



## ENaC β (phospho Thr615) Polyclonal Antibody

Catalog No	BYab-16350
Isotype	lgG
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 $\beta$ (T615) Polyclonal Antibody detects endogenous levels of ENaC $\beta$ 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	≥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

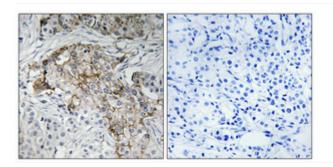
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	mineralocorticoid receptor. Autosomal recessive PHA1 is characterized by an often fulminant presentation in the neonatal period with dehydration, hyponatraemia, hyperkalaemia, metabolic acidosis, failure to thrive and weight loss.,disease:Defects 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. Negetive contrl (right) obtaned from antibody was pre-absorbed by immunogen peptide.

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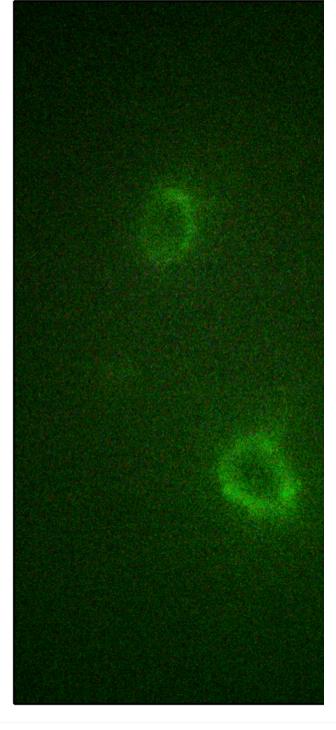


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Immunofluorescence analysis of COS7 cells, using Nonvoltage-gated Sodium Channel 1 (Phospho-Thr615) Antibody. The picture on the right is blocked with the phospho peptide.



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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.

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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.

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