Effects of Abomasal Infusion of Arginine-, Threonine-, or Histidine-Deficient Amino Acid Mixtures on Mammary Amino Acid Metabolism in Lactating Goats (Postprint)

Authors: Hu Zhiyong, Sun Defeng, Li Jiwei, Yan Zhengui, Lin Xueyan, Wang Zhonghua

Date: 2017-10-10T00:00:00+00:00

Abstract

The present study was conducted to investigate the effects of abomasal infusion of mixed amino acids deficient in arginine (Arg), threonine (Thr), or histidine (His) on amino acid metabolism in the mammary gland of lactating goats. Four Saanen dairy goats in mid-lactation were surgically fitted with abomasal cannulas (for amino acid infusion), carotid artery and mammary vein catheters (for blood sample collection), and a blood flow probe in the external pudendal artery (to record mammary blood flow). The experimental goats were limit-fed a basal diet to meet maintenance energy and protein requirements. Glucose and mixed amino acids formulated according to the amino acid composition of rumen microbial protein were infused abomasally. A 4×4 Latin square design was adopted, with the control group receiving complete mixed amino acids and the experimental groups receiving mixed amino acids deficient in Arg, Thr, or His, respectively. The experiment consisted of 4 periods, each lasting 7 days, with the first 4 days as the infusion period and the last 3 days as the sampling period. The results showed that: 1) Single amino acid deficiency infusion had no significant effect on milk yield or milk protein yield ($P>0.05$). 2) Arg and Thr deficiency significantly increased mammary blood flow (P 0.05) and decreased arterial and venous concentrations of these amino acids, with mammary clearance rates increasing by 25.9% and 199%, respectively. Single amino acid deficiency infusion had no significant effect on mammary uptake or the ratio of mammary uptake to output ($P>0.05$). 3) Arg, Thr, and His deficiency increased lactation conversion efficiency by 25.4% (4.02 vs. 5.04, P 0.05), 34.5% (9.09 vs. 12.23, P 0.05), and 14.6% (10.51 vs. 12.04, $P>0.05$), respectively.

Keywords: Amino acids; Mammary blood flow; Lactation performance; Lactation conversion efficiency

Journal: Acta Zoonutrimenta Sinica Submission status: Published in journal

Citation: ChinaXiv:201711.00730 (or this version ChinaXiv:201711.00730V1) DOI:10.12074/201711.00730V1 CSTR:32003.36.ChinaXiv.201711.00730.V1 Sci-Tech Blockchain TXID: 3c33d46d-3309-45f1-9a8d-f13790a0ce7b

Recommended citation format: Hu Zhiyong, Sun Defeng, Li Jiwei, Yan Zhengui, Lin Xueyan, Wang Zhonghua. Effects of abomasal infusion of mixed amino acids deficient in arginine, threonine, or histidine on amino acid metabolism in the mammary gland of lactating goats. Acta Zoonutrimenta Sinica: https://chinaxiv.org/abs/201711.00730. [ChinaXiv:201711.00730V1]

Full Text

Effects of Abomasal Infusion of Amino Acid Mixture without Arginine, Threonine or Histidine on Mammary Amino Acid Metabolism in Lactating Goats

HU Zhiyong, SUN Defeng, LI Jiwei, YAN Zhengui, LIN Xueyan, *WANG Zhonghua*

(College of Animal Science and Technology, Shandong Agricultural University, Tai’ an 271018, China)

