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Rabbit Anti-epithelial Sodium Channel gamma antibody

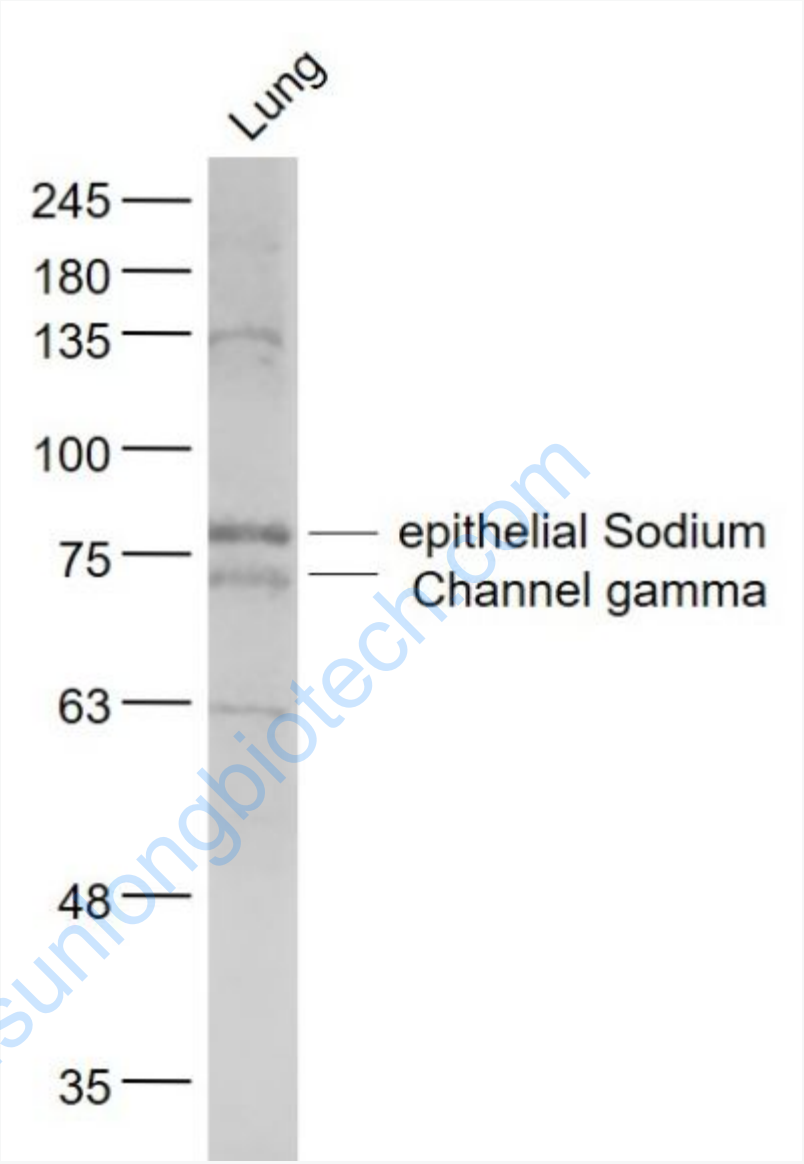
SL4263R

Product Name:	epithelial Sodium Channel gamma
Chinese Name:	上皮钠离子Channel proteinγ/γENaC抗体
Alias:	Amiloride sensitive epithelial sodium channel gamma subunit; Amiloride sensitive sodium channel subunit gamma; ENaC gamma subunit; ENaCg; ENaCgamma; Epithelial Na(+) channel subunit gamma; Epithelial Na+ channel subunit gamma; Gamma ENaC; Gamma NaCH; Nonvoltage gated sodium channel 1 subunit gamma; PHA 1; PHA1; SCNEG; SCNN 1G; SCNN1G; Sodium channel nonvoltage gated 1 gamma; SCNNG_HUMAN.
Organism Species:	Rabbit
Clonality:	Polyclonal
React Species:	Human,Mouse,Rat,Dog,Rabbit,
Applications:	WB=1:500-2000ELISA=1:500-1000IHC-P=1:400-800（Paraffin sections need antigen repair） not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Molecular weight:	71kDa
Cellular localization:	The cell membrane
Form:	Lyophilized or Liquid
Concentration:	1mg/ml
immunogen:	KLH conjugated synthetic peptide derived from human epithelial Sodium Channel gamma:188-290/649<Extracellular>
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:	Epithelial sodium channels are amiloride-sensitive members of the Degenerin/epithelial sodium channel (Deg/ENaC) superfamily of ion channels. Members of this superfamily of ion channels share organizational similarity in that they all possess two short intracellular amino and carboxyl termini, two short membrane spanning segments, and a large extracellular loop with a conserved cysteine-rich region. There are three homologous isoforms of the ENaC (alpha, beta, and gamma) protein. ENaC in the kidney, lung, and colon plays an essential role in trans-epithelial sodium and fluid balance. ENaC also mediates aldosterone-dependent sodium reabsorption in the distal nephron of the kidney, thus regulating blood pressure. ENaC is thought to be regulated, in part, through association with the cystic fibrosis transmembrane conductance regulator (CFTR) chloride ion channel. Gain-of-function mutations in beta- or gamma-ENaC can cause severe arterial hypertension (Liddel's syndrome) and loss-of-function mutations in alpha- or beta-ENaC causes pseudohypoaldosteronism (PHA-1). **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. **Subcellular Location:** Apical cell membrane; Multi-pass membrane protein. Note=Apical membrane of epithelial cells. **Post-translational modifications:** Phosphorylated on serine and threonine residues. Ubiquitinated; this targets individual subunits for endocytosis and proteasome-mediated degradation. **DISEASE:** Defects in SCNN1G are a cause of Liddle syndrome (LIDDS) [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. Defects in SCNN1G are the cause of bronchiectasis with or without elevated sweat chloride type 3 (BESC3) [MIM:613071]. 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. **Similarity:** Belongs to the amiloride-sensitive sodium channel (TC 1.A.6) family. SCNN1G subfamily. **SWISS:** P51170 **Gene ID:** 6340 **Database links:** Entrez Gene: 6340 Human Entrez Gene: 20278 Mouse Entrez Gene: 24768 Rat Omim: 600761 Human SwissProt: P51170 Human SwissProt: Q9WU39 Mouse SwissProt: Q28738 Rabbit SwissProt: P37091 Rat Unigene: 371727 Human Unigene: 35247 Mouse Unigene: 10360 Rat **Important Note:** This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

Picture:



Sample:

Lung (Mouse) Lysate at 40 ug

Primary: Anti- epithelial Sodium Channel gamma (SL4263R) at 1/1000 dilution

Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000 dilution

Predicted band size: 71 kD

Observed band size: 71/81 kD