Effects of β-Adrenergic Agonists on Cellular Metabolism

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Introduction

Presentations at previous Reciprocal Meat Conferences (Ricks et al., 1984; Beermann et al., 1985) and the presentations immediately preceding mine have described in detail the influence of β-adrenergic agonists, such as clenbuterol, cimaterol or ractopamine, on growth and carcass traits in livestock. We now can say with some certainty that the feeding of β-adrenergic agents to livestock increases the rate of growth of muscle and decreases fat accretion. However, we are considerably more uncertain about the cellular mechanism of action of these compounds. The pronounced and highly reproducible results (at least in ruminant species) elicited by β-adrenergic agents provide animal scientists with a powerful tool for investigating the regulation of muscle and adipose tissue growth and development.

It is my intent to provide a summary of the research that has been performed with swine, sheep and cattle that sheds some light on the cellular mechanisms involved in the modification of muscle and adipose tissue growth in animals exposed to β-adrenergic agents. Most of the research is quite recent, and I will depend heavily on material published at present only in abstract form. Although I will reference some of the work accomplished with laboratory species, I will focus primarily on livestock species because, as you will see, the effects we observe appear to be highly species-specific.

Overview

There are several cellular mechanisms by which β-adrenergic agonists could increase the rate of muscle growth and depress adipose tissue growth; these were summarized in detail by Ricks et al. (1984). Briefly, the growth of any tissue is the result of the balance between synthesis and degradation. In muscle, net growth implies that myofibrillar protein synthesis occurs at a rate greater than that of myofibrillar protein degradation. Adipose tissue growth primarily involves the net accretion of lipid, i.e., the balance between triacylglycerol biosynthesis and turnover (lipolysis). However, even the process of lipid accretion can be regulated by protein synthesis or degradation, because the enzymes that catalyze the processes of lipid metabolism can change in amount in response to hormonal and nutritional stimuli.

Table 1 lists those cellular effects of β-adrenergic agents that have been well characterized in laboratory species. All of these metabolic events are mediated by a cAMP, the concentration of which rises in response to the activation of adenylate cyclase upon interaction of specific catecholamines with their receptors. It is likely that, as in rats, β_2-adrenergic agents stimulate glycogen degradation and lipolysis in tissues of livestock species. Obviously, it is difficult to conceive how the increased degradation of glycogen elicited by β_2-adrenergic agents could result in an increase in muscle mass, and indeed it is unlikely that glycogen degradation is responsible for the muscle growth response caused by these compounds. However, an elevated rate of lipolysis certainly should result in a reduction of adipocyte volume, hence adipose tissue mass. Similarly, increasing muscle mass could involve either increased myofibrillar protein synthesis or a depression of the rate of myofibrillar protein turnover. The following will focus on metabolic changes that have been documented for livestock species.

Swine

Adipose tissue

The growth and carcass responses of swine to the feeding of β-adrenergic agents (Dalrymple et al., 1984; Jones et al, 1985; Moser et al., 1986) typically are less than those elicited in sheep (Baker et al., 1984; Thornton et al., 1985; Beermann et al., 1986; Hamby et al., 1986) or cattle (Ricks et al., 1984; Miller et al., 1986; Smith et al., 1987). In a recent study, Mersmann and coworkers were unable to elicit any growth response in young pigs fed cimaterol (Mersmann et al., 1987).

The variable response of swine to the feeding of β-adrenergic agents may be the function of the high specificity of porcine adipose tissue catecholamine receptors. Lipolysis in swine adipose tissue is stimulated readily by many β-adrenergic agonists, most notably isoproterenol, which is a pure β-adrenergic agonist. Unlike most species, swine adipose tissue exhibits extreme agonist structural specificity for the stimulation of lipolysis; thus, their adrenergic receptor cannot be classified into a β_1- or β_2-subtype (Mersmann, 1984). Mersmann (1987) demonstrated that, as anticipated, the infusion of clenbuterol into pigs resulted in an elevation of serum nonesterified fatty acids (NEFA) (Figure 1). However, this was not due to a direct stimulation of lipolysis by clenbuterol, since it has not been possible to demonstrate a

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Table 1. Metabolic Effects of β_2-Adrenergic Agonists.