Abstract: This study aimed to investigate the effects of abomasal infusion of amino acid (AA) mixture lacking arginine (Arg), threonine (Thr), or histidine (His) on mammary amino acid metabolism in lactating goats. Four Saanen dairy goats in mid-lactation were fitted with abomasal cannulas (for AA infusion), carotid artery and mammary vein catheters (for blood sampling), and blood flowmeter probes in the external pudendal artery (to record mammary blood flow). The experimental goats were restrictively fed a basal diet to meet maintenance energy and protein requirements. Abomasal infusion of glucose and an AA mixture formulated according to rumen microbial protein amino acid composition was administered. A 4×4 Latin square design was employed, with the control group receiving complete AA mixture and the experimental groups receiving AA mixtures deficient in Arg, Thr, or His. The trial comprised four periods, each lasting 7 days (4 days of infusion followed by 3 days of sampling). The results showed: (1) Infusion of AA mixture lacking a single AA had no significant effect on milk yield or milk protein yield ($P>0.05$). (2) Arg and Thr deficiency significantly increased mammary blood flow (P 0.05), reduced arterial and venous concentrations of the deficient AA, and increased mammary clearance rates by 25.9% and 199%, respectively. Single AA deficiency had no significant effect on mammary AA uptake or the absorption-to-output ratio ($P>0.05$). (3) Arg, Thr, and His deficiency increased lactation conversion efficiency by 25.4% (4.02 vs. 5.04, P 0.05), 34.5% (9.09 vs. 12.23, P 0.05), and 14.6% (10.51 vs. 12.04, $0.05<P$ 0.10), respectively. These findings suggest

that lactating goats can increase dietary AA supply to the mammary gland for the deficient AA by elevating mammary blood flow and clearance rate, thereby improving the lactation conversion efficiency of the deficient AA.

Keywords: amino acid; mammary blood flow; lactation performance; lactation conversion efficiency

Factors influencing the lactation conversion efficiency of essential amino acids (EAA) include milk yield, dietary AA supply, and metabolic characteristics of the digestive tract and liver. Milk yield affects the partitioning of AA between maintenance and lactation, while dietary AA supply influences post-hepatic EAA availability and thus lactation conversion efficiency.

Measurements of intestinal absorption and portal vein flux of AA in dairy cows reveal substantial variation in AA uptake by portal-drained viscera (PDV). Histidine shows minimal loss, whereas leucine (Leu), threonine (Thr), and some non-essential amino acids (NEAA) exhibit substantial losses. Bequette et al. observed Leu and methionine (Met) oxidation in the PDV of sheep. The low portal recovery of Thr may be related to pancreatic oxidation. Therefore, PDV metabolism of EAA directly affects both the quantity and profile of AA entering the circulation.

Liver removal of AA also varies considerably, consequently influencing post-hepatic EAA supply. Cant et al. classified EAA into two groups based on hepatic removal characteristics: Group 1 EAA with low hepatic clearance, including branched-chain AA (BCAA) and lysine (Lys); and Group 2 EAA, including His, phenylalanine (Phe), and Met, which are extensively removed by the liver with degradation enzymes primarily localized in the liver. The net removal rates of His, Phe, and Met were 0.36, 0.38, and 0.49 times their portal absorption, respectively. Given the high hepatic blood flow, EAA entering the liver originate mainly from arterial blood (artery supplying PDV plus hepatic artery), and net hepatic EAA uptake primarily derives from peripheral tissue recycling, catabolizing AA not utilized by peripheral tissues. Thus, AA metabolic characteristics of the digestive tract and liver affect EAA lactation conversion efficiency.

Mabjeesh et al. demonstrated that when EAA supply decreases, the mammary gland can adjust blood flow velocity and AA transport capacity to meet lactation requirements. Therefore, within a certain range, reduced EAA supply can increase the lactation conversion efficiency of the deficient AA. This study investigated the magnitude of increased lactation conversion efficiency through single EAA deficiency infusion to reflect AA metabolic characteristics in the digestive tract and liver, ultimately providing a reference for rational composition of intestinally digestible AA.

1.1 Experimental Animals and Diet

Four healthy multiparous Saanen dairy goats in mid-lactation [milk yield (1.5±0.25) kg/d, body weight (50±5) kg] were selected. One month before the trial, goats underwent surgery for abomasal cannula installation (for AA infusion), carotid artery exteriorization and fixation with vascular catheters (for blood sampling), direct mammary vein catheter installation, and ultrasonic blood flow probe placement on the left external pudendal artery (MC6PSS-LS-WCS10-GC, Transonic Systems Inc., USA) to record mammary blood flow. Goats were individually housed in metabolic cages and fed complete pelleted diet every 2 h via automatic feeders to ensure ad libitum intake: Goat No. 1: 70.5 g/meal, No. 2: 73.9 g/meal, No. 3: 66.1 g/meal, No. 4: 68.1 g/meal.

