

## 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    | <a href="#">340024</a> ; | <a href="#">Q695T7</a> ; |
| Mouse    |                          | <a href="#">Q9D687</a> ; |
| Rat      |                          | <a href="#">Q2A865</a> ; |

**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

## | Contact information

Orders: order@immunoway.com  
Support: tech@immunoway.com  
Telephone: 877-594-3616 (Toll Free), 408-747-0185  
Website: <http://www.immunoway.com>  
Address: 2200 Ringwood Ave San Jose, CA 95131 USA



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