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Original Research Article

Dietary *N*-carbamylglutamate or L-arginine improves fetal intestinal amino acid profiles during intrauterine growth restriction in undernourished ewes

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ABSTRACT

Our previous studies demonstrated that prenatal in utero growth restriction impairs postnatal intestinal function. Thus, improving postpartal intestinal absorption capacity and growth by manipulating the maternal diet prepartum is of importance. This work was conducted to determine whether supplementation of *N*-carbamylglutamate (NCG) or rumen-protected L-arginine (RP-Arg) increased fetal intestinal amino acid (AA) profiles in intrauterine growth retardation (IUGR) fetuses. On d 35 of gestation, Hu ewes ($n = 32$) carrying twin fetuses were randomized into 4 groups (8 ewes and 16 fetuses in each group), where diets were as follows: 100% of nutrient requirements recommended by National Research Council (NRC, 2007) (CON); 50% of nutrient requirements recommended by NRC (2007) (RES); RES + RP-Arg (20 g/d), (RES + ARG); and RES + NCG (5 g/d), (RES + NCG). On d 110 of gestation, both fetal and maternal tissues were collected and weighed. Compared with RES, solute carrier family 1, member 5 (*SLC1A5*) was upregulated ($P < 0.05$) within fetal jejunum, duodenum and ileum when supplementing NCG and RP-Arg. Relative to RES, RP-Arg or NCG supplementation to RES resulted in upregulation ($P < 0.05$) of peptide transporter 1 protein abundance within the fetal ileum. NCG or RP-Arg supplementation to RES also upregulated phosphorylated mechanistic target of rapamycin (pmTOR)-to-mTOR ratio in the fetal ileum induced by IUGR ($P < 0.05$). As a result, during IUGR, supplementation of Arg or NCG affected intestinal AA profiles in the fetus in part through controlling mTOR signal transduction as well as AA and peptide transport. Future studies should be conducted to understand the role (if any) of the placenta on the improvement of growth and AA profiles independent of the fetal intestine. This would help demonstrate the relative contribution of intestinal uptake in fetal life.

1. Introduction

Nutrient insufficiency to the conceptus alters function and growth of the placenta, leading to intrauterine growth retardation (IUGR) (Wu et al., 2006). Although from a conceptual point of view the situation may be evident, actual identification of nutritional insufficiencies may be more complex. According to most neonatologists, a newborn that is small for gestational date, likely due to IUGR, is one whose weight falls below the 10th percentile (Romo

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