Event/enzyme affected	*Acute effect*	*Long-term effect(s)*
Glycogen degradation		
Glycogen phosphorylase	Stimulation	Depletion of glycogen stores
Glycogen synthesis		
Glycogen synthetase	Inhibition	Depletion of glycogen stores
Fatty acid biosynthesis		
Acetyl-CoA carboxylase	Inhibition	Reduction of lipid stores/decrease in adipose tissue mass
Triacylglycerol biosynthesis		
Glycerophosphate acyltransferase	Inhibition	Reduction of lipid stores/decrease in adipose tissue mass
Phosphatidate phosphohydrolase	Inhibition	
Lipolysis		
Hormone-sensitive lipase	Stimulation	Reduction of lipid stores/decrease in adipose tissue mass
Protein synthesis		
Transcription*	Stimulation	Increase in protein amount
Translation*	Stimulation	
Protein degradation		
Cathepsins**	Inhibition	Increase in protein amount
Calcium-activated proteases**	Inhibition	

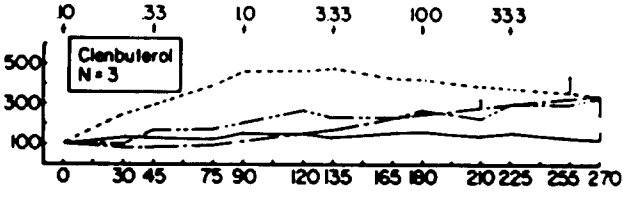
*Specifically affected enzymes have not been identified. Additionally, cAMP has been shown to decrease the transcription of specific genes in liver.
**Only indirect evidence exists to implicate these proteases in protein degradation.

stimulation of lipolysis above basal rates in porcine adipose tissue slices incubated with clenbuterol in vitro (Mersmann et al., 1987; Figure 2). Additionally, Mersmann et al. (1987) demonstrated that plasma fatty acids were elevated only slightly (from 979 eq/l to 1,199 eq/l) in pigs fed .50 mg cimaterol·kg^{-1} diet, and even then only in those pigs fed low (14%) protein levels. Others (Merkel et al., 1987) reported significantly elevated glycerol in serum from pigs fed the phenethanolamine β-adrenergic agonist, ractopamine. It is likely that, while lipolysis may play a role in the effects of β-adrenergic agents on swine carcass development, depending on the agonist used, it may not be through a direct effect of the compound on lipolysis in porcine adipose tissue.

The effects of β-adrenergic agonists on adipose tissue accretion also could be mediated through modifications in the rate of de novo lipid biosynthesis. The activities of several enzymes of the fatty acid and triacylglycerol biosynthetic pathways are inhibited as a result of exposure of adipose tissue to catecholamines (Table 1). A recent study (Merkel et al., 1987) demonstrated that the feeding of ractopamine to pigs resulted in depressed lipogenesis and reduced lipogenic enzyme activities in vitro in subcutaneous adipose tissue. However, as with lipolysis, lipogenesis is not affected directly by clenbuterol in porcine adipose tissue slices incubated in vitro (Rule et al., 1987). Thus, observed reductions in lipogenesis in vitro may have been due to less energy being available for fatty acid biosynthesis. This, in turn, would likely have been the result of increased rates of myofibrillar protein synthesis.

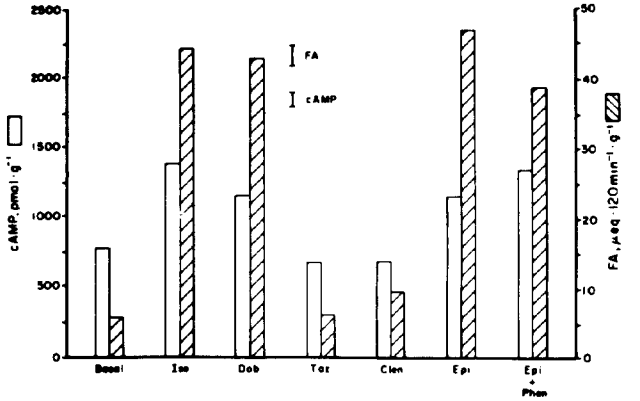
Recent research has indicated that the feeding of β-adrenergic agonists to mice reduced the sensitivity of the adipose tissue to insulin (Mills and Orcutt, 1987). This might provide another indirect mechanism by which β-adrenergic agonists depress fat accretion in swine.

Figure 1



Plasma nonesterified fatty acid concentrations in response to the infusion of clenbuterol in pigs. From Mersmann (1987).

Figure 2



Lipolysis in vitro in porcine subcutaneous adipose tissue exposed to a variety of β-adrenergic agonists. From Hu et al. (1987).

Muscle

The accretion of muscle mass involves a balance between the processes of protein synthesis and protein degradation, or turnover. In this discussion, protein synthesis includes transcription of the genes encoding myofibrillar proteins as well as translation of the RNA transcripts. Degradation is assumed to involve the proteolytic activities of both the cathepsins and the calcium-activated proteases.

