes exit proliferation and begin to differentiate, expression of the genes encoding de novo lipogenesis (e.g., ATP-citrate lyase and acetyl coenzyme A carboxylase) increases markedly and measurably precedes lipid filling (Bernlohr et al., 1985). In bovine adipose tissue in vivo, de novo lipogenic enzyme activities and lipogenesis from acetate are virtually undetectable at weaning, despite extensive lipid filling of adipocytes by this time (Smith et al., 1984; Schiavetta et al., 1990). Thus, extensive adipocyte differentiation precedes induction of the genes encoding de novo lipogenic enzymes by several months in bovine adipose tissue.

In the current investigation, we hypothesized that stearoyl coenzyme A desaturase (**SCD**) gene expression would precede de novo lipogenesis in bovine adipose tissue. In doing so, SCD gene expression would serve as a marker of terminal differentiation (i.e., lipid filling) in adipose tissue from this species. To address our hypothesis, we measured SCD mRNA, adiposity, and lipogenesis in tailhead s.c. adipose tissue from steer calves at several times before and after weaning.

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## Materials and Methods

*Animals.* Thirty male Angus calves were selected from a spring calf crop from the Texas A&M University Agricultural Research, McGregor. Calves were allotted to one of five sampling groups and were sorted to minimize variation in age within each sampling group. Calves were castrated at 1 to 2 wk of age. Ages at sampling were 2.5 wk and 5, 7.5, 12, and 18 mo (Table 1). Calves either were returned to native bermudagrass pasture with their respective dams (preweaning to yearling) or were returned to the feedlot pens after sampling. Calves from all groups were weaned at approximately 229 d of age (214 kg). The yearling calves were adapted over a 3-wk period to a concentrate diet (control diet of Huerta-Leidenz et al., 1991). Calves were weighed at birth and at weaning; the calves sampled at 12 mo were weighed again at sampling, and the calves sampled at 18 mo were weighed approximately every 4 wk from weaning until slaughter.

*Adipose Tissue Sampling.* The animal protocol was approved by the Texas A&M University Laboratory Animal Care and Use Committee (AUP#93-671). S. B. Smith was certified in asepsis and aseptic techniques and in the procedure for collecting adipose tissue from the tailhead region of calves by the Texas A&M University Animal Care and Use Training Program.

Calves either were restrained on a wooden pallet (2.5-wk-old calves) or in a squeeze chute (other age groups). An area of approximately 200 $cm^2$ was sheared closely and then alternately scrubbed three times each with ChlorHex Scrub (Vedco, St. Joseph, MO) or 70% ethanol. A total of 2 mL of 2% lidocaine HCl (Western Veterinary Supply, Porterville, CA) was injected in three to four locations around the biopsy site. After 5 min, a V-shaped incision, approximately 5 cm on each side, was made through the hide with a sterile scalpel. The incision was made 10 cm laterally from the medial line and 10 cm cranially from the base of the tail. The flap was lifted by gently scraping the underlying connective tissues. All s.c. adipose tissue within the biopsy site was removed; a portion was snap-frozen in liquid nitrogen, and the remainder was used for the measurement of lipogenesis and cellularity. The underlying muscle was left unharmed. After removal of the adipose tissue, the area was blotted with sterile gauze and the incision was stapled closed (Royal AutoSuture Skin Stapler, United States Surgical Corp., Norwalk, CT). The area of the incision was sprayed with Furox (10% furazolidone; Sovay Animal Health, Mendota Heights, MN) and screw worm spray (Anchor Products, St. Joseph, MO).

Samples were obtained from the 18-mo-old steers at slaughter. Cattle were transported approximately 170 km to the Texas A&M University Rosenthal Meat Science and Technology Center. Tailhead s.c. adipose tissue was sampled immediately after removal of the hide, from the same area as was sampled by biopsy technique from the other age groups. Carcass data were collected after 48 h at 4°C by trained Texas A&M University personnel.

