The Effects of *In Utero* Exposure of Lambs to a β-Adrenergic Agonist on Prenatal and Postnatal Muscle Growth, Carcass Cutability, and Meat Tenderness[1]

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ABSTRACT: The objectives of the present experiment were to examine the effects of *in utero* exposure to a β-adrenergic agonist ($L_{644,969}$) on prenatal and postnatal muscle growth and meat tenderness of lambs. Thirty twin-pregnant Composite IV (1/2 Finnsheep, 1/8 Dorset, 1/8 Rambouillet, 1/8 Targhee, 1/8 Suffolk) ewe lambs were used for this experiment. All ewes were fed an alfalfa hay-corn-based diet throughout gestation and lactation. From d 25 to 95 of gestation, the diet of one-half of the ewes contained 2 ppm of $L_{644,969}$ on an as-fed basis. Treatment did not ($P > .05$) affect lamb weights at any point in the growth cycle (birth to 43 kg). Heart weights of neonatal and market lambs were increased ($P < .05$) by *in utero* exposure to $L_{644,969}$. However, weights of lamb carcass components and weights of individual muscles were not affected by treatment ($P > .05$). Additionally, treatment did not alter the activities of any of the components of the calpain proteolytic system in neonatal or market lambs. Concomitantly, there was no effect of treatment on myofibril fragmentation index or Warner-Bratzler shear force. Moreover, there was no effect of treatment on muscle fiber type distributions, fiber sizes, or apparent fiber number. It seems that the lack of an effect of treatment on apparent fiber number would explain the lack of an effect on muscle weight. Thus, *in utero* exposure to $L_{644,969}$ does not seem to have promise as a method for improving lamb carcass cutability. Other methods of improving the rate and composition of lamb carcass growth while maintaining acceptable meat tenderness must be developed.

Key Words: β-Adrenergic Agonist, Carcasses, Meat Yield, Lamb, Tenderness

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Introduction

It is widely accepted that the meat industry must increase the efficiency of lean meat production. When fed during the latter stages of the finishing phase, β-adrenergic agonists (**BAA**) have been shown to increase carcass cutability and decrease meat tenderness in lamb (Hamby et al., 1986; Lee et al., 1988; Wang et al., 1988; Beermann et al., 1989; Kretchmar et al., 1990; Koohmaraie and Shackelford, 1991; Koohmaraie et al., 1991; Pringle et al., 1993). The increased muscle hypertrophy and decreased meat tenderness associated with BAA treatment has been attributed to increased calpastatin activity (Higgins et al., 1988; Kretchmar et al., 1989; Koohmaraie and Shackelford, 1991; Koohmaraie et al., 1991; Pringle et al., 1993). Heretofore, efforts to achieve BAA-induced muscle growth without concomitant decreases in meat tenderness have been unsuccessful. Withdrawal of these compounds before slaughter does eliminate the BAA-induced toughening, but the cutability effect is also lost (Gwartney et al., 1991).

Maltin et al. (1990) demonstrated that *in utero* exposure to a BAA decreased postnatal muscle growth in rats. Kim et al. (1994) reported that treatment of sows with a BAA during either of the first two-thirds of pregnancy resulted in increased longissimus area in the carcasses of the progeny. The mechanisms by which prenatal BAA treatment alters muscle growth have not yet been fully explored. Moreover, it has not been established whether prenatal BAA treatment has an effect on meat tenderness. Additionally, the effects of *in utero* exposure to a BAA have not been determined in a ruminant species. Thus, the objectives of the present experiment were to determine the effects of *in utero* exposure to a β-adrenergic agonist

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on prenatal and postnatal muscle hyperplasia and hypertrophy, growth rate, feed efficiency, carcass cutability, and meat tenderness.