The basal diet was formulated according to the UK AFRC (1993) standards for dairy goats and pelleted. During the trial, goats were restrictively fed the pelleted basal diet to meet maintenance energy and protein requirements. Diet composition and nutrient levels are shown in Table 1.

1.2 Experimental Treatments

A 4×4 Latin square design was used, with the control group (C) receiving complete AA mixture and three experimental groups receiving AA mixtures deficient in Arg (-Arg), Thr (-Thr), or His (-His). The trial consisted of 4 periods, each 7 days long (4 days of infusion followed by 3 days of sampling).

Based on milk yield recorded during the 3 days preceding each period, metabolizable energy (ME) and metabolizable protein (MP) requirements for lactation were calculated according to AFRC (1993). AA infusion amounts were calculated based on lactation requirements (Table 2). Abomasal infusion of glucose and AA mixture formulated according to rumen microbial protein AA composition provided energy and protein for lactation. The complete AA mixture composition is shown in Table 3.

AA infusion solutions and glucose solutions were prepared with physiological saline, pH adjusted to approximately 7.4 with HCl and NaOH solutions. The prepared infusion mixture was placed in a beaker on a constant temperature magnetic stirrer, mixed uniformly, and then continuously infused into the abomasum for 24 h. During the sampling period, indwelling vascular needles were installed in the carotid artery and mammary vein for blood collection, milk samples were collected, and blood flow was measured.

1.3 Sample Collection and Analysis

During each sampling period, milk samples were collected at 08:00 and 18:00, with a few drops of hydrogen peroxide added and stored at -20°C. Before analysis, milk samples were thawed in a 37°C water bath. Lactose, milk fat, and milk protein yields and percentages were determined using an infrared milk composition analyzer (78110, Foss, USA). Milk protein hydrolyzed AA concentrations were measured by liquid chromatography-mass spectrometry (UPLC-MSMS, AB Sciex, USA).

Prior to blood collection, 20 mL syringes and intravenous extension lines connected to indwelling needles were soaked in 1:500 heparinized saline. Continuous slow blood withdrawal was performed using a self-made blood collection device composed of indwelling needles and syringes connected to a syringe pump (W0109-1, Baoding Langer Constant Flow Pump Co., Ltd., Hebei) at 10 mL/h. Five mL was placed in heparinized tubes, centrifuged at 3,000×g for 15 min at 4°C to separate plasma for blood component analysis. The remaining 5 mL was placed in vacuum tubes containing heparin sodium (750 IU). Both whole blood and plasma samples were stored at -20°C. Plasma urea nitrogen, glucose, and total protein concentrations were determined using an automatic biochemical analyzer (7020, Hitachi, Japan). Plasma nitric oxide (NO) concentration was measured by nitrate reductase method (kit purchased from Nanjing Jiancheng Bioengineering Institute). Whole blood AA concentrations were measured by liquid chromatography-mass spectrometry (UPLC-MSMS, AB Sciex, USA).

Blood flow data collection: Mammary blood flow was continuously measured by ultrasonic blood flow probes (MC6PSS-LS-WCS10-GC, Transonic Systems Inc., USA) placed in the external pudendal artery.

1.4 Calculation Formulas

Mammary AA clearance rate (L/d) = [(CA - CMV)/CMV] × MBF

Mammary AA uptake from whole blood (mol/h) = (CA - CMV) × MBF/24

Absorption-to-output ratio = Mammary AA uptake from whole blood (mol/d) / Milk AA output (mol/d)

AA lactation conversion efficiency = Milk AA output (mol/d) / Intestinally absorbable AA flux (mol/d)

Where: CA and CMV are AA concentrations in carotid artery and mammary vein whole blood (mol/L), respectively; MBF is mammary (external pudendal artery) blood flow (L/d), expressed as the average value throughout the period.

