SIGNIFICANCE STATEMENT

Renal amino acid reabsorption is crucial for the maintenance of whole body homeostasis and its impairment leads to several diseases such as cystinuria and Hartnup disorder. Whereas these and other well described aminoacidurias are caused by defects of luminal transport proteins, only one aminoaciduria, specifically of cationic amino acids, is associated with the dysfunction of a basolateral transporter. This work demonstrates *in vivo* the functional cooperation of two basolateral neutral amino acid transporters, LAT2 and TAT1, and shows that another basolateral transporter, y^+LAT1, can largely compensate for their defect. These findings reveal synergistic and compensatory reabsorption mechanisms in renal epithelial cells that can explain why no neutral aminoaciduria due to the defect of basolateral transporters has been identified in humans.