Materials and Methods

Animals. The Roman L. Hruska U.S. Meat Animal Research Center Animal Care and Use Committee approved the use of animals in this study. Thirty twin-pregnant Composite IV (1/2 Finnsheep, 1/8 Dorset, 1/8 Rambouillet, 1/8 Targhee, 1/8 Suffolk) ewe lambs (7 mo of age) were selected for this experiment from a large group of lambs that were naturally mated following estrus synchronization with 3 mL of Lutalyse® (Upjohn, Kalamazoo, MI). Ewes that had conceived during the first 7 d of the breeding season and that had two functional corpus lutea (as determined by laparoscopy) on d 21 of gestation were included in this experiment. On d 21 of gestation, ewes were moved from an outdoor breeding facility, laparoscoped, and penned individually in 1.5-m^2 pens in a climate-controlled building (25 ± 2°C; 14 h of light/d). Ewes were fed an alfalfa hay-corn-based diet (Table 1) throughout the remainder of gestation and lactation. From d 25 to 95 of gestation, the diet of one-half of the ewes contained 2 ppm of $L_{644,969}$ (Merck, Sharp and Dohme, Rahway, NJ) on an as-fed basis. Feed consumption was determined daily and ewes were weighed weekly until d 130 of gestation. Ewes were shorn on d 40 of gestation and all subsequent weights were adjusted upward to account for the weight of the fleece. On d 89 of gestation, ewes were dewormed and vaccinated against clostridia with 15 mL of Tramisol (Pitman-Moore, Mundelein, IL) and 2 mL of Covexlin (Coopers Animal Health, Kansas City, KS), respectively.

One lamb, from each set of twins, was killed within 24 h of birth to assess the effects of *in utero* exposure to BAA on prenatal growth. The remaining lamb from each set of twins and all single lambs were reared conventionally. Lambs were tail-docked and males were castrated at birth. Lambs were weaned at 57 to 63 d of age. From weaning until slaughter, lambs were allowed ad libitum access to a standard growing-finishing diet (Table 1). Lamb weights were determined weekly from birth to slaughter and feed consumption was determined weekly throughout the growing-finishing phase. Lambs were slaughtered at a mean weight of 43 kg BW. To facilitate collection of postmortem data, lambs were slaughtered in two groups with 3 wk between. The oldest half of each treatment group was included in the first slaughter group.

At slaughter, the head, pelt, heart, lungs, liver, spleen, kidneys, kidney-pelvic fat, and viscera (gastrointestinal tract) were weighed. The viscera were not emptied before weighing.

The longissimus muscle was removed from the left side of each carcass within 20 min after death for determination of the activities of the components of the calpain proteolytic system. Samples were assayed for activity of μ- and m-calpain and calpastatin according to Koohmaraie (1990).

Following chilling (24 h at 2°C), carcass sides were dissected and individual weights of all the major muscles, combined weight of the minor muscles, and weights of fat and bone were recorded. The muscles that were weighed individually were the adductor, biceps femoris, infraspinatus, longissimus, psoas group (psoas major and psoas minor combined), quadriceps femoris (rectus femoris, vastus medialis, vastus intermedius, and vastus lateralis combined), semimembranosus, semitendinosus, supraspinatus, gluteus group (gluteus medius, gluteus profundus, and gluteus accessorius combined). Muscle nomenclature was based on Tucker et al. (1952).

Longissimus myofibril fragmentation index (Culler et al., 1978) and Warner-Bratzler shear force were determined after 1, 7, and 14 d postmortem for the market lambs. Due to the small size of the neonatal lamb carcasses, Warner-Bratzler shear force could not be determined and myofibril fragmentation index could only be determined twice (1 and 14 d postmortem).

Longissimus muscles were obtained at 24 h postmortem from the market lambs and several .7-cm^3 samples were frozen on cork in liquid nitrogen-cooled isopentane and stored at –70°C. Transverse cryostat sections, 10 μm thick, were cut and allowed to air dry.

