

**bs-13752R****[ Primary Antibody ]****Claudin 16 Rabbit pAb**

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**— DATASHEET —**

<b>Host:</b> Rabbit	<b>Isotype:</b> IgG	<b>Applications:</b> <b>IHC-P</b> (1:100-500) <b>IHC-F</b> (1:100-500) <b>IF</b> (1:100-500) <b>ICC/IF</b> (1:100-500) <b>ELISA</b> (1:5000-10000)  <b>Reactivity:</b> (predicted: Human, Mouse, Rat, Rabbit, Sheep, Cow, Chicken, Dog)  <b>Predicted MW.:</b> 34 kDa  <b>Subcellular Location:</b> Cell membrane
<b>Clonality:</b> Polyclonal		
<b>GeneID:</b> 10686	<b>SWISS:</b> Q9Y5I7	
<b>Target:</b> Claudin 16		
<b>Immunogen:</b> KLH conjugated synthetic peptide derived from human Claudin 16: 95-150/305. < Extracellular >		
<b>Purification:</b> affinity purified by Protein A		
<b>Concentration:</b> 1mg/ml		
<b>Storage:</b> 0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol. Shipped at 4°C. Store at -20°C for one year. Avoid repeated freeze/thaw cycles.		
<b>Background:</b> Tight junctions mediate the regulation of the paracellular pathway between epithelial and endothelial cells. They form close connections to eliminate the extracellular space and regulate the flow of solutes between cells. The human gene PCLN-1 (paracellin-1) is related to the claudin family of integral membrane proteins, which localize to tight junctions. PCLN-1 contains four transmembrane domains and intracellular amino and carboxy termini, characteristic of the other claudin family members, and is detected only at the tight junctions of kidney tissue. PCLN-1 forms an intercellular pore and controls the resorption of magnesium and calcium in the thick ascending limb of Henle (TAL). Mutations in PCLN-1 cause renal magnesium wasting, which may contribute to a rare autosomal recessive disease, renal hypomagnesemia with hypercalciuria and nephrocalcinosis.		

**— SELECTED CITATIONS —**

- **[IF=5.717]** Hwee-Yeong Ng. et al. Effect of Dapagliflozin and Magnesium Supplementation on Renal Magnesium Handling and Magnesium Homeostasis in Metabolic Syndrome. Nutrients. 2021 Nov;13(11):4088 IHC ;Rat. 34836340