The feeding of β-adrenergic agonists to laboratory species consistently increases the rate of muscle accretion, even in hypophysectomized rats (Thiel et al., 1987). As a means of elucidating the cellular mechanisms by which these compounds influence muscle hypertrophy, several studies of muscle development and metabolism have been conducted with muscle cell cultures. However, these often have produced conflicting results. Forsberg et al. (1986) reported that cimaterol had no effect on protein synthesis, but reduced protein degradation, in rat myoblasts (Table 2). Cimaterol had no effect on either process in mouse myoblasts (Forsberg et al., 1986; Table 2). Similarly, cimaterol had no effect on protein synthesis or degradation in cultures of L6 myoblasts (Roeder et al., 1987). It should be noted that studies of this nature measure total protein metabolism, and are not designed to investigate the synthesis or turnover of specific myofibrillar proteins. Because contractile proteins comprise only approximately fifty percent of the proteins in muscle, investigations of their specific metabolism may yield results that differ from investigations of total protein metabolism.

Work in swine in vivo has provided somewhat more insight into the mechanism of action of β-adrenergic agents in increasing muscle growth. Feeding trials with pigs have demonstrated that cimaterol elicited small, but significant, increases in *longissimus* muscle cross-sectional area (Moser et al., 1986) although, as mentioned previously, some investigators have been unable to demonstrate any effects of cimaterol on carcass traits (Mersmann et al., 1987). Another β-adrenergic agent, ractopamine, appears to be more effective in stimulating muscle accretion in swine (Anderson et al., 1987; Bergen et al., 1987). In addition to increasing muscle mass, ractopamine increases nitrogen retention (Anderson et al., 1987) and appears to both enhance protein synthesis and lower protein degradation as measured by indirect methods in vivo (Bergen et al., 1987; Table 3). Considering the extreme agonist specificity demonstrated by porcine adipose tissue, it would not be surprising if muscle receptor specificities accounted for the differences between studies done with pigs and those performed with rat or mouse cell culture systems.

Table 2. Effects of β-Adrenergic Agonists on Protein Synthesis and Degradation in Cell Culture Systems.

Cell culture system	*Effect*	
	Synthesis	*Degradation*
Rat myoblasts[a]	No effect	Decrease
Mouse myoblasts[a]	No effect	No effect
L6 myoblasts[b]	No effect	No effect

[a]From Forsberg et al. (1986).
[b]From Roeder et al. (1987).

Table 3. Effects of Ractopamine on Protein Synthesis and Degradation in Swine[a].

Parameter	*Effect*
Semitendinosus muscle protein content	14% increase
Fractional protein synthesis rate	10% increase
Protein synthesis in vitro	No effect
Protein degradation in vitro	No effect

[a]From Bergen et al. (1987).

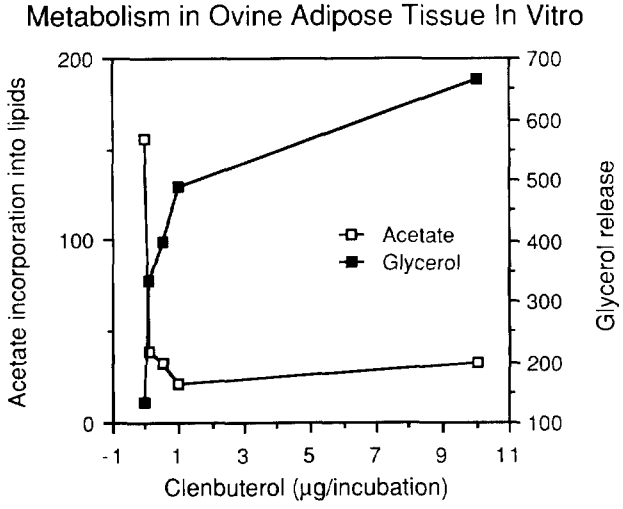
Sheep

Adipose tissue

Sheep (or growing lambs) exhibit perhaps the most pronounced response to the feeding of β-adrenergic agents. Marked reductions in subcutaneous fat thickness and corresponding decreases in carcass fatness have been elicited readily by cimaterol or clenbuterol (Baker et al., 1984; Thornton et al., 1985; Beermann et al., 1986; Hamby et al., 1986). As would be predicted from the β_2-adrenergic chemical nature of these compounds, the feeding of cimaterol to lambs significantly increased plasma NEFA concentrations, even after 12 weeks of treatment (Beermann et al., 1985). Unlike results observed for swine adipose tissue, clenbuterol stimulated lipolysis in ovine adipocytes incubated in vitro (Thornton et al., 1985; Peterla et al., 1987; Figure 3). Ovine adipose tissue also differs from swine adipose tissue in that lipogenesis in vitro was inhibited markedly by acute exposure to a β-adrenergic agonist (Thornton et al., 1985; Figure 3).