Table 1. Ingredient and nutrient composition of ewe and lamb diets on an as-fed basis

Item	Ewe diet	Lamb diet
Ingredient		
Alfalfa hay, %	50.0	20.0
Corn, %	41.9	59.3
Soybean meal, %	3.0	15.0
Liquid molasses, %	2.5	3.0
Limestone, %	1.0	1.0
Ammonium chloride, %	.5	.5
Steambone meal, %	.5	.5
Sodium chloride, %	.5	.5
Vitamin ADE, ppm	500.0	500.0
Trace minerals, ppm	60.0	60.0
Rumensin 60, ppm	80.0	80.0
Aureomycin 50, ppm	500.0	500.0
Vitamin E, ppm	340.0	450.0
Nutrient		
DM, %	89.5	88.8
TDN, %	67.4	80.5
CP, %	14.4	17.5
Ca, %	1.0	.9
P, %	.4	.5
Cu, ppm	8.1	9.5
Monensin, ppm	11.0	11.0
Chlortetracycline, ppm	55.0	55.0

Sections were stained according to the procedures for simultaneous staining of bovine muscle fiber types described by Solomon and Dunn (1988). A minimum of 200 fibers per animal were classified as βR, αR, or αW according to the classification of Ashmore and Doerr (1971). Fiber areas were measured by Microcomp PM (Southern Micro Instruments, Atlanta, GA) interactive image analysis for planar morphometry.

Apparent fiber number was determined (Swatland, 1984) on psoas major and semitendinosus at two locations (3 cm from the origin or insertion). At each sample location the area of the muscle was measured and two samples were obtained and frozen as described above. Fiber density was determined on four contiguous fields from each location. Fiber density and muscle area data were used to calculate apparent fiber number.

Statistical Analysis. For all dependent variables associated with the ewes, one-way ANOVA were conducted to assess the effect of treatment (SAS, 1988). For all dependent variables associated with the lambs, sex was included in a factorially arranged randomized model (SAS, 1988).

Results and Discussion

Effects of β-*Adrenergic Agonist*

Growth rate and feed efficiency of the ewes were not affected by $L_{644,969}$ (Table 2). In contrast, growth rate and feed efficiency of feedlot lambs have been reported to increase 10 to 15% with $L_{644,969}$ (Convey et al., 1987; Koohmaraie et al., 1991; Pringle et al., 1993). β-adrenergic agonist treatment did not affect the percentage of ewes that had twin lambs, which is indicative of no effect of treatment on embryo or fetus survival. Moreover, lamb birth weights and total fetal weights (the sum of the birth weights of all the lambs born in a litter) were not altered by treatment.

In utero exposure to $L_{644,969}$ did not affect lamb weights at any point in the growth cycle (birth to 43 kg); consequently, slaughter weights did not differ between treatments (Table 3). Of all the traits measured in this experiment, only heart weight was affected by *in utero* exposure to $L_{644,969}$. Heart weights of neonatal and market lambs were increased (21 and 13%, respectively) by *in utero* exposure to $L_{644,969}$. Maltin et al. (1990) reported that *in utero* exposure to clenbuterol resulted in increased fetal heart weights in rats. Moreover, placental transfer of clenbuterol has been observed in human pregnancy (Pelkonen et al., 1982).

Whereas it has been reported that *in utero* exposure to a β-adrenergic agonist decreased hindlimb muscle weights in rats (Maltin et al., 1990) and increased longissimus area in pigs (Kim et al., 1994), *in utero* exposure to $L_{644,969}$ did not affect weights of lamb carcass components or individual muscles in the present experiment (Table 4).

Treatment did not alter the activities of any of the components of the calpain proteolytic system in neonatal or market lambs (Table 5). This was consistent with the lack of effect of treatment on myofibril fragmentation index or Warner-Bratzler shear force.

Kim et al. (1994) reported that *in utero* exposure to salbutamol resulted in an increased proportion of type I fibers in the deep portion of the semitendinosus of pigs. In contrast, for longissimus and psoas major, the distribution of muscle fiber types was not affected in

Table 2. Effect of β-adrenergic agonist treatment on growth and reproductive performance of ewes and birth weights of lambs

