

S6A19 Rabbit pAb

CatalogNo: YN1347

Key Features

Host Species

- Rabbit

Reactivity

- Human, Mouse, Rat

Applications

- WB, ELISA

MW

- 69kD (Observed)

Isotype

- IgG

Recommended Dilution Ratios

WB 1:500-2000

ELISA 1:5000-20000

Storage

Storage*

-15°C to -25°C/1 year (Do not lower than -25°C)

Formulation

Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

Basic Information

Clonality

Polyclonal

Immunogen Information

Immunogen

Synthesized peptide derived from part region of human protein

Specificity

S6A19 Polyclonal Antibody detects endogenous levels of protein.

Target Information

Gene name

SLC6A19 B0AT1

Protein Name	Sodium-dependent neutral amino acid transporter B(0)AT1 (Solute carrier family 6 member 19) (System B(0) neutral amino acid transporter AT1)		
	Organism	Gene ID	UniProt ID
	Human	340024;	Q695T7;
	Mouse		Q9D687;
	Rat		Q2A865;
Cellular Localization	Cell membrane ; Multi-pass membrane protein . Apical cell membrane ; Multi-pass membrane protein . Colocalizes with ACE2 on the apical membrane of cells lining villi of the jejunum, ileum and on kidney proximal tubules. .		
Tissue specificity	Robust expression in kidney and small intestine, with minimal expression in pancreas (PubMed:18424768, PubMed:15286787). Also expressed in stomach, liver, duodenum, ileocecum, colon and prostate. Not detected in testis, whole brain, cerebellum, fetal liver, spleen, skeletal muscle, uterus, heart or lung.		
Function	Disease:Defects in SLC6A19 are a cause of Hartnup disorder (HND) [MIM:234500]. HND is an autosomal recessive abnormality of renal and gastrointestinal neutral amino acid transport noted for its clinical variability. First described in 1956, HND is characterized by increases in the urinary and intestinal excretion of neutral amino acids. Individuals with typical Hartnup aminoaciduria may be asymptomatic, some develop a photosensitive pellagra-like rash, attacks of cerebellar ataxia and other neurological or psychiatric features. Although the definition of HND was originally based on clinical and biochemical abnormalities, its marked clinical heterogeneity has led to it being known as a disorder with a consistent pathognomonic neutral hyperaminoaciduria.,Function:Transporter that mediates epithelial resorption of neutral amino acids across the apical membrane of epithelial cells in the kidney and intestine. It appears that leucine is the preferred substrate, but all large neutral non-aromatic L-amino acids bind to this transporter. Uptake of leucine is sodium-dependent. In contrast to other members of the neurotransmitter transporter family, does not appear to be chloride-dependent.,similarity:Belongs to the sodium:neurotransmitter symporter (SNF) family.,tissue specificity:Robust expression in kidney and small intestine, with minimal expression in pancreas. Also expressed in stomach, liver, duodenum, ileocecum, colon and prostate. Not detected in testis, whole brain, cerebellum, fetal liver, spleen, skeletal muscle, uterus, heart or lung.,		

Validation Data

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