



# ENaC $\beta$ (phospho Thr615) Monoclonal Antibody

<b>Catalog No</b>	BYmab-16350
<b>Isotype</b>	IgG
<b>Reactivity</b>	Human;Mouse;Rat
<b>Applications</b>	WB
<b>Gene Name</b>	SCNN1B
<b>Protein Name</b>	Amiloride-sensitive sodium channel subunit beta
<b>Immunogen</b>	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
<b>Specificity</b>	Phospho-ENaC $\beta$ (T615) Monoclonal Antibody detects endogenous levels of ENaC $\beta$ protein only when phosphorylated at T615.
<b>Formulation</b>	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
<b>Source</b>	Monoclonal, Mouse,IgG
<b>Purification</b>	The antibody was affinity-purified from mouse antiserum by affinity-chromatography using epitope-specific immunogen.
<b>Dilution</b>	WB 1:500-2000
<b>Concentration</b>	1 mg/ml
<b>Purity</b>	$\geq 90\%$
<b>Storage Stability</b>	-20°C/1 year
<b>Synonyms</b>	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
<b>Observed Band</b>	68kD
<b>Cell Pathway</b>	Apical cell membrane ; Multi-pass membrane protein . Cytoplasmic vesicle membrane . Apical membrane of epithelial cells. .
<b>Tissue Specificity</b>	Detected in placenta, lung and kidney (PubMed:7762608). Expressed in kidney (at protein level) (PubMed:22207244).
<b>Function</b>	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

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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],

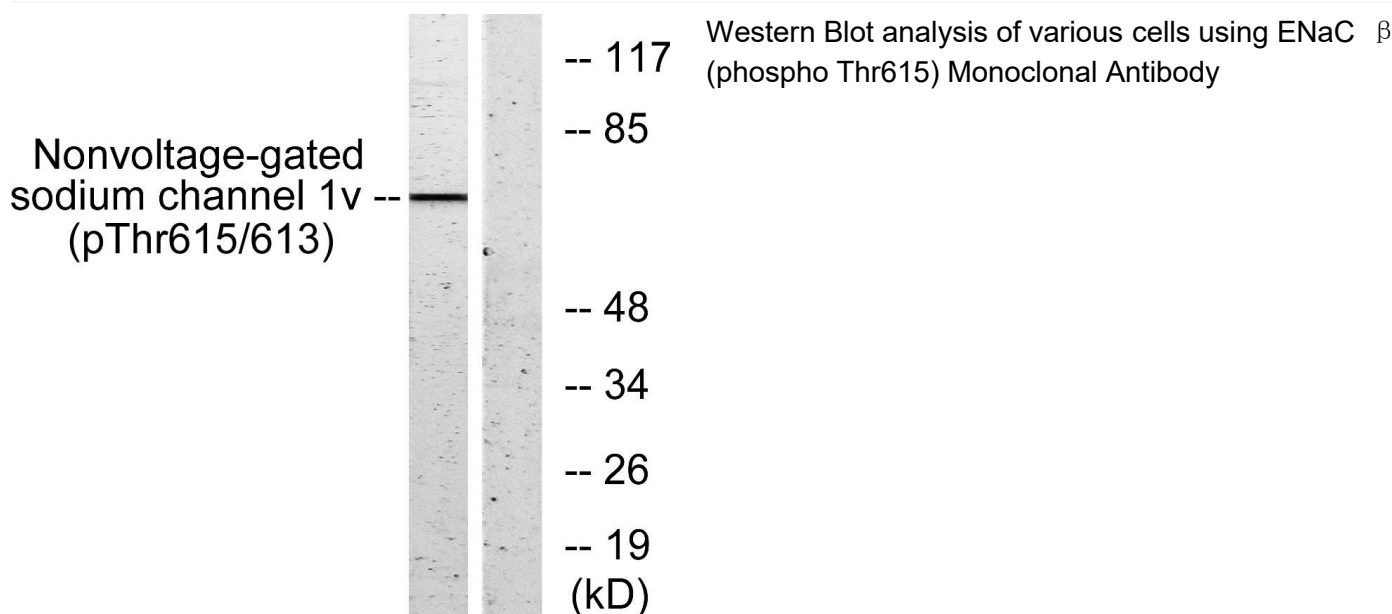
## matters needing attention

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



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