Trait[a]	Control	Treated	SEM
Initial wt, kg	47.2	47.2	1.1
Final wt, kg	63.9	62.1	2.0
Prepartum wt, kg	72.6	70.1	1.9
Average daily gain, kg			
$L_{644,969}$ trial[b]	.24	.21	.02
Withdrawal[c]	.25	.23	.03
Overall	.24	.22	.01
Feed efficiency, g of gain/kg of feed			
$L_{644,969}$ trial[b]	192.1	202.1	11.4
Withdrawal[c]	144.3	140.4	15.1
Overall	173.2	181.5	4.8
Twinning frequency, %	73.3	80.0	11.3
Birth wt, kg	3.2	3.2	.1
Fetal wt, kg[d]	5.5	5.7	.4

[a]None of the traits was affected by treatment ($P > .05$).
[b]Day 25 to 95 of gestation.
[c]Day 95 to 130 of gestation.
[d]Fetal weight is the sum of the birth weights of all the lambs born in a litter.

Table 3. Effect of *in utero* exposure to a β-adrenergic agonist on slaughter weight, feed efficiency, hot carcass weight, and weights of dress-off items

	Neonatal lambs			Market lambs		
Trait	Control	Treated	SEM	Control	Treated	SEM
Slaughter wt, kg	3.0	3.1	.2	42.9	42.9	1.3
Feed efficiency, g of gain/kg of feed	—	—	—	178.7	166.3	4.5
Spleen wt, g	4.2	4.2	.4	62.2	60.9	2.7
Kidney wt, g	17.0	18.8	.9	120.9	123.1	4.1
Heart wt, g	23.9[b]	29.0[a]	1.4	179.2[b]	202.9[a]	6.0
Lungs wt, g	59.4	64.1	4.0	402.0	407.4	20.6
Liver wt, g	58.6	63.4	3.3	743.0	738.5	29.7
Kidney-pelvic fat wt, g	20.5	24.4	1.6	976.0	1,219.3	97.5
Head wt, g	329.0	336.9	12.0	1,849.8	2,201.7	159.5
Pelt wt, g	407.0	446.4	25.7	5,208.9	5,085.8	151.6
Viscera wt, g	362.7	376.9	29.8	8,460.7	8,499.3	337.1
Hot carcass wt, kg	1.5	1.5	.1	21.8	22.1	.7

[a,b]Within a row and within an age group, means with different superscripts differ ($P < .05$).

Table 4. Effect of *in utero* exposure to a β-adrenergic agonist on weights of carcass components and dissected muscles

	Neonatal lambs			Market lambs		
Trait[a]	Control	Treated	SEM	Control	Treated	SEM
Lean wt, g	345.5	359.0	25.2	5,409.3	5,540.2	198.2
Fat wt, g	14.1	21.6	2.5	3,087.2	3,124.5	210.5
Bone wt, g	316.1	335.7	19.4	1,835.8	2,029.4	82.0
Adductor wt, g	7.2	8.3	.5	138.4	127.1	4.8
Biceps femoris wt, g	16.9	17.8	1.2	302.6	308.5	11.1
Gluteus group wt, g	11.6	12.0	1.0	226.5	237.8	8.7
Infraspinatus wt, g	8.6	9.4	.8	132.7	148.9	7.3
Longissimus dorsi wt, g	24.9	27.2	1.8	564.1	580.1	21.1
Psoas group wt, g	9.3	10.1	.6	123.9	127.2	5.1
Quadriceps femoris wt, g	28.6	29.9	2.0	408.3	406.6	11.8
Semimembranosus wt, g	14.6	15.7	1.2	285.4	287.3	10.0
Semitendinosus wt, g	5.5	6.2	.4	111.9	109.7	3.5
Supraspinatus wt, g	9.2	9.9	.6	113.5	113.0	3.8

[a]None of the traits was affected by treatment ($P > .05$).