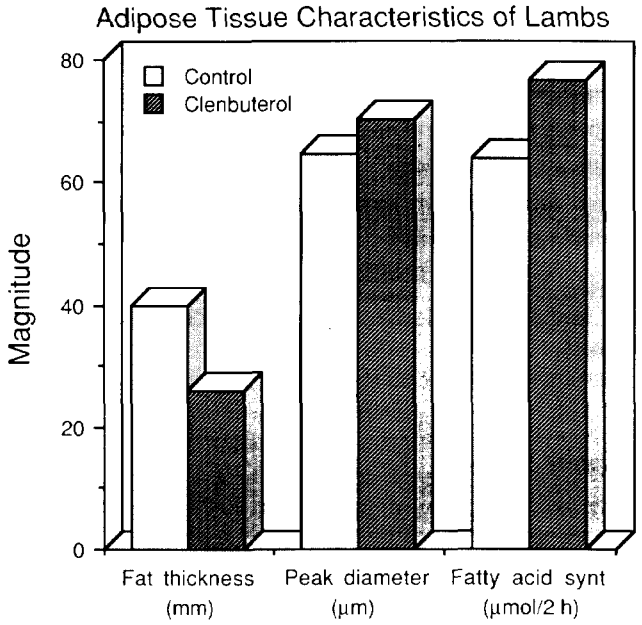
Coleman et al. (1985) and Hu et al. (1987) demonstrated that, in spite of marked decreases in subcutaneous fat thickness in clenbuterol-fed lambs, lipogenesis in subcutaneous adipose tissue from the treated lambs was unaffected, or

Figure 3



Lipolysis and lipogenesis in ovine adipose tissue incubated in vitro with clenbuterol. From Thornton et al. (1985).

Figure 4



Subcutaneous fat thickness, adipocyte volume and lipogenesis in vitro in adipose tissue from untreated lambs and lambs fed clenbuterol. From Coleman et al. (1985) and Hamby et al. (1986).

even increased, by dietary clenbuterol (Figure 4). These findings would rule out chronically depressed lipogenesis as the basis for the effects of β-adrenergic agonists on ovine adipose tissue growth.

A paradox reported by Coleman et al. (1985) was that, in spite of a 35% reduction in subcutaneous fat thickness in clenbuterol-fed lambs, relative to untreated lambs (Hamby et al., 1986), there was actually a numerical increase in adipocyte volume (Figure 4). This corresponded to the increase in lipogenesis observed in the adipose tissue from the clenbuterol-fed lambs (Coleman et al., 1985; Figure 4). One mechanism by which adipose tissue mass could be decreased in the face of increased adipocyte volume is through a reduction in total adipocyte number, either by blocking preadipocyte hyperplasia or by inhibiting recruitment (lipid filling) of previously divided adipocytes. Correspondingly, Jones et al. (1987) recently reported that ractopamine reduced proliferation of primary cultures of rat adipocytes by approximately 50% (Table 4). Conversely, ractopamine increased differentiation (lipid filling) of adipocytes, which would corroborate the elevated lipogenesis observed in adipose tissue from lambs fed clenbuterol (Coleman et al., 1985) or cimaterol (Hu et al., 1987). Thus, in growing lambs, β-adrenergic agents may have a two-fold effect on adipose tissue: a retardation of adipocyte hypertrophy through a direct stimulation of lipolysis; and a depression of adipose tissue accretion through a reduction in preadipocyte proliferation. Obviously, this assumes that hyperplasia is occurring at this stage of growth in lambs, an assumption which awaits substantiation.

Table 4. Effect of Ractopamine on Preadipocyte Proliferation and Differentiation in Primary Cell Culture[a].

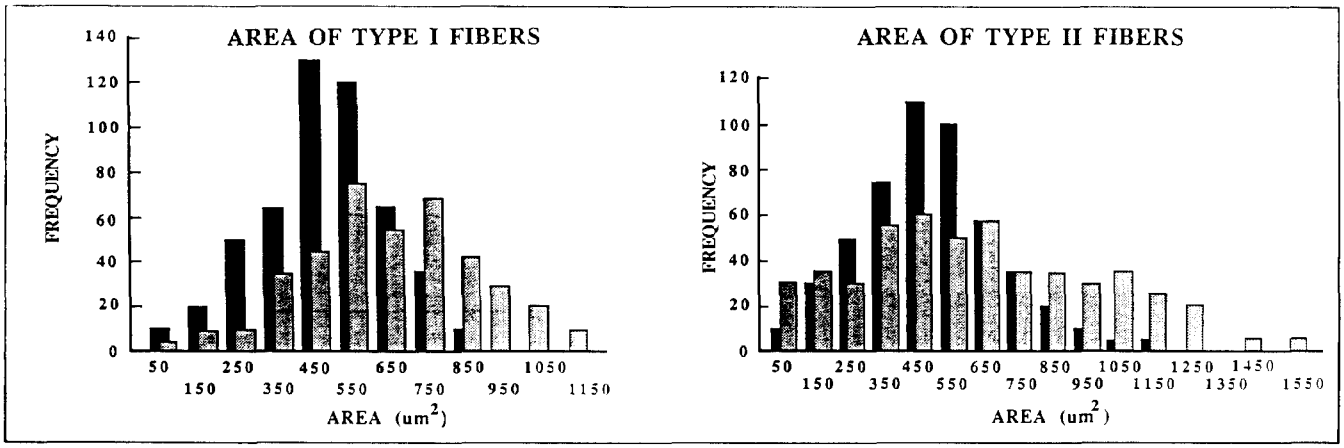
Parameter	*Effect*
Preadipocyte proliferation	58% reduction
Preadipocyte differentiation (lipid filling)	100% increase

