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Zilpaterol-HCl Reduces Urinary Excretion of N-tau-methylhistidine by Finishing Steers¹

D.W. Brake and E.C. Titgemeyer

Introduction

Zilpaterol-HCl is an orally active β -adrenergic agonist that repartitions nutrient use in cattle and has been approved for use during the final 20 to 40 days of the finishing period. Zilpaterol administration to finishing cattle increases average daily gain, feed efficiency, hot carcass weight, ribeye area, and dressing percentage; however, zilpaterol decreases meat tenderness, which is detectable by sensory panelists. Attenuation of zilpaterol's effect on tenderness would improve its benefits to cattle producers.

Decreases in tenderness of meat from cattle fed zilpaterol may be closely related to decreases in protein degradation in skeletal muscles. Urinary excretion of N-tau-methylhistidine (NMH) in cattle reflects skeletal muscle protein degradation *in vivo* and provides a convenient research measure of muscle protein degradation. We analyzed NMH excretion by cattle fed zilpaterol to estimate the breakdown rate of skeletal-muscle protein.

Materials and Methods

Twelve steers of British breeding were used in two sets of six and placed into two replicates of similarly designed trials conducted at different times. Steers were blocked in each set based on pretrial voluntary feed intake and treatments of zilpaterol were randomly assigned within each block. In each replicate, three steers were administered zilpaterol (60 mg/day) provided as Zilmax (Intervet Schering-Plough Animal Health, Millsboro, DE) throughout the trial, and three steers received no zilpaterol. Zilpaterol treatment was administered in a randomized block design.

Within each group of six steers, the three steers receiving the same zilpaterol treatment were used in concurrent 3×3 Latin squares and administered dietary protein treatments. Dietary protein treatments were three corn-based diets: control (10.2% crude protein), urea (13.3% crude protein), or dried distillers grains with solubles (DDGS; 14.9% crude protein). Treatments delivered DDGS (20% of dry matter) and urea (1% of dry matter) at inclusion rates similar to those used commonly in corn-based diets fed to finishing cattle. DDGS was selected as a supplemental protein source because of its relatively high content of undegradable intake protein. Urea was selected as a supplemental nitrogen source because it is completely ruminally degraded.

Steers were housed in metabolism crates to facilitate total collection of urine and feces. Steers were fed twice daily in equal amounts. Total urinary excretion of NMH and creatinine were analyzed to estimate skeletal-muscle protein degradation. Urinary creatinine excretion was used to estimate total skeletal muscle mass.

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Results and Discussion

We measured urinary NMH excretion to estimate *in vivo* skeletal muscle protein degradation and urinary creatinine excretion as a measure of total skeletal muscle protein mass. Thus, the ratio of urinary NMH excretion to urinary creatinine excretion was directly related to the proportion of skeletal muscle protein that was degraded.

Total daily excretion of urinary NMH was not affected by zilpaterol ($P=0.70$; Table 1). Creatinine excretion was numerically increased by zilpaterol administration. These differences together led to an NMH:creatinine ratio that was less ($P<0.01$) for steers receiving zilpaterol than for those not receiving zilpaterol (Figure 1). Zilpaterol reduced skeletal-muscle protein degradation *in vivo* when expressed as a proportion of total skeletal-muscle protein mass.

In this trial, steers fed zilpaterol unexpectedly had greater ($P<0.01$) feed intakes than control steers. Generally, skeletal muscle protein turnover (both protein synthesis and protein degradation) is increased as feed intake increases; however, zilpaterol reduced skeletal muscle protein turnover in spite of greater feed intakes that should have increased skeletal muscle protein turnover.

We observed no effect of dietary protein or its interaction with zilpaterol ($P>0.5$) on urinary excretion of NMH or creatinine or on the ratio between the two. We interpreted this to suggest that dietary protein type had no effect on protein degradation or that our model was not sensitive enough to detect differences.

Implications

Zilpaterol administration to cattle receiving corn-based diets reduced skeletal-muscle protein degradation. This might explain, in part, reductions in meat tenderness for cattle fed zilpaterol.

Table 1. Effect of zilpaterol-HCl (Zilmax) on N-tau-methylhistidine and creatinine excretion in steers consuming corn-based diets supplemented with no protein (control), with dried distillers grains with solubles (DDGS), or with urea

Item	Zilpaterol			No zilpaterol			SEM	P-value		
	Control	DDGS	Urea	Control	DDGS	Urea		Zilpaterol	Diet	Interaction
Number of steers	4	5	4	6	5	5				
N-tau-methylhistidine, mmol/day	1.20	1.20	1.23	1.24	1.26	1.28	0.11	0.71	0.87	0.99
Creatinine, g/day	15.4	15.4	14.8	13.1	13.6	13.8	1.1	0.20	0.89	0.52
N-tau-methylhistidine:creatinine, mmol:g	75.9	77.5	81.4	94.4	92.9	92.5	5.4	<0.01	0.92	0.76

Figure1. Effect of zilpaterol on the ratio of urinary excretion of N-tau-methylhistidine (NMH) to creatinine in steers consuming corn-based diets supplemented with no protein (control), with dried distillers grains with solubles (DDGS), or with urea.