Table 5. Effect of *in utero* exposure to a β-adrenergic agonist on the calpain proteolytic system, myofibril fragmentation index, and Warner-Bratzler shear force

	Neonatal lambs			Market lambs		
Trait[a]	Control	Treated	SEM	Control	Treated	SEM
Calpastatin activity, units/g	4.8	5.5	.5	2.3	2.1	.3
μ-calpain activity, units/g	1.5	1.3	.1	.9	.8	.1
m-calpain activity, units/g	2.2	2.2	.1	1.1	1.1	.0
Myofibril fragmentation index						
1 d postmortem	37.5	35.2	2.0	58.3	53.9	1.8
7 d postmortem	—	—	—	80.7	79.4	2.0
14 d postmortem	77.0	73.1	2.5	84.3	83.3	2.0
Warner-Bratzler shear force, kg						
1 d postmortem	—	—	—	8.6	8.9	.5
7 d postmortem	—	—	—	5.1	5.1	.5
14 d postmortem	—	—	—	3.2	3.5	.3

[a]None of the traits was affected by treatment ($P > .05$).

Table 6. Effect of *in utero* exposure to a β-adrenergic agonist on muscle fiber sizes and distributions and apparent fiber number of market lambs[a]

Muscle	Fiber type	Control	Treated	SEM
		Fiber area, μm^2		
Longissimus	β-red	1,114	1,019	86
	α-red	1,241	1,195	97
	α-white	1,543	1,507	85
	Overall	1,378	1,316	81
Psoas major	β-red	800	855	38
	α-red	572	583	32
	α-white	909	868	43
	Overall	770	759	30
		Fiber type distribution, %		
Longissimus	β-red	15	17	2
	α-red	31	30	3
	α-white	54	53	3
Psoas major	β-red	23	19	1
	α-red	34	36	1
	α-white	43	44	1
		Apparent fiber no.		
Psoas major		327,765	338,952	29,705
Semitendinosus		640,694	632,561	27,806

[a]None of the traits was affected by treatment ($P > .05$).

the present experiment (Table 6). Moreover, treatment did not affect fiber areas for longissimus and psoas major or apparent fiber number of psoas major and semitendinosus. Maltin et al. (1990) observed that total fiber number and muscle weight were reduced for rats exposed to clenbuterol during pregnancy. It would seem that the effect (or lack thereof) of *in utero* exposure to a β-adrenergic agonist is mediated through its effect (or lack thereof) on fiber number. It is not apparent why *in utero* exposure to $L_{644,969}$ did not affect fiber number in sheep.

Numerous differences existed between our experiment and previous investigations of the effects of *in utero* exposure to a β-adrenergic agonist that might have contributed to the conflicting results among these studies. These differences included species, drug, dosage, and timing and duration of treatment. That heart weight of neonatal and market-weight lambs was altered by treatment serves as circumstantial evidence that $L_{644,969}$ crossed the placenta and affected lamb development. However, it cannot be ruled out that higher concentrations of $L_{644,969}$ would have resulted in alterations in lamb muscle development. Moreover, alteration of the timing of treatment might have resulted in alterations in lamb muscle development. However, that there was not even a tendency for treatment to increase muscle growth rates suggests that alterations in the protocol would not result in a significant effect in lambs.

Table 7. Effect of sex on slaughter weight, feed efficiency, hot carcass weight, and weights of dress-off items

	Neonatal lambs			Market lambs		
Trait	Ewe	Ram	SEM	Ewe	Wether	SEM
Slaughter wt, kg	2.8	3.3	.2	41.6	44.1	1.3
Feed efficiency, g of gain/kg of feed	—	—	—	.2	.2	.0
Spleen wt, g	4.1	4.3	.4	59.5	63.6	2.7
Kidney wt, g	17.1	18.6	.9	121.5	122.5	4.1
Heart wt, g	24.0[b]	29.2[a]	1.4	179.7[b]	202.4[a]	5.9
Lungs wt, g	62.3	61.5	4.0	378.9	430.5	20.3
Liver wt, g	59.1	62.9	3.3	726.0	755.4	29.1
Kidney-pelvic fat wt, g	23.6	21.3	1.7	1,092.9	1,102.3	95.7
Head wt, g	311.6	356.6	12.2	1,743.9[b]	2,307.7[a]	156.5
Pelt wt, g	393.4	465.4	25.8	5,011.8	5,282.9	148.8
Viscera wt, g	336.8	401.6	30.5	8,261.1	8,698.8	330.8
Hot carcass wt, kg	1.4	1.6	.1	20.8[b]	22.9[a]	676.7

[a,b]Within a row and within an age group, means with different superscripts differ ($P < .05$).