1.5 Statistical Analysis

Data were analyzed using the Linear-Models-ANOVA procedure in SAS 8.2 according to Latin square design. Duncan' s multiple comparison was used for mean separation. The statistical model was:

Yijk = + i + j + k + eijk

Where: is the random variable, i is the treatment effect, j is the period effect, k is the animal effect, and eijk is the interaction effect of treatment, period, and animal. Results provided three P-values for treatment, period, and animal effects. Significance was determined based on treatment P-values: P 0.05 indicated significant difference, and $0.05<P$ 0.10 indicated a trend.

2.1 Lactation Performance

As shown in Table 4, infusion of AA mixture lacking Arg, Thr, or His had no significant effect on milk yield ($P>0.05$) and did not differ significantly in milk protein percentage or milk protein yield ($P>0.05$). Compared with group C, the -Arg group showed significantly increased milk fat percentage and milk fat yield (P 0.05); the -Arg and -His groups showed significantly decreased lactose percentage (P 0.05); the -Arg group showed significantly increased fat-to-protein ratio (1.10 vs. 0.89, P 0.05), while the -Thr and -His groups showed an increasing trend in fat-to-protein ratio ($0.05<P$ 0.10).

2.2 Plasma Biochemical Indices and Mammary Blood Flow

As shown in Table 5, infusion of AA mixture lacking Arg, Thr, or His had no significant effect on plasma glucose, total protein, or NO concentrations ($P>0.05$). Compared with group C, the -Arg group showed a decreasing trend in total protein concentration ($0.05<P$ 0.10), while the -Thr group showed an increasing trend ($0.05<P$ 0.10). The -Thr group exhibited significantly increased urea nitrogen concentration (P 0.05), while the -His and -Arg groups showed an increasing trend in plasma urea nitrogen ($0.05<P$ 0.10). Mammary blood flow increased by 22.2% and 20.6% in the -Thr and -Arg groups, respectively (P 0.05).

2.3 Blood Amino Acid Concentrations in Jugular Arteries

As shown in Table 6, compared with group C, the -Arg group showed significantly decreased Arg concentration in jugular artery blood (P 0.05); the -Thr group showed significantly decreased Thr concentration (P 0.05) and significantly increased His concentration (P 0.05); the -His group showed no significant change in His concentration ($P>0.05$).

2.4 Blood Amino Acid Concentrations in Mammary Veins

As shown in Table 7, compared with group C, the -Arg group showed a decreasing trend in Arg concentration in mammary vein blood ($0.05<P$ 0.10); the -Thr group showed significantly decreased Thr concentration (P 0.05) and significantly increased His concentration (P 0.05); the -His group showed no significant change in His concentration ($P>0.05$).

2.5 Mammary Amino Acid Clearance Rates

As shown in Table 8, compared with group C, the -Arg group showed no significant effect on Arg mammary clearance rate ($P>0.05$), but numerically increased from 178.34 to 224.56 (25.9% increase). The -Thr group showed significantly increased Thr mammary clearance rate (P 0.05), rising from 232.4 to 695.4 (199% increase), while His mammary clearance rate significantly decreased (P 0.05). The -His group showed no significant change in His mammary clearance rate ($P>0.05$).

2.6 Mammary Amino Acid Uptake

As shown in Table 9, single AA deficiency infusion had no significant effect on mammary uptake of the deficient AA ($P>0.05$). Compared with group C, the -Thr group showed significantly decreased His mammary uptake (P 0.05).

2.7 Mammary Amino Acid Absorption-to-Output Ratio

As shown in Table 10, single AA deficiency infusion had no significant effect on the absorption-to-output ratio of the deficient AA ($P>0.05$). Compared with group C, the -Thr group showed significantly decreased absorption-to-output ratio for His (P 0.05) and significantly increased ratios for Arg, Met, Leu, Phe, Ile, and Asp (P 0.05). The total EAA absorption-to-output ratio increased from 1.10 to 1.48 (P 0.05).

