



ENaC β (phospho Thr615) Polyclonal Antibody

Catalog No	YP-Ab-16350
Isotype	IgG
Reactivity	Human;Mouse;Rat
Applications	WB;IHC;IF;ELISA
Gene Name	SCNN1B
Protein Name	Amiloride-sensitive sodium channel subunit beta
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
Specificity	Phospho-ENaC β (T615) Polyclonal Antibody detects endogenous levels of ENaC β protein only when phosphorylated at T615.
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Source	Polyclonal, Rabbit,IgG
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.
Dilution	Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. Immunofluorescence: 1/200 - 1/1000. ELISA: 1/10000. Not yet tested in other applications.
Concentration	1 mg/ml
Purity	$\geq 90\%$
Storage Stability	-20°C/1 year
Synonyms	SCNN1B; Amiloride-sensitive sodium channel subunit beta; Beta-NaCH; Epithelial Na(+) channel subunit beta; Beta-ENaC; ENaCB; Nonvoltage-gated sodium channel 1 subunit beta; SCNEB
Observed Band	68kD
Cell Pathway	Apical cell membrane ; Multi-pass membrane protein . Cytoplasmic vesicle membrane . Apical membrane of epithelial cells. .
Tissue Specificity	Detected in placenta, lung and kidney (PubMed:7762608). Expressed in kidney (at protein level) (PubMed:22207244).
Function	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

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],

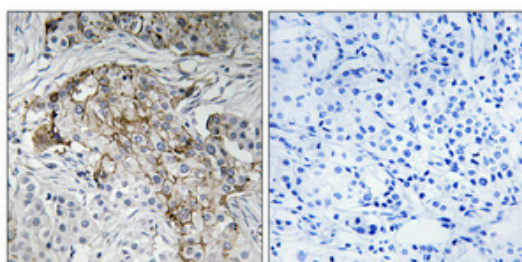
matters needing attention

Avoid repeated freezing and thawing!

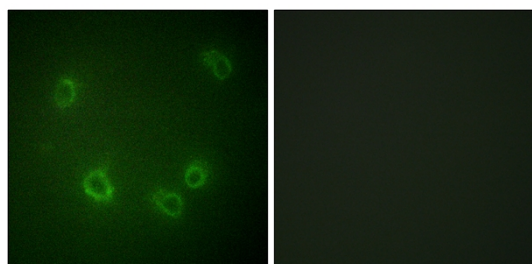
Usage suggestions

This product can be used in immunological reaction related experiments. For more information, please consult technical personnel.

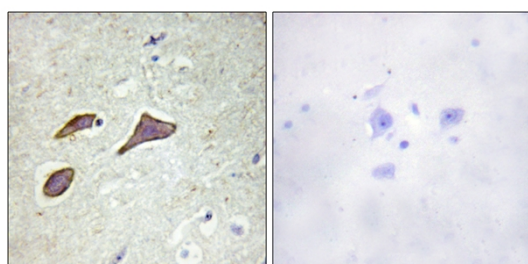
Products Images



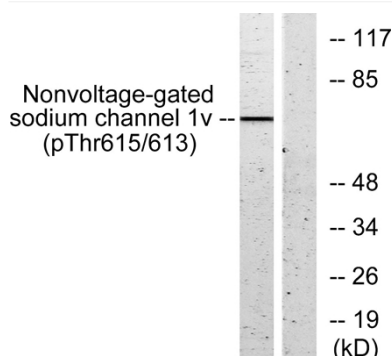
Immunohistochemical analysis of paraffin-embedded Human breast cancer. Antibody was diluted at 1:100(4° overnight). High-pressure and temperature Tris-EDTA,pH8.0 was used for antigen retrieval. Negative contrl (right) obtained from antibody was pre-absorbed by immunogen peptide.



Immunofluorescence analysis of COS7 cells, using Nonvoltage-gated Sodium Channel 1 (Phospho-Thr615) Antibody. The picture on the right is blocked with the phospho peptide.



Immunohistochemistry analysis of paraffin-embedded human brain, using Nonvoltage-gated Sodium Channel 1 (Phospho-Thr615) Antibody. The picture on the right is blocked with the phospho peptide.



Western blot analysis of lysates from HeLa cells, using Nonvoltage-gated Sodium Channel 1 (Phospho-Thr615) Antibody. The lane on the right is blocked with the phospho peptide.