Table 8. Effect of sex on weights of carcass components and dissected muscles

	Neonatal lambs			Market lambs		
Trait[a]	Ewe	Ram	SEM	Ewe	Wether	SEM
Lean wt, g	327.7	382.4	24.7	9,926.6[b]	11,099.7[a]	365.1
Fat wt, g	13.0[b]	22.8[a]	2.5	2,994.2	3,217.5	206.6
Bone wt, g	305.0	351.3	18.7	1,721.2[b]	2,144.0[a]	80.5
Adductor wt, g	7.0	8.5	.5	124.2[b]	141.3[a]	4.7
Biceps femoris wt, g	15.8	19.1	1.2	281.9[b]	329.2[a]	10.9
Gluteus group wt, g	10.5[b]	13.4[a]	.9	221.8	242.4	8.5
Infraspinatus wt, g	8.3	9.8	.8	135.4	146.2	7.1
Longissimus dorsi wt, g	23.7	28.7	1.7	528.1[b]	616.0[a]	20.7
Psoas group wt, g	8.9[b]	10.6[a]	.6	111.9[b]	139.2[a]	5.0
Quadriceps femoris wt, g	27.4	31.7	1.9	381.4[b]	433.4[a]	11.5
Semimembranosus wt, g	13.9	16.7	1.1	267.1[b]	305.5[a]	9.8
Semitendinosus wt, g	5.2[b]	6.6[a]	.4	101.5[b]	120.1[a]	3.5
Supraspinatus wt, g	9.0	10.3	.6	106.6[b]	119.9[a]	3.7

[a,b]Within a row and within an age group, means with different superscripts differ ($P < .05$).

Effects of Sex

Sex did not affect slaughter weights of neonatal or market lambs; however, at market weight, wethers had higher head and hot carcass weights than ewes (Table 7). Among neonatal and market lambs, males had higher heart weights than did females (Table 7).

At market weight, wethers had higher weights of lean and bone than ewes (Table 8); however, the relative proportion of each carcass component was not affected by sex (data not presented in tabular form). Whereas ewes have been reported to be fatter than wethers (Oliver et al., 1967), ewes tended to have a lower percentage of the carcass composed of fat than wethers in the present experiment. Dissected muscle weight differences between sexes paralleled the hot carcass weight differences between sexes. Among neonatal lambs, hot carcass weights were numerically higher for rams than for ewes ($P = .12$). Concomitantly, all muscle weights were numerically higher for rams, the gluteus group, psoas group, and semitendinosus were significantly heavier. Among market lambs, hot carcass weights and weights of 8 of the 10 muscles were higher ($P < .05$) for rams than for ewes.

Sex did not affect ($P > .05$) any of the components of the calpain system, myofibril fragmentation index, or Warner-Bratzler shear force (Table 9). However, Warner-Bratzler shear force at d 14 postmortem tended ($P = .06$) to be lower for wethers than for ewes, possibly due to a trend ($P = .11$) toward reduced calpastatin activity for wethers compared to ewes. Oliver et al. (1967) and Summers et al. (1978) reported that longissimus shear force did not differ between ewes and wethers.

Sex did not affect muscle fiber areas for longissimus or psoas major (Table 10). Moreover, the distribution of fiber types was similar among sexes. However, apparent fiber number of semitendinosus was significantly higher ($P < .05$) for wethers than for ewes and apparent fiber number of psoas major was numerically

Table 9. Effect of sex on the calpain proteolytic system, myofibril fragmentation index, and Warner-Bratzler shear force

	Neonatal lambs			Market lambs		
Trait[a]	Ewe	Ram	SEM	Ewe	Wether	SEM
Calpastatin activity, units/g	4.9	5.3	.5	2.5	1.9	.3
μ-calpain activity, units/g	1.4	1.4	.1	.9	.8	.1
m-calpain activity, units/g	2.2	2.2	.1	1.1	1.2	.03
Myofibril fragmentation index						
1 d postmortem	37.7	36.1	1.6	58.3	53.9	1.7
7 d postmortem	—	—	—	78.0	82.1	1.9
14 d postmortem	75.5	74.4	2.5	82.6	85.0	2.0
Warner-Bratzler shear force, kg						
1 d postmortem	—	—	—	9.0	8.4	.5
7 d postmortem	—	—	—	5.6	4.6	.5
14 d postmortem	—	—	—	3.7	2.9	.3

[a]None of the traits was affected by gender ($P > .05$).