2.8 Mammary Amino Acid Lactation Conversion Efficiency

As shown in Table 11, compared with group C, the -Arg group showed significantly increased Arg lactation conversion efficiency ($P<0.05$), rising from 4.02 to 5.04. The -Thr group showed significantly increased Thr lactation conversion efficiency (P 0.05), rising from 9.09 to 12.23. The -His group showed an increasing trend in His lactation conversion efficiency ($0.05<P$ 0.10).

3.1 Mammary Blood Flow

In two studies by Bequette et al. involving intravenous infusion of AA mixture lacking Leu and abomasal infusion of AA mixture lacking His, blood flow increased by 17% and 36%, respectively. Other studies have also demonstrated that AA deficiency or imbalance significantly increased or tended to increase mammary blood flow. In the present study, abomasal infusion of AA mixture singly deficient in Arg or Thr significantly increased mammary blood flow by 22.2% and 20.6%, respectively, consistent with previous reports. However, the effect on mammary blood flow may not be a direct effect of the deficient AA. Bequette et al. proposed that increased mammary blood flow might be related to reduced His supply to the mammary gland, as His decarboxylation in the mammary gland produces histamine, which has vasoconstrictive effects. When His concentration in the external pudendal artery decreases, mammary histamine synthesis decreases, reducing vasoconstriction and thereby increasing mammary blood flow. However, in this study, Thr deficiency significantly increased arterial His concentration while still significantly increasing mammary blood flow, indicating that His is not a decisive factor in mammary blood flow regulation.

Weekes et al. reported that increased mammary blood flow is usually accompanied by elevated NO concentration, which acts as a vasodilator. In this study, plasma NO concentration changes did not correspond with mammary blood flow changes; notably, Arg deficiency decreased plasma NO concentration by 38.3% while increasing mammary blood flow by 22.2%. Therefore, excluding differences in NO production and sampling sites and experimental errors, these results also demonstrate that NO is not a decisive factor in mammary blood flow regulation. The regulatory mechanism of mammary blood flow when nutrient supply to mammary tissue changes requires further investigation.

3.2 Arteriovenous Amino Acid Concentrations

Numerous studies have demonstrated that single AA infusion, whether intravenous or abomasal/duodenal, typically increases arterial concentration of that AA, while single AA deficiency infusion decreases its arterial concentration. Bequette et al. found that abomasal infusion of complete AA mixture lacking His in lactating goats decreased His arterial concentration from 73 mol/L to 8 mol/L without significantly affecting most other AA concentrations. In a study by Cant et al. with lactating dairy cows, infusion of His-deficient AA mixture into the iliac artery at 30 g/d significantly decreased His arterial concentration from 42 mol/L to 13 mol/L, consistent with the present study. Arg or Thr deficiency significantly decreased their arterial concentrations, while His deficiency had no significant effect. The reason may be that in Bequette et al.’ s His deficiency study, the complete mixture group received 4.4 g/d His, a 250% increase in His infusion. In Korhonen et al.’ s study, His concentration did not change significantly with infusion rates from 0 to 2 g/d. In this study, AA composition in the complete mixture was based on goat rumen microbial protein, with His infusion at 0.6-1.06 g, plus effects of rumen-undegraded His from the basal diet, thus not causing significant changes in His arterial concentration.

Bequette et al. summarized that mammary vein AA concentrations are strongly correlated with arterial concentrations, i.e., venous AA concentrations change with arterial concentrations. This study confirmed this: Thr venous concentration decreased significantly with arterial concentration, and Arg deficiency tended to decrease free Arg concentration in venous blood.

