

Product name:	ENaC β (phospho Thr615) Rabbit Polyclonal Antibody
Cat number:	ABN04607
Conjugate:	Unconjugated
Size:	100 μ L
Clone:	Polyclonal
Concentration:	1mg/ml
Host:	Rabbit
Isotype:	IgG
Immunogen:	The antiserum was produced against synthesized peptide derived from human Nonvoltage-gated Sodium Channel 1 around the phosphorylation site of Thr615. AA range:581-630
Reactivity:	Human,Mouse,Rat
Applications:	WB 1:500-1:2000,IHC 1:100-1:300,ICC/IF 1:200-1:1000,ELISA 1:5000-1:20000
Molecular Weight:	68kDa
Purification:	Affinity purification
Form:	Liquid
Buffer:	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.
Storage:	Store at 4°C short term. Aliquot and store at -20°C for 12 months. Avoid freeze/thaw cycles.

Background:

Nonvoltage-gated, amiloride-sensitive, sodium channels control fluid and electrolyte transport across epithelia in many organs. These channels are heteromeric complexes consisting of 3 subunits: alpha, beta, and gamma. This gene encodes the beta subunit, and mutations in this gene have been associated with pseudohypoaldosteronism type 1 (PHA1), and Liddle syndrome. [provided by RefSeq, Apr 2009],disease:Defects in SCNN1B are a cause of autosomal recessive pseudohypoaldosteronism type 1 (PHA1) [MIM:264350]. PHA1 is a rare salt wasting disease resulting from target organ unresponsiveness to mineralocorticoids. There are 2 forms of PHA1: the autosomal recessive form that is severe, and the dominant form which is more milder and due to defects in mineralocorticoid receptor. Autosomal recessive PHA1 is characterized by an often fulminant presentation in the neonatal period with dehydration, hyponatraemia, hyperkalaemia, metabolic acidosis, failure to thrive and weight loss.,disease:Defects in SCNN1B are a cause of Liddle syndrome [MIM:177200]. It is an 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.,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.,PTM:Phosphorylated on serine and threonine residues.,similarity:Belongs to the amiloride-sensitive sodium channel family.,subcellular location:Apical membrane of epithelial cells.,subunit:Heterotetramer of two 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.,