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Rabbit Anti-phospho-SCNN1B (Thr615) antibody

SL5701R

Product Name:	phospho-SCNN1B (Thr615)
Chinese Name:	磷酸化上皮钠通道β2抗体
Alias:	SCNN1B(phospho Thr615); SCNN1B(phospho T615); Amiloride sensitive sodium channel subunit beta; Beta NaCH; ENaC beta; ENaCB; Epithelial Na(+) channel subunit beta; Epithelial Na+ channel beta subunit; Epithelial Na+ channel subunit beta; Epithelial sodium channel beta 2 subunit; Epithelial sodium channel beta 3 subunit; Nonvoltage gated sodium channel 1 beta subunit; Nonvoltage gated sodium channel 1 subunit beta; SCNEB; SCNN 1B; SCNNB_HUMAN; Sodium channel nonvoltage gated 1 beta (Liddle syndrome); Sodium channel nonvoltage gated 1 beta.
Organism Species:	Rabbit
Clonality:	Polyclonal
React Species:	Human,Mouse,Rat,Dog,Cow,Horse,Rabbit,Guinea Pig,
Applications:	WB=1:500-2000ELISA=1:500-1000IHC-F=1:400-800IF=1:100-500（Paraffin sections need antigen repair） not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Molecular weight:	73kDa
Cellular localization:	The cell membrane
Form:	Lyophilized or Liquid
Concentration:	1mg/ml
immunogen:	KLH conjugated Synthesised phosphopeptide derived from human SCNN1B around the phosphorylation site of Thr615:PG(p-T)PP<Cytoplasmic>
Lsotype:	IgG
Purification:	affinity purified by Protein A
Storage Buffer:	0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.
Storage:	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.

PubMed:	PubMed
Product Detail:	SCNN1B is a subunit of the epithelial sodium channel, ENaC. ENac has high sodium selectivity, low conductance, and amiloride sensitivity. The functional channel of ENaC is composed of at least 3 subunits, alpha (SCNN1A), beta (SCNN1B), and gamma (SCNN1G). The 3 subunits show sequence similarities to one another, indicating descent from a common ancestral gene. Each encodes a protein containing 2 transmembrane domains, with intracellular amino and carboxyl termini. **Function:** Sodium permeable non-voltage-sensitive ion channel inhibited by the diuretic amiloride. Mediates the electrodiffusion of the luminal sodium (and water, which follows osmotically) through the apical membrane of epithelial cells. Controls the reabsorption of sodium in kidney, colon, lung and sweat glands. Also plays a role in taste perception. **Subunit:** Probable heterotrimer containing one alpha, one beta and one gamma subunit. A delta subunit can replace the alpha subunit. Interacts with the WW domains of NEDD4, NEDD4L, WWP1 and WWP2. Interacts with the full length immature form of PCSK9 (pro-PCSK9). **Subcellular Location:** Apical cell membrane; Multi-pass membrane protein. Note=Apical membrane of epithelial cells. **Post-translational modifications:** Phosphorylated on serine and threonine residues (By similarity). **DISEASE:** Pseudohypoaldosteronism 1, autosomal recessive (PHA1B) [MIM:264350]: A rare salt wasting disease resulting from target organ unresponsiveness to mineralocorticoids. PHA1B is a severe form involving multiple organ systems, and characterized by an often fulminant presentation in the neonatal period with dehydration, hyponatremia, hyperkalemia, metabolic acidosis, failure to thrive and weight loss. Note=The disease is caused by mutations affecting the gene represented in this entry. The degree of channel function impairment differentially affects the renin-aldosterone system and urinary Na/K ratios, resulting in distinct genotype-phenotype relationships in PHA1 patients. Loss-of-function mutations are associated with a severe clinical course and age-dependent hyperactivation of the renin-aldosterone system. This feature is not observed in patients with missense mutations that reduce but do not eliminate channel function. Markedly reduced channel activity results in impaired linear growth and delayed puberty (PubMed:18634878). Liddle syndrome (LIDDS) [MIM:177200]: Autosomal dominant disorder characterized by pseudoaldosteronism and hypertension associated with hypokalemic alkalosis. The disease is caused by constitutive activation of the renal epithelial sodium channel. Note=The disease is caused by mutations affecting the gene represented in this entry. Bronchiectasis with or without elevated sweat chloride 1 (BESC1) [MIM:211400]: A

	debilitating respiratory disease characterized by chronic, abnormal dilatation of the bronchi and other cystic fibrosis-like symptoms in the absence of known causes of bronchiectasis (cystic fibrosis, autoimmune diseases, ciliary dyskinesia, common variable immunodeficiency, foreign body obstruction). Clinical features include sub-normal lung function, sinopulmonary infections, chronic productive cough, excessive sputum production, and elevated sweat chloride in some cases. Note=The disease is caused by mutations affecting the gene represented in this entry. **Similarity:** Belongs to the amiloride-sensitive sodium channel (TC 1.A.6) family. SCNN1B subfamily. **SWISS:** P51168 **Gene ID:** 6338 **Database links:** Entrez Gene: 6338Human Entrez Gene: 20277Mouse Entrez Gene: 24767Rat Omim: 600760Human SwissProt: P51168Human SwissProt: Q9WU38Mouse SwissProt: P37090Rat Unigene: 414614Human Unigene: 7709Mouse Unigene: 9807Rat **Important Note:** This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Picture:	

	Sample: Siha (Human)Cell Lysate at 40 ug A549 (Human) Cell Lysate at 40 ug Primary: Anti-p-SCNN1B(Thr615)(SL5701R)at 1/300 dilution Secondary: IRDye800CW Goat Anti-RabbitIgG at 1/20000 dilution Predicted band size: 73kD Observed band size: 73kD