3.3 Mammary Amino Acid Metabolism

Mammary clearance rate of AA from milk artery blood reflects mammary AA uptake and utilization. Mammary AA clearance rate integrates changes in arteriovenous difference and mammary blood flow, providing better indication of mammary AA uptake than absorption rate alone. Bequette et al. reported that in an intravenous infusion study with Leu-deficient AA mixture, Leu deficiency decreased arterial concentration from 94 mol/L to 72.6 mol/L and venous concentration from 54.4 mol/L to 35.9 mol/L, while Leu mammary extraction rate increased from 52% to 71%. In Bequette et al.’ s abomasal infusion study with His-deficient AA mixture, His arterial concentration decreased significantly from 73 mol/L to 8 mol/L, and His mammary clearance rate increased 43-fold, while other AA clearance rates decreased to 1/3-1/2. Therefore, when AA supply is deficient or imbalanced, the mammary gland can self-regulate by altering AA absorption capacity and increasing clearance rate. In this study, abomasal infusion of Arg-deficient AA mixture did not significantly affect Arg mammary clearance rate but numerically increased it from 178.34 to 224.56 (25.9% increase). Abomasal infusion of Thr-deficient AA mixture significantly increased Thr mammary clearance rate from 232.4 to 695.4 (199% increase). Thus, when AA deficiency reduces arterial concentration of a specific AA, the mammary gland enhances its absorption and transport capacity for that AA.

Mammary AA uptake represents the most direct result of the mammary gland’ s self-regulation to enhance AA transport and absorption. Current understanding suggests that mammary uptake of EAA is largely unaffected by AA deficiency and can be maintained stable through self-regulation. Bequette et al. reported that in an intravenous infusion study with Leu-deficient AA mixture, Leu deficiency significantly decreased arterial concentration from 94 mol/L to 72.6 mol/L, but net mammary uptake of Leu did not change significantly. In Bequette et al.’ s abomasal infusion study with His-deficient AA mixture, His arterial concentration decreased significantly from 73 mol/L to 8 mol/L, yet mammary His net uptake remained unchanged with only a slight decrease. In this single AA deficiency study, mammary uptake of the deficient AA did not decrease significantly with reduced arterial concentration, maintaining relative stability and supporting the argument that mammary AA uptake is independent of plasma AA concentration. Mammary AA absorption and transport are highly efficient but are not the limiting step for milk protein synthesis.

In summary, when supply of a specific AA to the mammary gland is deficient, the mammary gland can significantly enhance its absorption capacity for that AA by increasing blood flow and clearance rate, thereby eliminating or at least alleviating the deficiency of nutritional substrates for milk protein synthesis caused by the AA deficiency. This aligns with the view proposed by Bequette et al. and Mackle et al. that the mammary gland can regulate blood flow and AA transport capacity according to its needs to meet lactation requirements.

In this study, Thr deficiency significantly decreased His absorption-to-output ratio, while significantly increasing ratios for Arg, Met, Leu, Phe, Ile, and Asp, with total EAA absorption-to-output ratio increasing from 1.10 to 1.48. Considering that Thr deficiency significantly increased arterial His concentration and decreased His mammary extraction rate and uptake, Thr deficiency likely severely impaired His uptake by mammary epithelial cells, making His a limiting factor for milk protein synthesis, reducing milk protein synthesis and secretion, and decreasing the amount of EAA used for milk protein synthesis. The excess EAA absorbed by the mammary gland were then oxidized or utilized for other non-lactation functions, resulting in increased absorption-to-output ratios for other EAA. The mechanism by which Thr deficiency affects His uptake remains unclear.

Additionally, this study revealed large variations in mammary absorption-to-output ratios among EAA. Arg showed the highest ratio (2.77-4.38), while His ratios were all less than 1. Raggio et al. found that at high supply levels, His mammary uptake equaled net splanchnic flux, but at low supply levels, uptake was lower than net splanchnic flux, suggesting that supply may not meet lactation requirements. This indicates that at low supply levels, endogenous supplementation from His-containing peptides or proteins may be necessary. In this study, mammary uptake of free His was far lower than the total His secreted into milk, possibly related to endogenous supplementation from His peptides or proteins.

