

SFB 35 Colloquia in Membrane Transport

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"Amino acid transport across epithelia and blood brain barrier"

Small intestine and kidney proximal tubule epithelia are important cellular barriers where transcellular (re)absorption of amino acid takes place. The defect of the luminal sodium-dependent amino acid transporter B0AT1 (SLC6A19) causes Hartnup disorder. This transporter and other members of the SLC6 family require for their efficient surface expression in kidney and intestine association with collectrin (Tmem27) or ACE2, respectively. Since some mutations of B0AT1 differentially interact with collectrin and ACE2, a new mechanism leading the variable phenotype of Hartnup disorder is proposed. The closely related and previously orphan transporter XT2 (SC6A18) is shown to function as a higher affinity neutral amino acid transporter and to localize to the late kidney proximal tubule. We thus propose to rename it B0AT3. The basolateral amino acid transport machinery of these epithelia is less well understood and involves the interplay of exchangers and facilitated diffusion pathways such that it can fulfil, next to the efflux function, a role for specialized metabolic tasks and cellular housekeeping. A similarly complex task is that of the brain microvascular endothelial cells of the blood brain barrier across which an amino acid concentration gradient is also maintained.