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RUMINAL AMMONIA LOAD DOES NOT AFFECT HISTIDINE UTILIZATION IN GROWING STEERS

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Summary

Fermentation of dietary protein in the rumen leads to ammonia absorption, which could impair amino acid utilization in cattle. Our study was conducted to determine the effects of rumen ammonia load on histidine utilization. Six ruminally cannulated Holstein steers (318 lb) housed in metabolism crates were used in a 6 × 6 Latin square design. Treatments were arranged as a 3 × 2 factorial and included: 0, 1.5, or 3 grams/day L-histidine infused abomasally; and 0 or 80 grams/day urea infused ruminally to supply a metabolic ammonia load. As expected, urea infusions increased rumen ammonia and plasma urea concentrations. No change in nitrogen retention, a measure of lean tissue growth, occurred in response to urea. Retained nitrogen increased with histidine supply, and the maximal response occurred with 1.5 grams/day of histidine, suggesting that this amount was near the supplemental requirement. Our research revealed that increases in ammonia load did not demonstrate a metabolic cost in terms of whole body protein deposition, regardless of whether histidine was limiting. Thus, although an excess protein supply may not be economically efficient or environmentally friendly, it does not appear to directly penalize animal performance.

Introduction

Intestinal supply of amino acids, the building blocks of protein, must meet animal requirements in order to optimize lean muscle growth. Restriction of a single dietary essential amino acid may limit growth. Previous research indicated that with our experimental model we can create a histidine deficiency in calves that is useful for studying factors that affect the efficiency of amino acid use.

In ruminants, ammonia is produced within the rumen as a result of fermentation of dietary protein. This ammonia subsequently is absorbed through the rumen wall and transported to the liver where it is detoxified. There is some evidence suggesting that ammonia detoxification may contribute to the inefficient use of dietary amino acids. This is because ammonia is detoxified to urea in the liver, and amino acids can be consumed metabolically during the process of urea synthesis. However, other research suggests that amino acid use for the synthesis of urea from ammonia is not quantitatively important. Our study was conducted to determine if an increased ammonia load has a negative impact on protein deposition when histidine is the most limiting amino acid.

Experimental Procedures

Six ruminally cannulated Holstein steers averaging 318 lb were used in a 6 × 6 Latin square to determine effects of rumen ammonia load on utilization of histidine. Steers were housed in metabolism crates and fed 5.5 lb/day (dry matter basis) of a basal diet containing 83% soybean hulls, 7.6% wheat straw, 4% molasses, 5% minerals/vitamins, and 0.4% urea.

To insure that histidine was the first limiting amino acid for lean tissue deposition, all steers received abomasal infusions that con-

tained 250 grams/day amino acids, which supplied adequate amounts of all essential amino acids except histidine, and 300 grams/day glucose. Vitamin B-6, folic acid, and vitamin B-12 also were supplemented to all steers to ensure that they were not limiting. All steers also received ruminal infusions that contained 180 grams/day acetate, 180 grams/day propionate, and 45 grams/day butyrate to supply energy without increasing microbial protein supply. Treatments were continuously infused, arranged as a 3 × 2 factorial, and included: 0, 1.5, or 3 grams/day L-histidine infused abomasally; and 0 or 80 grams/day urea infused ruminally to supply a metabolic ammonia load. The two infusions were continuously supplied by a peristaltic pump through tubing that passed through the rumen cannula with one line terminating in the rumen and one in the abomasum.

Experimental periods were 6 days, with 2 days for adaptation to treatment and 4 days for total fecal and urinary collection to allow measurement of nitrogen retention, a measure of lean tissue deposition. Rumen fluid was collected 2, 4, and 6 hours after feeding to determine rumen ammonia concentration. Blood samples were collected from the jugular vein 5 hours after feeding on day 6 of each period for plasma urea analysis.

Results and Discussion

Nitrogen balance data are presented in Table 1. Retained nitrogen increased linearly ($P<0.01$) with histidine supplementation, indicating that the control steers were histidine deficient. Nitrogen retention leveled off between 1.5 grams/day and 3 grams/day supplemental histidine, indicating that 1.5 grams/day was near the steers' requirements for growth.

Urea infusions increased ($P<0.01$) rumen ammonia from 8.6 to 19.7 m*M*. Increases in rumen ammonia concentration show that the non-protein nitrogen was hydrolyzed in the rumen to ammonia. Plasma urea concentrations increased from 2.7 to 5.1 m*M* when urea was infused, also indicating that an increased ammonia load was achieved through the urea treatment.

Due to the nitrogen infused as urea, total nitrogen intake increased for steers receiving 80 grams/day urea. Fecal nitrogen was similar among all treatments, which suggests that the extra nitrogen infused as urea was absorbed. No change in nitrogen retention occurred in response to urea, nor was there a histidine by urea interaction for nitrogen retention. As a result, our study indicates that an increased ammonia load did not change how efficiently the calves used histidine for growth. Increased ruminal ammonia concentrations did not negatively impact animal performance. Despite the lack of effect on growth, feeding of diets that yield a large ammonia load may not be economically efficient or environmentally friendly.

The maximal response to histidine occurred with 1.5 grams/day supplementation, suggesting that this amount was near the requirement. By using the difference between nitrogen retention for 0 and 1.5 grams/day supplementation, efficiency of histidine deposition in lean tissue can be calculated. Grams of nitrogen retained can be converted to grams of crude protein retained per day and from this we can calculate the amount of histidine retained (2.5 grams of histidine/100 grams of crude protein). In our study, steers deposited an additional 0.98 grams of histidine for each 1.5 grams that were supplemented, which equates to an efficiency of deposition of supplemental histidine of 65%, a value near that used by several models for prediction of cattle performance.

In our experiment with growing steers, increases in the ammonia load did not demonstrate a metabolic cost in terms of whole body protein deposition, regardless of whether histidine was limiting.

Table 1. Effect of Supplemental Histidine and Urea on Nitrogen Balance of Growing Steers

	No Urea			80 grams/day Urea			
Nitrogen, grams/day	No His	1.5 His[1]	3 His[1]	No His	1.5 His[1]	3 His[1]	SEM
Total intake	87.8	89.4	91.3	124.7	127.4	127.6	
Fecal	15.0	15.5	15.3	15.0	15.4	15.4	0.8
Urinary[ab]	41.7	37.9	37.8	77.6	72.4	72.2	2.2
Retained[a]	31.1	35.9	38.2	31.8	39.6	39.8	1.9

[1]1.5 His = 1.5 grams/day histidine; 3 His = 3 grams/day histidine.

[a]Linear effect of histidine level ($P<0.01$).

[b]Effect of urea level ($P<0.01$).