*Lipogenesis In Vitro.* Pieces of tailhead s.c. adipose tissue (50 to 100 mg) were incubated fresh in duplicate in 3 mL of Krebs-Henselheit buffer (pH 7.4) as described by Smith et al. (1996) with modifications. Flasks contained 10 m*M* acetate and 10 m*M* glucose plus 1 $\mu$Ci of [1-$^{14}$C]acetate per flask in Krebs-Henselheit buffer. Subsequent extraction steps were as described previously (Smith et al., 1996).

*Cellularity.* Adipocytes were liberated from fresh adipose tissue samples as described previously (May et al., 1994). Adipose tissue samples were frozen at –25°C and sliced in 1-mm-thick sections to facilitate tissue fixation. Fixed cells were filtered through 240-, 64-, and 20-$\mu$m nylon mesh screens using .01% Triton in .154 *M* NaCl. The cells from the 20-$\mu$m screen were counted and sized with a Coulter Counter, Model ZM, equipped with a channelizer, Model 256 (Coulter Electronics, Hialeah, FL), using a 280-$\mu$m aperture. The cells from the 64-$\mu$m screen were counted on the same equipment with a 400-$\mu$m aperture. Adipocyte sizes were divided into 10-$\mu$m intervals; those counted with the smaller

Table 1. Growth characteristics of pre- and postweaning calves

| | Group | | | | | |
|---|---|---|---|---|---|---|
| Item | 2.5 wk | 5 mo | 7.5 mo | 12 mo | 18 mo | SEM |
| Birth weight, kg | 32.8 | 34.4 | 29.8 | 34.2 | 32.6 | .9 |
| Weaning weight, kg | 165.4[a] | 203.8 | 213.5 | 205.0 | 203.8 | 4.5 |
| Average daily gain to weaning, kg/d | .74[a] | .85 | .80 | .82 | .88 | .02 |
| Age at sampling, d | 17.8 | 158.8 | 228.7 | 360.8 | 542.0 | 33.2 |
| Weight at sampling, kg[b] | 45.9 | 170.6 | 213.5 | 227.8 | 487.1 | 31.0 |

[a]Weaning weight and average daily gain of the calves sampled at the youngest age (2.5 wk) were less ($P = .004$) than values for the other age groups.

[b]Sampling weights for the 2.5-wk-old and 5-mo-old calves were calculated from average daily gains from birth to weaning. Sampling weights for the other groups were actual weights at each age (n = 6).

aperture were observed in channels between 20 and 60 μm, whereas those counted with the larger aperture were in channels ranging from 60 to 190 μm. Cells occurring in the 60-μm channels from both apertures were summed.

*Preparation and Analysis of RNA.* Total RNA was isolated from adipose tissue samples in Tri Reagent (Sigma Chemical Co., St. Louis, MO), which essentially employs the guanidine thiocyanate-phenol-chloroform extraction method of Chomczynski and Sacchi (1987). Because of the high lipid content of the samples, an additional phenol, chloroform, isoamyl alcohol (25:24:1, vol/vol) extraction step was included. Yield and purity were determined by measurement of absorbances at 260 and 280 nm.

For slot blot analysis, 11 μg of total RNA was incubated at 65°C for 5 min in three volumes (vol/vol) of the following solutions: 500 μL of formamide, 162 μL of formaldehyde (37% solution), and 100 μL of 10× MOPS (20 m*M* 3-[N-morpholino] propane sulfonic acid), .005 *M* sodium acetate, 5 m*M* EDTA (pH 7.0). Samples were chilled on ice and one volume of ice-cold 20× SSC was added. The RNA was applied to an OptiTran nitrocellulose membrane (Schleicher & Schuell, Keene, NH) in the slots of a commercial slot blot apparatus (Schleicher & Schuell). The membranes were prehybridized in 5× SSC, 5× Denhardt's, .5% SDS, and .1 mg/mL salmon sperm DNA at 55°C for 2 to 3 h.

