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# Abomasal amino acid infusion in postpartum dairy cows: Effect on whole-body, splanchnic, and mammary amino acid metabolism

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## ABSTRACT

Nine Holstein cows with rumen cannulas and indwelling catheters in splanchnic blood vessels were used in a generalized randomized incomplete block design with repeated measures to study the effect of increased early postpartum AA supply on splanchnic and mammary AA metabolism. At calving, cows were blocked according to parity (second and third or greater) and allocated to 2 treatments: abomasal infusion of water (CTRL; n = 4) or free AA with casein profile (AA-CN; n = 5) in addition to a basal diet. The AA-CN infusion started with half of the maximal dose at the calving day (1 d in milk; DIM) and then steadily decreased from 791 to 226 g/d until 29 DIM. On 5, 15, and 29 DIM, 6 sample sets of arterial, portal, hepatic, and mammary blood were taken at 45-min intervals. Over the whole period, increasing AA supply increased milk (+7.8 ± 1.3 kg/d) and milk protein yields (+220 ± 65 g/d) substantially. The increased milk yield was not supported by greater dry matter intake (DMI) as, overall, DMI decreased with AA-CN (−1.6 ± 0.6 kg/d). Arterial concentrations of essential AA were greater for AA-CN compared with CTRL. The net portal-drained viscera (PDV) release of His, Met, and Phe was greater for AA-CN compared with CTRL, and the net PDV recovery of these infused AA ranged from 72 to 102% once changes in DMI were accounted for. The hepatic removal of these AA was increased equivalently to the increased net PDV release, resulting in an unaltered net splanchnic release. The net PDV release of Ile, Leu, Val, and Lys tended to be greater for AA-CN, and the net PDV recovery of these infused AA ranged from 69 to 73%, indicating increased PDV metabolism with AA-CN. The fractional hepatic removal of these AA did not differ from zero and was unaffected by the increased supply. Consequently, the splanchnic release of these AA was approximately equivalent to their net PDV release for both CTRL and AA-CN. Overall, greater early postpartum AA supply increased milk and milk protein yields substantially based on increased mammary AA uptake. The PDV metabolism of branched-chain AA and Lys were increased, whereas it seemed to be unaffected for other essential AA when the intestinal AA supply was increased. On a net basis, the liver removed more group 1 AA (His, Met, Phe, and Trp) for anabolism and catabolism when the early postpartum AA supply was increased. Thus, increasing the postpartum AA supply increased splanchnic and mammary consumption of AA; hence, the protein deficiency persisted.

**Key words:** dairy cow, parturition, amino acid, splanchnic, mammary

## INTRODUCTION

It has long been recognized that the postpartum cow is in negative energy and protein balance because the high demand created by initiation of lactation is not covered by equivalent increases in nutrient supply from dietary intake. The consequences of negative energy balance and associated fat mobilization have received great focus during the past decades (e.g., Grummer, 1995; Ingvartsen et al., 2003; Loor, 2010). In contrast, the concomitant negative protein balance has received little attention, even though the extent and potential negative consequences of negative protein balance have been acknowledged (Grummer, 1995; Bell et al., 2000). The extent of postpartum negative protein balance was estimated to be greatest around 7 d postpartum, diminishing thereafter and becoming positive during wk 4 postpartum (Bell et al., 2000). Recently, a similar pattern was observed by relating the release of EAA from total splanchnic tissues to the secretion of EAA into milk protein (Larsen and Kristensen, 2009; Dalbach et al., 2011). Consequently, AA are mobilized during this period from skeletal muscles (Gibb et al., 1992) and

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postpartum involution of smooth muscle in the uterus (Gibb et al., 1992; Andrew et al., 1994).

Several decades ago, Ørskov et al. (1977) observed a positive response in milk production to casein infusion in early lactation cows compared with no response to glucose infusion. Similarly, Larsen et al. (2014b) observed a large increase in milk and milk protein yields in response to an abomasal casein infusion initiated at calving. In this later study, approximately two-thirds of the additional protein supply was utilized to support the increased milk protein synthesis. However, the partitioning of the additionally supplied AA in the early postpartum period among splanchnic, mammary, and other peripheral tissues has yet to be investigated. Available quantitative data on hepatic uptake of AA in the periparturient dairy cows have indicated that the fractional hepatic removal of individual EAA is greater prepartum and in the first week postpartum compared with later in the postpartum period, despite a lower AA supply (Doepel et al., 2009; Larsen and Kristensen, 2013). However, no data are available on the effect of increasing AA supply in the first weeks of lactation on the coordination of AA metabolism between portal-drained viscera (**PDV**), hepatic tissue, and mammary gland.