[a]From Jones et al. (1987).

Muscle

Because substantial postnatal increases in muscle fiber number have not been reported for livestock species, it is likely that the increases in muscle mass elicited by β-adrenergic agonists are the result of myofiber hypertrophy. Correspondingly, Beermann and coworkers (Beermann et al., 1985) reported 30% increases in the cross-sectional areas of both Type I and Type II myofibers from *semitendinosus* muscle in lambs fed cimaterol (Figure 5). Hamby et al. (1986) reported that longissimus muscle strips obtained from clenbuterol-fed lambs exhibited greater rates of glycogen synthesis and overall glucose utilization in vitro than muscle from untreated lambs, which suggested larger Type II (glycolytic) myofibers in the longissimus muscle of the treated lambs. Decreased myofibrillar protein turnover may have been the basis for the muscle hypertrophy in lambs, since

Figure 5



Myofiber distributions in semitendinosus muscle from untreated lambs (solid bars) and lambs fed cimaterol. From Beermann et al. (1985).

Table 5. Cathepsin-B Activity in Liver and Muscle in Control and Cimaterol-Fed Sheep[a].

Tissue	Treatment Group	
	Control	Cimaterol
	nmol/mg wet wt/hr	
Liver	118 ± 6	139 ± 17
Muscle	53 ± 7	29 ± 5

[a]From Forsberg et al. (1987).

Forsberg et al. (1987) reported that dietary cimaterol reduced cathepsin B activity in ovine *semitendinosus* muscle (Table 5).

Cattle

Adipose tissue

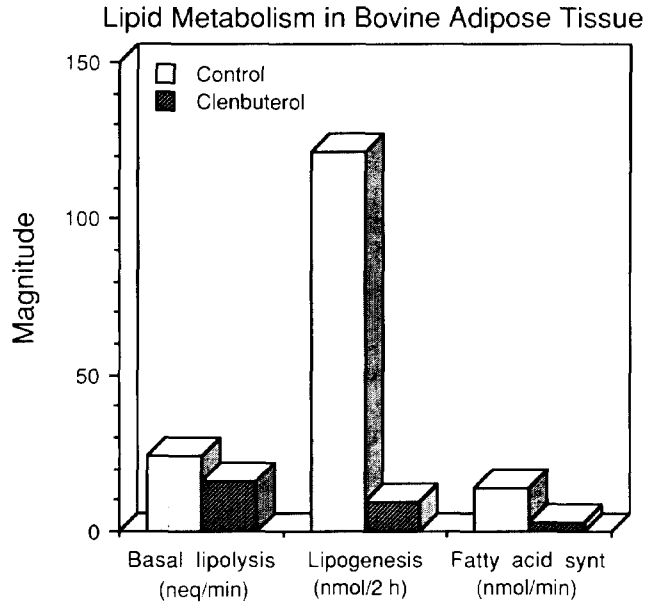
The feeding of clenbuterol to feedlot steers (Ricks et al., 1984) or heifers (Miller et al., 1986) clearly depresses adipose tissue accretion. However, substantially less information concerning the role of lipolysis in the reduction of carcass fat elicited by β-adrenergic agents exists for cattle than for the other livestock species. Miller et al. (1986) documented a significant reduction in the basal rate of lipolysis in subcutaneous adipose tissue from clenbuterol-fed heifers (Figure 6). However, lipogenesis in vitro and lipogenic enzyme activities also were depressed markedly by clenbuterol feeding (Miller et al., 1986; Figure 6), so the lower basal lipolytic rate may have been reflective of an overall lower metabolic rate of the adipose tissue from the clenbuterol-treated heifers. No other information is available that would directly implicate lipolysis as the basis for the effects of β-adrenergic agents on adipose tissue metabolism in cattle, and major metabolic differences between ovine and bovine adipose tissue metabolism (Smith and Prior, 1986) preclude extending results observed for growing lambs to those observed in cattle.