A porcine SCD cDNA was generously provided by N. Nathalie, Faculté des Sciences Agronomiques de Gembloux, UER Biologie Moléculaire et Physiologie Animale, Belgium. The SCD cDNA-containing plasmid was digested with *Sca*I, and the insert was amplified by PCR using the universal sequencing primers (Gibco BRL Life Technologies, Grand Island, NY). The PCR-generated SCD DNA was radiolabeled with [$\alpha$-$^{32}$P]dCTP with the Random Primers DNA Labeling System (Gibco BRL Life Technologies) and hybridized to the RNA at 55°C for 16 h in the prehybridization solution without the salmon sperm DNA. A duplicate blot containing 1 μg of total RNA was hybridized with a radiolabeled mouse Deca Probe 18S rat ribosomal cDNA (Ambion, Austin, TX).

An uncoupling protein (**UCP**) DNA also was generated by PCR. The template DNA was the bovine calf UCP cDNA (generously provided by L. Casteilla, Centre de Recherche, CNRS, France) linearized with *Eco*RI. The primers were 5′-CTC AGC GGG CCT AAC GAC-3′ and 5′-GTT TGT TTT TCA CCA GGG-3′, which produced a PCR product approximately 350 bp in size. The UCP probe has been used previously in our laboratory to quantify UCP mRNA in bovine perirenal brown adipose tissue (Martin et al., 1997, 1998). The PCR-generated UCP probe was radiolabeled with [$\alpha^{32}$P]dCTP by random primer method and hybridized and rinsed as described above. One microgram of total RNA, extracted from the perirenal (brown adipose) tissue from one newborn calf, was used as a positive control.

Rinsed membranes for all probes were placed with Kodak X-AR5 x-ray film (Eastman Kodak, Rochester, NY) at –70°C for 1 to 10 d. After autoradiography, slot blots were scanned using an LKB 2202 Ultroscan Laser Densitometer (Bromma, Sweden), and the intensities of the bands were determined. The density of the SCD mRNA bands was corrected by ribosomal RNA.

*Source of Chemicals.* Unless otherwise stated, biochemicals were purchased from Sigma Chemical Co. and Fisher Scientific (Fairlawn, NJ). Radiolabeled materials were obtained from Amersham (Arlington Heights, IL).

*Data Analysis.* Data were statistically analyzed by the Super Anova program (Abacus Concepts, Berkeley, CA). The model was a one-way analysis of variance (Guenther, 1964), and the source of variation was age. Data are presented as means ± overall SEM. If the age effects were significant ($P < .05$), means were separated with the Fisher's Protected LSD method, which was contained in the same software program.

## Results

*Growth and Carcass Characteristics.* Birth weights were not different ($P = .54$) among sampling groups (Table 1). Average daily gains and weaning weights were significantly lower for the 2.5-wk sampling group, suggesting that obtaining biopsies from the youngest calves impaired subsequent growth.

Calves were not weighed between birth and weaning. Body weights at the 2.5-wk and 5-mo sampling periods thus were estimated from the calculated average daily gains (Table 1; Figure 1). Actual body weights were obtained for all subsequent sampling groups. The steers sampled at 12 and 18 mo exhibited markedly depressed growth between weaning and 12 mo of age (i.e., while on pasture). Growth rate increased markedly after adaptation to the high-concentrate diet (Figure 1). Fat thickness and USDA yield grades for the 18-mo-old steers indicated that the cattle had accumulated considerable adipose tissue mass by the end of the experiment (Table 2).

*Adiposity.* Virtually all of the adipocytes from the 2.5-wk-old calves were less than 40μm in diameter (Figure 2a), with a peak volume of 14 pL (Figure 2b). Tailhead s.c. adipose tissue from these calves was scant and resembled a loose mesh of connective tissue rather than adipose tissue.

