The L-type amino acid transporter LAT1 in leptin receptor-expressing neurons controls body weight and systemic energy homeostasis in mice.

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L-type amino acid transporter 1 (LAT1), encoded by solute carrier transporter 7a5 (*Slc7a5*), plays a crucial role in amino acid sensing and signal initiation in specific cell types. Although the pivotal expression and crucial role of LAT1 in the blood-brain-barrier have been determined, there is limited evidence regarding its potential role in neuronal cells. Here, we identify LAT1 as a pivotal amino acid transporter for the regulation of body weight control and systemic energy homeostasis through its expression in leptin receptor (LepR)-expressing neurons. The LepR-specific deletion of *Slc7a5* in mice led to obesity phenotype, with an increased white adipose tissue mass. Furthermore, *Slc7a5* deficiency in LepR-expressing neurons showed along with decreased insulin sensitivity, physical activity, and energy expenditure. These results suggest that LAT1 controls body weight and systemic energy homeostasis probably via its expression in hypothalamic neurons in mice, thereby providing a novel target for metabolic diseases such as obesity and type 2 diabetes.