The investigation of the effects of clenbuterol on growth in heifers (Miller et al., 1986; Smith et al., 1987) demonstrated that, unlike results observed for growing lambs, clenbuterol-feeding to heifers decreased subcutaneous adipocyte volume and subcutaneous fat thickness proportionally (Figure 7). Hence, it is likely that β-adrenergic agents depress adipose tissue growth in cattle, at least in part through a reduction in de novo fatty acid biosynthesis. This is in marked contrast to results observed in growing lambs.

The addition of clenbuterol to incubations of subcutaneous and intramuscular adipose tissue from untreated, market weight steers resulted in a small, nonsignificant depression in lipogenesis in subcutaneous adipose tissue, and had no effect whatsoever in intramuscular adipose tissue (Figure 8). Similarly, our laboratory (Miller et al., 1986) also reported that epinephrine had no effect on lipogenesis in bovine subcutaneous or intramuscular adipose tissue. Thus, lipogenesis in bovine adipose tissue is, at best, only marginally sensitive to inhibition by β-adrenergic agonists, providing further evidence for the marked differences in metabolism between ovine and bovine adipose tissues (Smith and Prior, 1986).

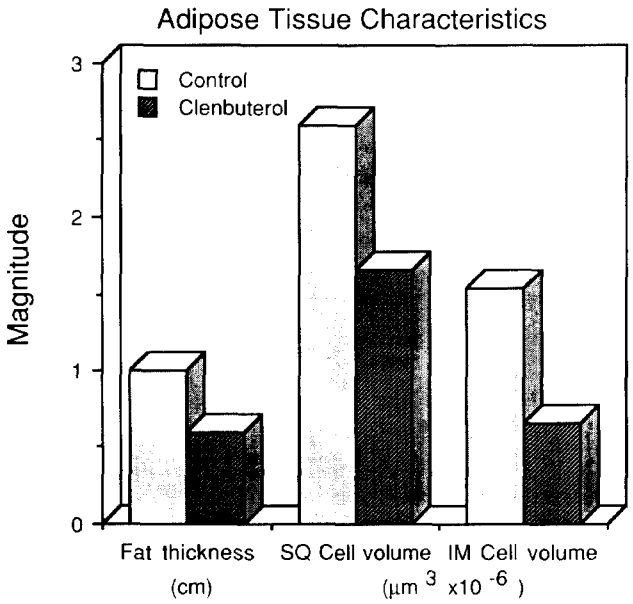
Because a stimulation of lipolysis in livestock fed β-adrenergic agonists would result in more energy being available for other metabolic processes, it could be argued that the elevated NEFA concentration in treated animals is the driving force for stimulating muscle protein synthesis. However, recent results from this laboratory have indicated that marked increases in *longissimus* muscle mass (25%) can be accomplished without a concomitant reduction in adipose

Figure 6



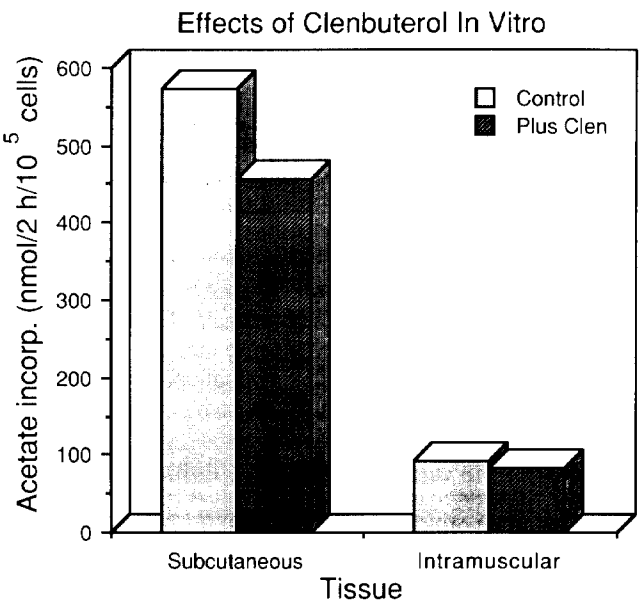
Basal lipolysis and lipogenesis in vitro in adipose tissue from untreated heifers and heifers fed clenbuterol. From Miller et al. (1986).

Figure 7



Subcutaneous fat thickness and adipocyte volume in untreated and clenbuterol-treated growing heifers. From Smith et al. (1987).