Therefore, the overall objective of the present study was to investigate the effect of increasing the AA supply to postpartum dairy cows on splanchnic and mammary AA metabolism, and on whole-body AA balance. We hypothesized that abomasal infusion of AA will increase the net PDV release of AA and, despite a concomitant increase in hepatic AA removal, the net splanchnic release of AA will increase, thereby allowing increased mammary AA uptake and milk protein yield.

## MATERIALS AND METHODS

The experimental protocol was approved by the Institutional Committee for Animal Care at Agriculture and Agri-Food Canada, Dairy and Swine Research and Development Centre in Sherbrooke (QC, Canada), and the animals were cared for in accordance with the guidelines of the Canadian Council on Animal Care (2009).

### *Animals and Experimental Design*

Nine multiparous Holstein cows (Table 1) were used in a generalized randomized incomplete block design with repeated measurements (5, 15, and 29 DIM). Cows were blocked according to parity (second: n = 4, >second: n = 5; Table 1) and, within block, randomly assigned to 1 of 2 treatments: continuous abomasal infusion of either water (**CTRL**, n = 4) or a mixture of free AA, all L-isomers, with casein profile (**AA-CN**; n = 5), both initiated on day of calving (designated as 1 DIM).

**Table 1.** Description of cows, calf birth weights (mean ± SD), and treated illnesses

| Item | Treatment[1] CTRL | AA-CN |
|---|---|---|
| Number of cows | 4 | 5 |
| Second parity, no. | 2 | 2 |
| >Second parity, no. | 2 | 3 |
| BW at dry off, kg | 735 ± 94 | 735 ± 104 |
| Previous 305-d ECM, kg | 10,410 ± 1,561 | 10,294 ± 1,361 |
| Calf birth weight, kg | 40.0 ± 5.0 | 44.9 ± 6.2 |
| Treated illnesses, no. | | |
| Calving problems | — | 1 |
| Mastitis | — | 1 |
| Metritis | — | — |
| Milk fever | — | 1 |
| Leg problems | — | — |
| Digestion disorders | — | 1 |
| Other[2] | 1 | 3 |

[1] Treatments were continuous abomasal infusion of water (CTRL) or a mixture of free AA with casein profile (AA-CN) from 1 to 29 DIM.

[2] Breath sounds, udder cleft dermatitis, foot fungus.

Before calving (11–20 wk), cows were fitted with rumen cannulas (Bar Diamond Inc., Parma, ID) according to Duffield (1999). During the dry period, approximately 7 wk before planned calving, cows were surgically implanted with a catheter in the hepatic portal vein, hepatic vein, 2 mesenteric veins, and 1 mesenteric artery (Huntington et al., 1989). During the surgery, the right carotid artery was also raised to a subcutaneous position to provide access to arterial blood if necessary. On the day of calving, an abomasal infusion device was placed in the abomasum by hand through the ruminal cannula and the reticular-omasal orifice. The infusion device was constructed with a flange to secure abomasal placement (Gressley et al., 2006) and was checked manually every third day and in the morning of each sampling day.

The AA-CN rate of infusion was (mean ± SD) 791 ± 12 g/d from 2 to 6 DIM, followed by daily decreases of 28.3 ± 1.0 g/d, with 3-d plateaus around the blood sampling days, giving 558 ± 2 g/d at 14 to 16 DIM and 226 ± 0.5 g/d at 28 to 30 DIM. At 1 DIM, the AA-CN rate of infusion was half of the maximal dose. The rates of infusion were estimated to cover the estimated decreasing EAA deficit with advancing DIM (Dalbach et al., 2011). The AA mixture was composed according to the AA composition of casein except that Tyr was replaced by Phe and part of the Glu by Gln, due to solubility constraints, as described previously (Galindo et al., 2011). The AA mixture was prepared every second or third day by mixing in hot tap water. Each day,