3.4 Lactation Performance

Current results on the effects of single AA deficiency in infused AA mixtures on milk yield, milk protein percentage, and milk protein yield are inconsistent. Doepel et al. found that abomasal infusion of Arg-deficient AA mixture significantly decreased Arg uptake but had no significant effects on milk yield, milk protein percentage, or milk protein yield. Bequette et al. found that intravenous infusion of Leu-deficient AA mixture significantly decreased Leu arterial concentration without affecting arteriovenous difference or mammary Leu uptake, and had no effect on milk yield or milk protein yield. However, Bequette et al. found that abomasal infusion of His-deficient AA mixture significantly decreased both His influx to and efflux from the mammary gland, but the reduction in efflux was insufficient to offset the reduction in influx, ultimately significantly decreasing milk protein percentage and tending to decrease milk yield. Milk protein yield decreased from 118 g/d to 97 g/d. The inconsistent results among studies are likely related to dietary interference, as basal diets may have already provided sufficient AA, preventing the achievement of AA deficiency. In this study, single AA deficiency in the infused mixture had no significant effects on milk yield, milk protein percentage, or milk protein yield, confirming the powerful self-regulatory capacity of the mammary gland when dietary effects are excluded. However, it should be noted that although Thr deficiency did not affect mammary Thr uptake, His uptake decreased significantly, becoming a limiting factor for milk protein synthesis and secretion, thus numerically reducing milk protein yield.

AA imbalance often leads to increased milk fat percentage and fat-to-protein ratio. Two possible reasons for increased fat-to-protein ratio due to AA deficiency or imbalance are: first, increased mammary blood flow during AA deficiency increases delivery of milk fat synthesis substrates to the mammary gland per unit time, enhancing milk fat synthesis; second, when certain AA are deficient, reduced milk protein synthesis within the mammary gland leaves other AA in relative excess, which are converted to milk fat synthesis substrates, promoting milk fat synthesis. In this study, Arg-deficient AA mixture infusion significantly increased milk fat percentage and yield and fat-to-protein ratio (0.89 vs. 1.01). Thr- and His-deficient AA mixture infusions showed non-significant but increasing trends in fat-to-protein ratio. Therefore, fat-to-protein ratio can reflect AA balance status to some extent.

3.5 Lactation Conversion Efficiency

According to the Cornell model, abomasal infusion of AA mixture singly deficient in Arg, Thr, and His decreased intestinally digestible AA flux by 18.4% (57.8 mmol/d vs. 47.2 mmol/d), 41.7% (53.2 mmol/d vs. 31.0 mmol/d), and 19.6% (23.1 mmol/d vs. 18.7 mmol/d), respectively. Milk artery AA flux decreased by 3.4% (13.9 mmol/d vs. 13.4 mmol/d), 34.8% (10.1 mmol/d vs. 6.6 mmol/d), and 14.7% (5.2 mmol/d vs. 4.4 mmol/d), respectively. Lactation conversion efficiency increased by 25.4% (4.02 vs. 5.04), 34.5% (9.09 vs. 12.23), and 14.6% (10.51 vs. 12.04), respectively. These results indicate that splanchnic tissues have buffering effects on Arg, Thr, and His deficiency, likely due to reduced AA catabolism in visceral organs such as the liver (for His) or pancreas (for Thr) when AA are deficient. The buffering effect was most pronounced for Arg, possibly related to de novo Arg synthesis in the kidneys. All AA deficiencies increased lactation conversion efficiency, indicating substantial potential for improving AA lactation conversion efficiency, with Arg showing the greatest potential. Two possible reasons are: first, endogenous Arg synthesis can compensate for infusion-induced deficiency; second, Arg has a high mammary absorption-to-output ratio, with uptake far exceeding milk output, allowing the mammary gland to improve Arg lactation conversion efficiency by reducing intramammary Arg oxidation.

Conclusions

1. Abomasal infusion of AA mixture singly deficient in Arg, Thr, or His had no significant effects on milk yield or milk protein percentage and did not significantly affect mammary uptake of the deficient AA, indicating that the mammary gland possesses self-regulatory capacity in AA absorption and transport.
2. Abomasal infusion of AA mixture singly deficient in Arg, Thr, or His increased the lactation conversion efficiency of the deficient AA.
3. Abomasal infusion of AA mixture singly deficient in Thr significantly increased Thr mammary clearance rate, while Arg and His deficiency had no significant effect on mammary clearance rate of the deficient AA.