By 5 mo of age, the small (< 40 μm diameter) population of adipocytes still was predominant. However, by this time a second, distinct population of adipocytes had appeared with a peak diameter of 80 μm (Figure 2a). This population of adipocytes was

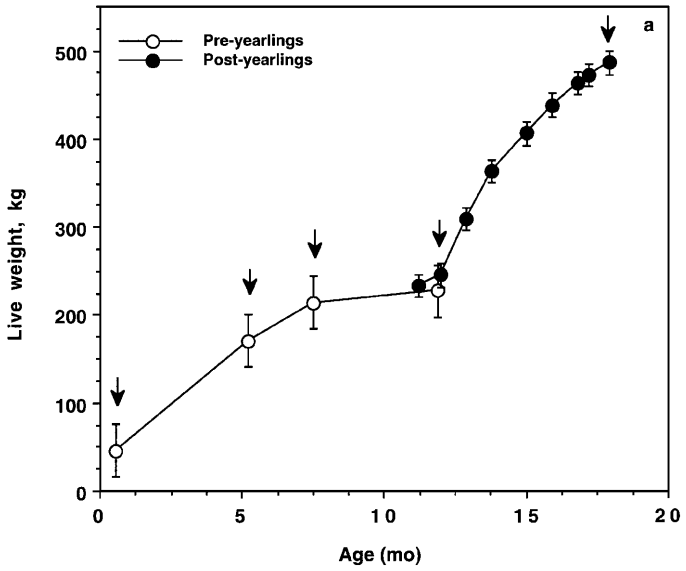


Figure 1. Live weights during the course of the experiment. Pre-yearling weights are those for each sampling group up to and including the 12-mo-old steers. Post-yearling weights were taken at approximately 28-d intervals for the steers to be sampled at 18 mo of age. Body weights at 2.5 wk and 5 mo were calculated from average daily gains for each sampling group. Overall standard errors of the means are attached to the symbols for each group. Arrows indicate sampling times.

larger still by 7.5 mo of age, with a peak diameter of 110 $\mu$m and a smaller proportion of adipocytes as large as 190 $\mu$m.

There was a dramatic shift in diameter distribution in the 12-mo-old, postweaning steers (Figure 2a). A new population of adipocytes, with a peak diameter of 30 $\mu$m, had appeared, and the larger population of adipocytes had atrophied to 80 $\mu$m peak diameter. These peaks persisted, albeit in lesser proportion, to 18 mo of age. However, by this time a much larger population of adipocytes had appeared (peak diameter = 170 $\mu$m, approximately 2,600 pL peak volume).

Table 2. Carcass characteristics of the 18-mo-old steers

| Item | Value | SEM |
|---|---|---|
| Carcass weight, kg | 292.9 | 7.9 |
| Adjusted fat thickness, cm | 1.60 | .13 |
| Longissimus muscle area, $cm^2$ | 70.8 | 3.8 |
| Kidney, pelvic, and heart fat, % | 2.42 | .15 |
| Marbling score[a] | $Sm^{61}$ | 23 |
| Yield grade | 3.51 | .31 |
| Quality grade[b] | $Ch^{20}$ | 8 |

[a]$Sm^{61}$ = $Small^{61}$, based on a 0-to-100 scale.
[b]$Ch^{20}$ = $Choice^{20}$, based on a 0-to-100 scale.

Mean adipocyte volumes for the 2.5-wk, 5-mo, and 7.5-mo-old steers were 14.3, 165.6, and 325.2 pL, respectively ($P < .001$) (Figure 3a). At 12 mo of age (after 4.5 mo of pasture feeding), mean adipocyte volume decreased ($P = .009$) to 117.1 pL; by 18 mo of age, mean adipocyte volume had increased ($P = .01$) to 321.6 pL. The number of adipocytes per 100 mg of adipose tissue doubled ($P = .006$) between 2.5 wk and 5 mo of age, concurrent with 11-fold increase in mean adipocyte volume (Figure 3a). In all other age groups, there was a reciprocal relationship between mean adipocyte volume and cell density.