Table 10. Effect of sex on muscle fiber sizes and distributions and apparent fiber number of market lambs[a]

Muscle	Fiber type	Ewe	Wether	SEM
		Fiber area, μm^2		
Longissimus	β-red	1,027	1,106	84
	α-red	1,236	1,200	95
	α-white	1,420	1,631	83
	Overall	1,298	1,397	79
Psoas major	β-red	848	807	38
	α-red	584	571	32
	α-white	889	889	42
	Overall	776	753	29
		Fiber type distribution, %		
Longissimus	β-red	14	18	2
	α-red	29	32	3
	α-white	57	50	2
Psoas major	β-red	21	21	1
	α-red	34	36	1
	α-white	45	43	1
		Apparent fiber no.		
Psoas major		299,575	367,142	29,149
Semitendinosus		585,798[b]	687,457[a]	27,286

[a,b]Within a row, means with different superscripts differ ($P < .05$).

higher ($P = .12$) for wethers. For the psoas major, fiber number (r = .57; $P = .001$) was more highly related to muscle weight than was fiber area (r = .15; $P = .46$). Because muscle mass differed to a greater extent between males and females than did fiber diameter, Joubert (1956) speculated that muscle fiber number was greater for males than females.

In conclusion, *in utero* exposure of lambs to $L_{644,969}$, a β-adrenergic agonist that has been shown to markedly improve carcass composition in market lambs via muscle hypertrophy, did not affect prenatal or postnatal muscle growth. Although altering the timing, duration, and level of $L_{644,969}$ treatment might result in an effect on muscle growth, results of this study did not give any indication that *in utero* exposure to a β-adrenergic agonist has any promise as a method for improving the composition of lamb carcasses. Increased fiber number rather than increased fiber size may be responsible for increased muscle mass of males compared to that of females.

Implications

In utero exposure of lambs to a β-adrenergic agonist did not affect growth. Thus, other methods of improving the rate and composition of lamb carcass growth while maintaining acceptable meat tenderness must be developed.

Literature Cited

Ashmore, C. R., and L. Doerr. 1971. Comparative aspects of muscle fiber types in different species. Exp. Neurol. 31:408.

Beermann, D. H., S. Y. Wang, G. Armbuster, H. W. Dickson, E. L. Rickes, and J. G. Larson. 1989. Influences of beta-agonist L-655,871 and electrical stimulation on post-mortem muscle metabolism and tenderness in lambs. Proc. Recip. Meat Conf. 42: 54.

Convey, E. M., E. Rickes, Y. T. Yang, M. A. McElligott, and G. Olson. 1987. Effects of the beta-adrenergic agonist L-644,969 on growth performance, carcass merit and meat quality. Proc. Recip. Meat Conf. 40:47.

Culler, R. D., F. C. Parrish, Jr., G. C. Smith, and H. R. Cross. 1978. Relationship of myofibril fragmentation index to certain chemical, physical, and sensory characteristics of bovine longissimus muscle. J. Food Sci. 43:1177.

Gwartney, B. L., C. R. Calkins, and S. J. Jones. 1991. The effect of cimaterol and its withdrawal on carcass composition and meat tenderness of broiler chickens. J. Anim. Sci. 69:1551.

Hamby, P. L., J. R. Stouffer, and S. B. Smith. 1986. Muscle metabolism and real-time ultrasound measurement of muscle and subcutaneous adipose tissue growth in lambs fed diets containing a beta-agonist. J. Anim. Sci. 63:1410.