References

[1] BERTHIAUME R, THIVIERGE M C, PATTON R A, et al. Effect of ruminally protected methionine on splanchnic metabolism of amino acids in lactating dairy cows[J]. Journal of Dairy Science, 2006, 89(5): 1621-1634.

[2] BEQUETTE B J, HANIGAN M D, LAPIERRE H. Mammary uptake and metabolism of amino acids by lactating ruminants[M]//D'MELLO J P F. Amino

acids in animal nutrition. Cambridge: CABI Publishing, 2003: 347-365.

[3] CANT J P, BERTHIAUME R, LAPIERRE H, et al. Responses of the bovine mammary glands absorptive supply single amino acids[J]. Canadian Journal of Animal Science, 2003, 83(3): 341-355.

[4] MABJEESH S J, KYLE C E, MACRAE J C, et al. Lysine metabolism by the mammary gland of goats at two stages of lactation[J]. Journal of Dairy Science, 2000, 83(5): 996-1003.

[5] AFRC. Agricultural and food research council[S]. Wallingford: CAB International, 1993.

[6] 甄玉国. 内蒙古白绒山羊氨基酸利用和蛋白质周转规律的研究 [D]. 博士学位论文. 呼和浩特: 内蒙古农业大学, 2002.

[7] BEQUETTE B J, BACKWELL F R C, MACRAE J C, et al. Effect of intravenous amino acid infusion on leucine oxidation across the mammary gland of the lactating goat[J]. Journal of Dairy Science, 1996, 79(12): 2217-2224.

[8] BEQUETTE B J, HANIGAN M D, CALDER A G, et al. Amino acid exchange by the mammary gland of lactating goats when histidine limits milk production[J]. Journal of Dairy Science, 2000, 83(4): 765-775.

[9] WEEKES T L, LUIMES P H, CANT J P. Responses to amino acid imbalances and deficiencies in lactating dairy cows[J]. Journal of Dairy Science, 2006, 89(6): 2177-2187.

[10] CANT J P, TROUT D R, QIAO F, et al. Milk composition responses to unilateral arterial infusion of complete and histidine-lacking amino acid mixtures to the mammary glands of cows[J]. Journal of Dairy Science, 2001, 84(5): 1192-1200.

[11] KORHONEN M, VANHATALO A, VARVIKKO T, et al. Responses to graded postruminal doses of histidine in dairy fed grass silage diets[J]. Journal of Dairy Science, 2000, 83(11): 2596-2608.

[12] HUHTANEN P, VANHATALO A, VARVIKKO T. Effects of abomasal infusions of histidine, glucose, and leucine on milk production and plasma metabolites of dairy cows fed grass silage diets[J]. Journal of Dairy Science, 2002, 85(1): 204-216.

[13] MACKLE T R, DWYER D A, INGVARTSEN K L, et al. Evaluation of whole blood and plasma in the interorgan supply of free amino acids for the mammary gland of lactating dairy cows[J]. Journal of Dairy Science, 2000, 83(6): 1300-1309.

[14] RAGGIO G, PACHECO D, BERTHIAUME R, et al. Effect of level of metabolizable protein splanchnic flux of amino acids lactating dairy cows[J]. Journal of Dairy Science, 2004, 87(10): 3461-3472.

[15] DOEPEL L, LAPIERRE H. Deletion of arginine from an abomasal infusion of amino acids decrease milk protein yield in Holstein cows[J]. Journal of Dairy Science, 2011, 94(2): 864-873.

[16] VARVIKKO T, VANHATALO A, JALAVA T, et al. Lactation and metabolic responses to graded abomasal doses of methionine and lysine in dairy cows fed grass silage diets[J]. Journal of Dairy Science, 1999, 82(12): 2659-2673.

Note: Figure translations are in progress. See original paper for figures.

Source: ChinaXiv —Machine translation. Verify with original.