*Gene Expression and Lipogenesis.* Uncoupling protein mRNA was not detectable at any sampling period, although the UCP probe bonded strongly to the RNA

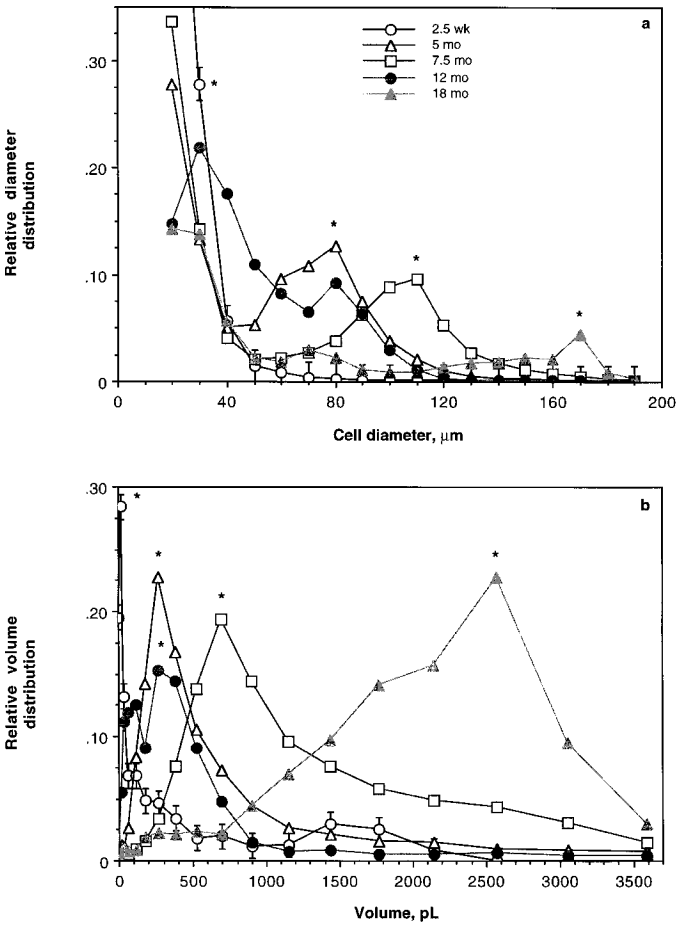


Figure 2. Changes in adipocyte diameter (a) and volume (b) during growth in preweaning (2.5 wk to 7.5 mo) and postweaning Angus steers. The relative proportion of adipocytes at 20 $\mu$m diameter for the 2.5-wk-old calves (off scale in a) was .632. Overall standard error of the mean is affixed to the symbols for the 2.5-wk-old steers. Values with asterisks are greater ($P < .05$) than lesser values at the indicated cell diameters and volumes. The relative volume distributions for the 5-mo-old and 12-mo-old steers at 270 pL were different at $P = .07$.

from perirenal brown adipose tissue (data not shown). The SCD:18S ratio was low until weaning and doubled ($P$ = .07) between 7.5 and 12 mo of age. Lipogenesis from acetate, expressed per 100 mg of adipose tissue, also was low throughout the suckling period but was four- to sixfold greater ($P$ < .001) in the 12- and 18-mo-old steers than in the preweaned calves (Figure 3b). On a cellular basis, the rate of lipogenesis remained depressed until 12 mo of age. Only in samples from the 18-mo-old steers was the rate of lipogenesis from acetate significantly greater than rates observed in the preweaning calves.

