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Goat Anti-SLC4A11 / BTR1 Antibody
Peptide-affinity purified goat antibody
Catalog # AF2212a

Specification

Goat Anti-SLC4A11 / BTR1 Antibody - Product Information

Application	**WB**
Primary Accession	Q8NBS3
Other Accession	NP_114423, 83959, 269356 (mouse), 311423 (rat)
Reactivity	**Human, Mouse, Rat**
Predicted	**Dog**
Host	**Goat**
Clonality	**Polyclonal**
Concentration	**100ug/200ul**
Isotype	**IgG**
Calculated MW	**98181**

Goat Anti-SLC4A11 / BTR1 Antibody - Additional Information

Gene ID 83959

Other Names
Sodium bicarbonate transporter-like protein 11, Bicarbonate transporter-related protein 1, Sodium borate cotransporter 1, NaBC1, Solute carrier family 4 member 11, SLC4A11, BTR1

Format
0.5 mg IgG/ml in Tris saline (20mM Tris pH7.3, 150mM NaCl), 0.02% sodium azide, with 0.5% bovine serum albumin

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions
Goat Anti-SLC4A11 / BTR1 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Goat Anti-SLC4A11 / BTR1 Antibody - Protein Information

Name SLC4A11

Synonyms BTR1

Function
Multifunctional transporter with an impact in cell morphology and differentiation. In the presence of borate B(OH)4(-), acts as a voltage-dependent electrogenic Na(+)-coupled B(OH)4(-)

cotransporter controlling boron homeostasis (PubMed:15525507). At early stages of stem cell differentiation, participates in synergy with ITGA5-ITGB1 and ITGAV-ITGB3 integrins and BMPR1A to promote cell adhesion and contractility that drives differentiation toward osteogenic commitment while inhibiting adipogenesis (By similarity). In the absence of B(OH)4(-), acts as a Na(+)-coupled OH(-) or H(+) permeable channel with implications in cellular redox balance (PubMed:15525507, PubMed:28642546). Regulates the oxidative stress response in corneal endothelium by enhancing antioxidant defenses and protecting cells from reactive oxygen species (PubMed:28642546). In response to hypo-osmotic challenge, also acts as a water permeable channel at the basolateral cell membrane of corneal endothelial cells and facilitates transendothelial fluid reabsorption in the aqueous humor (PubMed:23813972, PubMed:25007886, PubMed:31273259). In the presence of ammonia, acts as an electrogenic NH3/H(+) cotransporter and may play a role in ammonia transport and reabsorption in renal Henle's loop epithelium (PubMed:27581649).

Cellular Location
Cell membrane; Multi-pass membrane protein. Basolateral cell membrane; Multi-pass membrane protein

Tissue Location
Widely expressed. Highly expressed in kidney, testis, salivary gland, thyroid, trachea and corneal endothelium. Not detected in retina and lymphocytes. [Isoform 5]: The predominant isoform in corneal endothelium (at protein level).

Goat Anti-SLC4A11 / BTR1 Antibody - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

Goat Anti-SLC4A11 / BTR1 Antibody - Images

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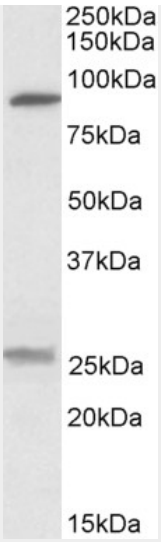

AF2212a (0.3 µg/ml) staining of Human Kidney lysate (35 µg protein in RIPA buffer). Primary incubation was 1 hour. Detected by chemiluminescence.

Goat Anti-SLC4A11 / BTR1 Antibody - Background

This gene encodes a voltage-regulated, electrogenic sodium-coupled borate cotransporter that is essential for borate homeostasis, cell growth and cell proliferation. Mutations in this gene have been associated with a number of endothelial corneal dystrophies including recessive corneal endothelial dystrophy 2, corneal dystrophy and perceptive deafness, and Fuchs endothelial corneal dystrophy. Multiple transcript variants encoding different isoforms have been described.

Goat Anti-SLC4A11 / BTR1 Antibody - References

Genetic analysis of patients with Fuchs endothelial corneal dystrophy in India. Hemadevi B, et al. BMC Ophthalmol, 2010 Feb 10. PMID 20144242.
Absence of phenotype-genotype correlation of patients expressing mutations in the SLC4A11 gene. Mehta JS, et al. Cornea, 2010 Mar. PMID 20118786.
Mutational spectrum of SLC4A11 in autosomal recessive CHED in Saudi Arabia. Aldahmesh MA, et al. Invest Ophthalmol Vis Sci, 2009 Sep. PMID 19369245.
Identification of mutations in the SLC4A11 gene in patients with recessive congenital hereditary endothelial dystrophy. Hemadevi B, et al. Arch Ophthalmol, 2008 May. PMID 18474783.
Mutation in the SLC4A11 gene associated with autosomal recessive congenital hereditary endothelial dystrophy in a large Saudi family. Shah SS, et al. Ophthalmic Genet, 2008 Mar. PMID 18363173.