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Thesis Defense

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Role of essential amino acid transporter LAT4 in mice

LAT4 (SLC43A2) is a facilitated diffusion uniporter specific for essential neutral amino acids highly expressed on the basolateral membranes of epithelial cells in small intestine (SI) and kidney. Its global knockout (KO) was previously shown to lead to a malnutrition-like phenotype with early postnatal death. To investigate in more details the function of LAT4 *in vivo*, we generated various tissue-specific and inducible knockout mouse models using a new conditional LAT4 null allele. We demonstrated that the KO of LAT4 in SI leads to an absorption defect accompanied by a delayed gastrointestinal motility and in kidney to a reabsorption defect of LAT4 substrates and of other neutral AAs mostly substrates of the amino acid antiporter LAT2, implying their functional cooperation. Nonetheless, knocking out LAT4 in the SI or in the kidney did not affect the general well-being of mice, suggesting an important role for LAT4 in other organs. Introducing the LAT4 KO in TAT1 full KO background, we showed that these two uniporters play together a crucial role for the net transport of almost all AAs in the kidney but that in SI their function can be compensated by an alternative transport mechanism.

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