## Discussion

A long-term goal of this laboratory is to document the ontogenic development of bovine adipose tissue, and we have focused on tailhead s.c. adipose tissue due to its accessibility in fetal and postnatal calves. Tailhead s.c. adipose tissue seems to differentiate in fetuses as brown adipose tissue, because we have observed typical brown adipocytes (i.e., adipocytes containing numerous highly differentiated mitochondria) in tailhead s.c. adipose tissue of newborn calves (Martin et al., 1998). We also have measured UCP mRNA in tailhead s.c. adipose tissue of fetal Angus and Brahman calves (S. B. Smith and G. E. Carstens, unpublished observations). Unlike perirenal adipose tissue, in which the concentration of UCP mRNA increases during the last trimester (Casteilla et al., 1989; Martin et al., 1998), the UCP mRNA concentration in s.c. adipose tissue declines steadily during the last trimester of fetal growth. We wished to determine whether preadipocytes that differentiated postnatally in tailhead s.c. adipose tissue also expressed UCP mRNA. This seems not to have been the case, because we were unable to detect UCP mRNA in the tailhead s.c. adipose tissue at any stage of postnatal development.

We previously demonstrated that the monounsaturated:saturated fatty acid ratio increases from approximately .9 to 1.3 in bovine tailhead s.c. adipose tissue during the first 8 mo postweaning and suggested that desaturase enzyme activity increased with age during this period (Huerta-Leidenz et al., 1991). The results of the current study were consistent with our earlier observations, in that SCD mRNA concentration (and, presumably, SCD enzyme activity) increased significantly after weaning.

What cannot be discerned in this study is whether the increase in SCD gene expression was due to the dramatic changes in diet (milk, forage, or grain) or maturation of the steers. Ntambi (1992) demonstrated that SCD mRNA was over fivefold higher in the livers of feed-restricted mice refed either tripalmitin or tristearin than in livers of feed-restricted mice refed a chow diet, providing strong support for the regulation of SCD gene expression by saturated fatty acids. The 18-carbon fatty acids present in pasture and the high-concentrate finishing diet would have been hydrogenated largely to stearic acid in the rumen (Ekeren et al., 1992). We cannot rule out the possibility that the elevated SCD mRNA we observed in s.c. adipose tissue from postweaning calves was caused by up-regulation of SCD gene expression in response to dietary 18-carbon fatty acids. However, if this was the case, we would have expected a larger response to adaptation to the high-concentrate diet, because it would have contained at least four times as much total lipid (approximately 4%) as native pasture.

In slaughter-weight steers and heifers, SCD enzyme activity and mRNA concentrations typically are