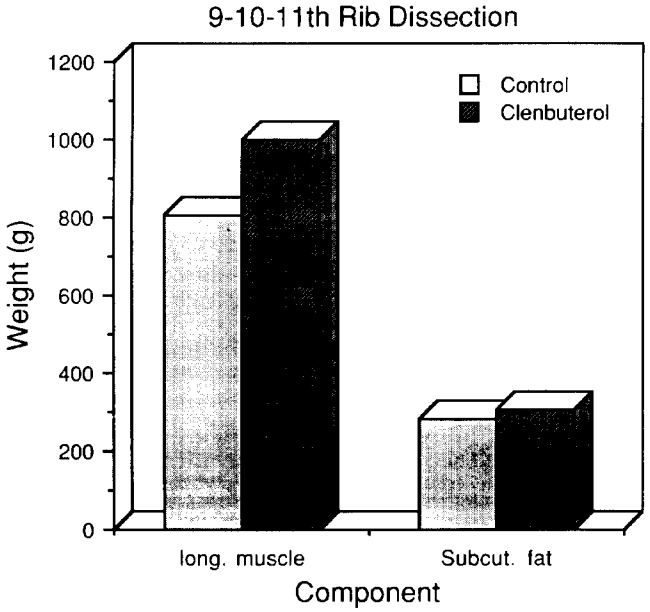
Figure 8



Lipogenesis in bovine subcutaneous (SQ) and intramuscular (IM) adipose tissue incubated with clenbuterol in vitro.

tissue mass (Figure 9). Thus, the provision of energy through the turnover of lipids is not prerequisite for fueling the additional protein synthesis occurring in cattle treated with clenbuterol. Eisemann and Huntington (1987) demonstrated that the feeding of clenbuterol to steers increased blood flow and oxygen uptake in the hindquarters of steers, so that increased nutrient supply as a result of the feeding of β-adrenergic agents may yet prove to be the driving force for stimulating muscle growth in cattle.

Figure 9



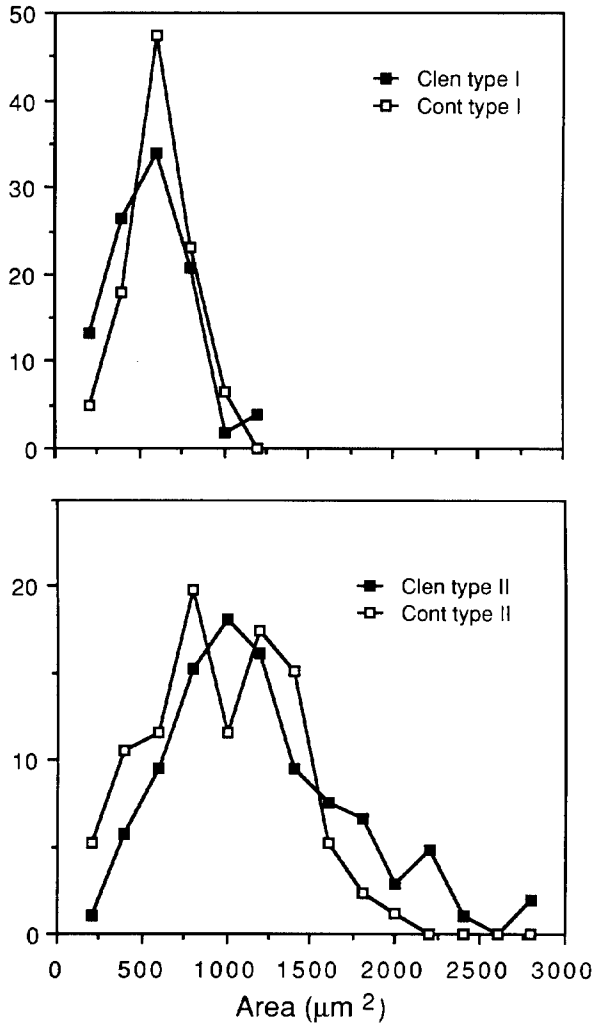
Rib dissections (9-10-11th rib) for control and clenbuterol-fed steers.

Muscle

In growing, finishing heifers, clenbuterol feeding resulted in an increase in the diameters of Type II myofibers, but had no effect on Type I myofibers in *longissimus* muscle (Wu et al., 1986; Figure 10). Because the increase in fiber diameter can more than account for the increase in *longissimus* muscle cross-sectional area in these animals (approximately 20%; Wu et al., 1986), it is likely that the greater rate of *longissimus* muscle growth in clenbuterol-treated heifers was due to increased hypertrophy of the muscle.

As described previously, increased nutrient supply to muscle in cattle fed β-adrenergic agonists may be the basis for the stimulation of muscle growth observed in these animals. Alternatively, β-adrenergic agents may increase muscle mass through a direct stimulation of myofibrillar protein synthesis, or through a decrease in protein turnover. Eisemann and coworkers reported a greater rate of α-amino nitrogen uptake in the hindlimbs of steers fed clenbuterol (Eisemann and Huntington, 1987), suggesting a greater rate of protein synthesis in those animals.