Higgins, J. A., Y. V. Lasslett, and P. J. Buttery. 1988. The relation between dietary restriction or clenbuterol (a selective β2 agonist) treatment on muscle growth and calpain proteinase (EC3.4.22.17) and calpastatin activities in lambs. Br. J. Nutr. 60:645.

Joubert, D. M. 1956. An analysis of factors influencing post-natal growth and development of the muscle fiber. J. Agric. Sci. 47: 59.

Kim, Y. S., R. D. Sainz, J. Ferlazzo, and N. M. Tulloh. 1994. Effect of maternal administration of salbutamol to sows on post-natal growth and carcass characteristics in the progeny. Aust. J. Agric. Res. 45:271.

Koohmaraie, M. 1990. Quantification of Ca^{2+}-dependent protease activities by hydrophobic and ion-exchange chromatography. J. Anim. Sci. 68:659.

Koohmaraie, M., and S. D. Shackelford. 1991. Effect of calcium chloride infusion on the tenderness of lambs fed a β-adrenergic agonist. J. Anim. Sci. 69:2463.

Koohmaraie, M., S. D. Shackelford, N. E. Muggli-Cockett, and R. T. Stone. 1991. Effect of the β-adrenergic agonist $L_{644,969}$ on muscle growth, endogenous proteinase activities, and postmortem proteolysis in wether lambs. J. Anim. Sci. 69:4823.

Kretchmar, D. H., M. R. Hathaway, and W. R. Dayton. 1989. In vivo effect of a β-adrenergic agonist on activity of calcium-dependent proteinases, their specific inhibitor, and cathepsins B and H in skeletal muscle. Arch. Biochem. Biophys. 275:228.

Kretchmar, D. H., M. R. Hathaway, R. J. Epley, and W. R. Dayton. 1990. Alterations in postmortem degradation of myofibrillar proteins in muscle of lambs fed a β-adrenergic agonist. J. Anim. Sci. 68:1760.

Lee, Y. B., H. Jung, Y. S. Kim, and R. H. Dalrymple. 1988. Effect of cimaterol (CL 263,780) on meat quality in lambs. J. Anim. Sci. 68(Suppl. 1):279 (Abstr.).

Maltin, C. A., M. I. Delday, and S. M. Hay. 1990. The effect of clenbuterol administration in utero and throughout lactation on pre- and post-natal muscle development in the rat. Growth Dev. Aging 54:143.

Oliver, W. M., Z. L. Carpenter, G. T. King, and J. M. Shelton. 1967. Qualitative and quantitative characteristics of ram, wether and ewe lamb carcasses. J. Anim. Sci. 26:307.

Pelkonen, O., R. Tuimala, and A. Kauppila. 1982. Placental transfer of clenbuterol early in pregnancy. Eur. J. Clin. Pharmacol. 22: 403.

Pringle, T. D., C. R. Calkins, M. Koohmaraie, and S. J. Jones. 1993. Effects over time of feeding a β-adrenergic agonist to wether lambs on animal performance, muscle growth, endogenous muscle proteinase activities, and meat tenderness. J. Anim. Sci. 71:636.

SAS. 1988. SAS/STAT® User's Guide. (Release 6.03). SAS Inst., Inc., Cary, NC.

Solomon, M. B., and M. C. Dunn. 1988. Simultaneous histochemical determination of three fiber types in single sections of ovine, bovine and porcine skeletal muscle. J. Anim. Sci. 66:255.

Summers, R. L., J. D. Kemp, D. G. Ely, and J. D. Fox. 1978. Effects of weaning, feeding systems and sex of lamb on lamb carcass characteristics and palatability. J. Anim. Sci. 47:622.

Swatland, H. J. 1984. Structure and Development of Meat Animals. Prentice-Hall, Englewood Cliffs, NJ.

Tucker, H. Q., M. M. Voegli, and G. H. Wellington. 1952. A cross sectional muscle nomenclature of the beef carcass. Michigan State College Press, East Lansing.

Wang, S.-Y., R. M. O'Connor, and D. H. Beermann. 1988. Effect of cimaterol on proteolytic activity and growth in lamb skeletal muscle. FASEB J. 2:A847.