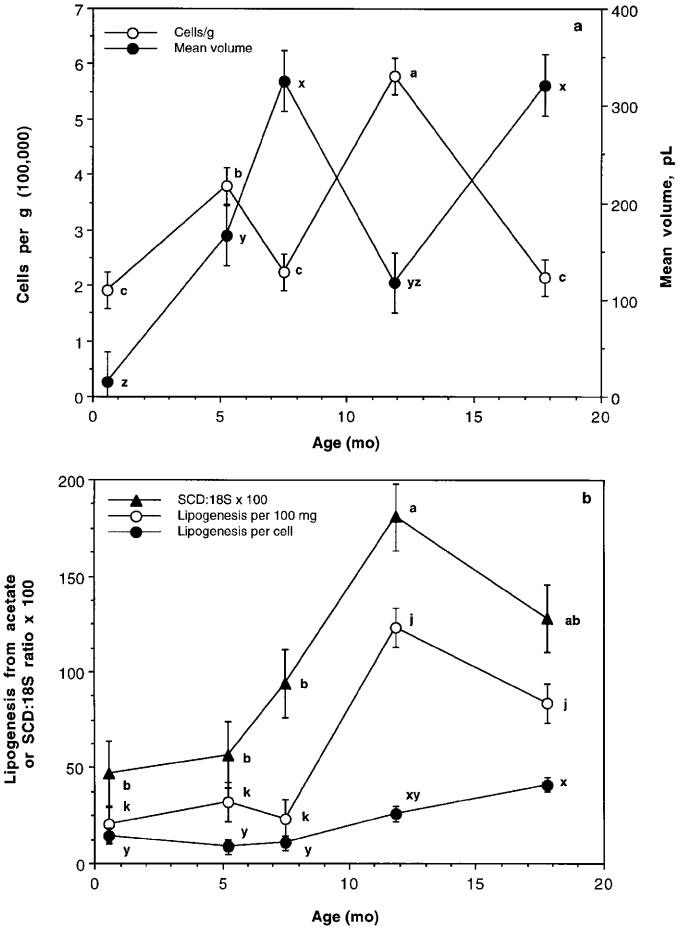


Figure 3. Changes in cellularity, lipogenesis, and stearoyl coenzyme A desaturase (SCD) gene expression during growth in preweaning (2.5 wk to 7.5 mo) and postweaning Angus steers. (a) Mean values for number of adipocytes per gram of adipose tissue and adipocyte volumes. (b) Lipogenesis and SCD gene expression. Overall standard errors of the means are affixed to the symbols for each item. [abc,jk,xy]Values within a measurement with the same superscript were not different ($P$ > .05).

highest in s.c. adipose tissue, intermediate in longissimus muscle, and low to undetectable in liver (St. John et al., 1991; Chang et al., 1992; Cameron et al., 1994). We hypothesized that the especially high level of SCD gene expression in bovine adipose tissue may be an indication of terminal differentiation of preadipocytes in fattening cattle. In 3T3-L1 preadipocytes, SCD mRNA is undetectable during the proliferative phase; upon differentiation, SCD mRNA increases 30-fold as the result of increased gene transcription (Bernlohr et al., 1985; Ntambi et al., 1988). More recently, SCD gene expression has been described as a marker for terminal differentiation (i.e., when preadipocytes irreversibly exit the proliferative phase, express lipogenic enzymes, and begin to fill with lipid; Ailhaud et al., 1992; Cornelius et al., 1994).

Robelin (1981) demonstrated apparent s.c. adipocyte hyperplasia between 4 and 7 mo of age and between 13 and 16 mo of age (summarized in Figure 4). A similar pattern was demonstrated by Cianzio et al. (1985) (i.e., apparent hyperplasia between 13 and 15 mo of age). We previously reported a significant increase in 9-10-11th rib s.c. adipocytes from 11 to 13.5 mo of age in Angus steers fed a high-concentrate diet (adjusted to total carcass s.c. adipocytes in Figure 4) (Schiavetta et al., 1990). These and other studies (e.g., Truscott et al., 1983) have provided indirect evidence for adipocyte hyperplasia in postweaning cattle. We also demonstrated [$^3$H]thymidine incorporation into DNA in s.c. and i.m. adipose tissue explants from heavy Angus and Wagyu steers (May et al., 1994), supporting the occurrence of preadipocyte hyperplasia in mature cattle. Therefore, we hypothesized that we would observe periods of elevated SCD gene expression as preadipocytes exited the proliferative phase and entered terminal differentiation. Furthermore, we hypothesized that this would precede, or be concurrent with, the onset of de novo lipogenesis.

The yearling steers of the present investigation inadvertently may have provided us with a unique model to investigate preadipocyte differentiation in vivo. During pasture feeding between weaning and 12 mo of age, the steers gained little weight, and the adipocytes decreased in peak volume from 700 to 270 pL. The weaned cattle were fed on native pasture during the summer months and experienced high temperature and humidity and low pasture quality. Thus, the reduction in adipocyte volume was caused by reduced energy intake (e.g., Smith and McNamara, 1990). During this time, a second peak of 115 pL appeared. Whereas the 270-pL peak was certain to represent adipocytes that had dilipidated in response to the high temperature and(or) poor pasture conditions, it is less clear why the second, smaller peak appeared during the time on pasture. The 115-pL peak may have represented a second population of dilipidated adipocytes, although it is unlikely that two distinct populations of dilipidated adipocytes would result from reduced energy intake. Alternatively, the 115-pL peak may have represented adipocytes that either had been recruited from a previously quiescent population or that resulted from true hyperplasia.