Figure 10

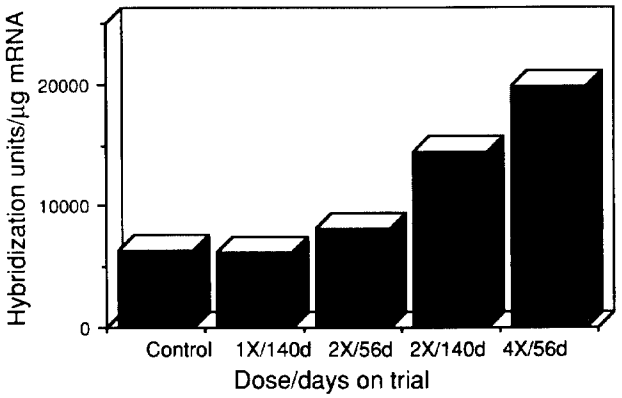


Longissimus muscle myofiber diameter distributions in control and clenbuterol-fed heifers. From Wu et al. (1986).

Precedence for the direct stimulation of gene transcription by hormones that increase cAMP was provided by Hod et al. (1984). They demonstrated that the mRNA corresponding to a gluconeogenic enzyme (phosphoenolpyruvate carboxykinase) was increased in hepatocytes incubated with cAMP. Increased mRNA levels presumably would lead to increased transcription of the mRNA, hence a greater rate of protein synthesis. Evidence that this is occurring in bovine *longissimus* muscle was provided by this laboratory (Smith et al., 1987; Figures 11 & 12). The feeding of ractopamine to steers increased the amounts of mRNA corresponding to myosin light chain-1 and myosin light chain-3 (Figure 11). Both proteins, which are integral components of thick filaments in striated muscle, are encoded by the same gene.

Figure 11

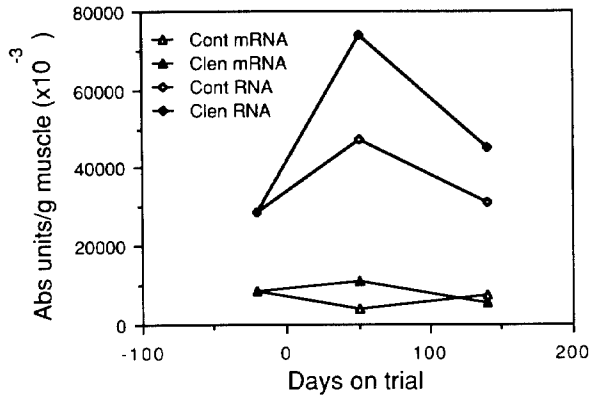
Myosin light chain-1/3 gene expression



Expression of the myosin light chain-1/3 gene in longissimus muscle of steers fed ractopamine at various doses or for various durations.

Figure 12

MLC-3 GENE EXPRESSION IN GROWING ANGUS STEERS



Expression of the myosin light chain-1/3 gene in longissimus muscle of growing steers. Clenbuterol was introduced into the diet of the treated steers at day 0 and removed from the diet at day 50.

More recent research has demonstrated that clenbuterol consumption by growing steers increased the myosin light chain mRNA, and this effect is lost when clenbuterol is removed from the diet (Garcia et al., 1987; Figure 12). Thus, there is some evidence in cattle to suggest that one of the mechanisms by which β-adrenergic agonists stimulate muscle growth is through an increase in protein synthesis. It still remains to be demonstrated that these compounds exert their effects through a direct interaction with the muscle catecholamine receptors.

Summary

Table 6 summarizes the effects of β-adrenergic agonists on muscle and adipose tissue metabolism in livestock species. Certainly, many of the effects appear to be species-specific, and also may be dependent upon the age of the animals. As suggested by this review, the use of β-adrenergic agents in our research provides us with a fascinating new and powerful tool for investigations of those factors that regulate growth and development in livestock.

Table 6. Summary of In Vivo and In Vitro Effects of β-Adrenergic Agonists on Muscle and Adipose Tissue Metabolism in Livestock Species.

Species/system/metabolic process	*Effect*
Swine/whole animal	
Lipolysis	Stimulated
Lipid synthesis	Inhibited or no effect
Protein synthesis	Small stimulation
Protein degradation	No available data
Incubations in vitro	
Lipolysis	No effect
Lipid synthesis	No effect
Protein synthesis/degradation	No effect
Sheep/whole animal	
Lipolysis	Stimulated
Lipid synthesis	Stimulated or no effect
Protein synthesis	No available data
Protein degradation	Inhibited
Incubations in vitro	
Lipolysis	Strongly stimulated
Lipid synthesis	Strongly inhibited
Protein synthesis/degradation	No available data
Cattle/whole animal	
Lipolysis	Inhibited (basal rate)
Lipid synthesis	Inhibited or no effect
Protein synthesis	Stimulated
Protein degradation	No available data
Incubations in vitro	
Lipolysis	No available data
Lipogenesis	Small inhibition
Protein synthesis/degradation	No available data

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