Between 12 and 18 mo of age, adipocytes underwent extensive lipid filling, such that 2,600-pL volume adipocytes constituted over 20% of the total adipocyte volume. Lipogenesis (per $10^5$ cells) rose steadily between weaning and slaughter, whereas SCD gene expression reached a peak at 12 mo of age. Thus, as we had hypothesized, SCD gene expression preceded de novo lipogenesis. Taken together, the gradual increase in lipogenesis and marked increase in SCD gene expression following weaning suggest that terminal differentiation of a population of preadipocytes in tailhead s.c. adipose tissue occurred during this period. If this is true, then some proportion of the preadipocytes present in the tailhead s.c. adipose tissue lost the ability to proliferate during the postweaning period as they entered terminal differentiation. The increase in cells per gram that we

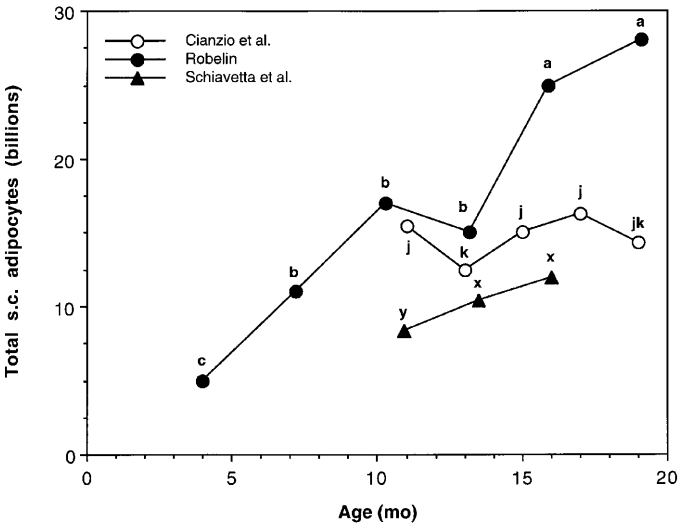


Figure 4. Apparent hyperplasia in s.c. adipose tissue from growing steers and bulls. Data are from Robelin (1981; Friesian bulls weaned at 4 mo of age and Charolais bulls weaned at 7.2 mo of age; bulls were fed 82% concentrate after weaning); Cianzio et al. (1985; Limosin, Maine Anjou, Angus, and Simmental crossbred steers weaned at 6 mo of age and fed 72% ground, shelled corn after weaning); and Schiavetta et al. (1990; Angus steers weaned at 6 mo of age, fed pasture until 11 mo of age, and fed 66% yellow corn/ground milo thereafter). The data from Schiavetta et al. (1990) were based on total s.c. adipocytes in the 9-10-11th rib section. The data were converted to total carcass s.c. adipocytes by estimating total carcass s.c. adipose tissue based on the 11-mo-old steers of Cianzio et al. (1985). [abc,jk,xy]Values for each study with the same superscripts were not different ($P > .05$).

observed between 2.5 wk and 5 mo of age in the current study suggests that hyperplasia also occurred during this period, yet SCD gene expression remained depressed. If true hyperplasia occurred in the preweaned calves, then the preadipocytes did not enter terminal differentiation at this time. This is supported by the low rate of de novo lipogenesis we observed in samples from the preweaned calves.

## Implications

We have provided evidence for the occurrence of preadipocyte terminal differentiation in tailhead subcutaneous adipose tissue of postweaning calves. This finding supports the growing body of evidence that postweaning development of bovine adipose tissue is characterized by periods of hyperplastic growth. If the processes of preadipocyte hyperplasia and(or) terminal differentiation are active in postweaning calves, then total adiposity of growing cattle may be especially sensitive to dietary or hormonal manipulations.

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