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SGK Antibody (N-term) Blocking Peptide
Synthetic peptide
Catalog # BP7056a

Specification

SGK Antibody (N-term) Blocking Peptide - Product Information

Primary Accession	O00141
Other Accession	NP_005618

SGK Antibody (N-term) Blocking Peptide - Additional Information

Gene ID 6446

Other Names
Serine/threonine-protein kinase Sgk1, Serum/glucocorticoid-regulated kinase 1, SGK1, SGK

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP7056a was selected from the N-term region of human SGK . A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

SGK Antibody (N-term) Blocking Peptide - Protein Information

Name SGK1

Synonyms SGK

Function
Serine/threonine-protein kinase which is involved in the regulation of a wide variety of ion channels, membrane transporters, cellular enzymes, transcription factors, neuronal excitability, cell growth, proliferation, survival, migration and apoptosis. Plays an important role in cellular stress response. Contributes to regulation of renal Na(+) retention, renal K(+) elimination, salt appetite, gastric acid secretion, intestinal Na(+)/H(+) exchange and nutrient transport, insulin-dependent salt sensitivity of blood pressure, salt sensitivity of peripheral glucose uptake, cardiac repolarization and memory consolidation. Up-regulates Na(+) channels: SCNN1A/ENAC, SCN5A and ASIC1/ACCN2, K(+) channels: KCNJ1/ROMK1, KCNA1-5, KCNQ1-5 and KCNE1, epithelial Ca(2+) channels: TRPV5 and TRPV6, chloride channels: BSND, CLCN2 and CFTR, glutamate

transporters: SLC1A3/EAAT1, SLC1A2 /EAAT2, SLC1A1/EAAT3, SLC1A6/EAAT4 and SLC1A7/EAAT5, amino acid transporters: SLC1A5/ASCT2, SLC38A1/SN1 and SLC6A19, creatine transporter: SLC6A8, Na(+)/dicarboxylate cotransporter: SLC13A2/NADC1, Na(+)-dependent phosphate cotransporter: SLC34A2/NAPI-2B, glutamate receptor: GRIK2/GLUR6. Up-regulates carriers: SLC9A3/NHE3, SLC12A1/NKCC2, SLC12A3/NCC, SLC5A3/SMIT, SLC2A1/GLUT1, SLC5A1/SGLT1 and SLC15A2/PEPT2. Regulates enzymes: GSK3A/B, PMM2 and Na(+)/K(+) ATPase, and transcription factors: CTNNB1 and nuclear factor NF-kappa-B. Stimulates sodium transport into epithelial cells by enhancing the stability and expression of SCNN1A/ENAC. This is achieved by phosphorylating the NEDD4L ubiquitin E3 ligase, promoting its interaction with 14-3-3 proteins, thereby preventing it from binding to SCNN1A/ENAC and targeting it for degradation. Regulates store-operated Ca(+2) entry (SOCE) by stimulating ORAI1 and STIM1. Regulates KCNJ1/ROMK1 directly via its phosphorylation or indirectly via increased interaction with SLC9A3R2/NHERF2. Phosphorylates MDM2 and activates MDM2-dependent ubiquitination of p53/TP53. Phosphorylates MAPT/TAU and mediates microtubule depolymerization and neurite formation in hippocampal neurons. Phosphorylates SLC2A4/GLUT4 and up- regulates its activity. Phosphorylates APBB1/FE65 and promotes its localization to the nucleus. Phosphorylates MAPK1/ERK2 and activates it by enhancing its interaction with MAP2K1/MEK1 and MAP2K2/MEK2. Phosphorylates FBXW7 and plays an inhibitory role in the NOTCH1 signaling. Phosphorylates FOXO1 resulting in its relocalization from the nucleus to the cytoplasm. Phosphorylates FOXO3, promoting its exit from the nucleus and interference with FOXO3-dependent transcription. Phosphorylates BRAF and MAP3K3/MEKK3 and inhibits their activity. Phosphorylates SLC9A3/NHE3 in response to dexamethasone, resulting in its activation and increased localization at the cell membrane. Phosphorylates CREB1. Necessary for vascular remodeling during angiogenesis. Sustained high levels and activity may contribute to conditions such as hypertension and diabetic nephropathy. Isoform 2 exhibited a greater effect on cell plasma membrane expression of SCNN1A/ENAC and Na(+) transport than isoform 1.

Cellular Location
Cytoplasm. Nucleus. Endoplasmic reticulum membrane. Cell membrane. Mitochondrion. Note=The subcellular localization is controlled by the cell cycle, as well as by exposure to specific hormones and environmental stress stimuli. In proliferating cells, it shuttles between the nucleus and cytoplasm in synchrony with the cell cycle, and in serum/growth factor-stimulated cells it resides in the nucleus. In contrast, after exposure to environmental stress or treatment with glucocorticoids, it is detected in the cytoplasm and with certain stress conditions is associated with the mitochondria. In osmoregulation through the epithelial sodium channel, it can be localized to the cytoplasmic surface of the cell membrane. Nuclear, upon phosphorylation

Tissue Location
Expressed in most tissues with highest levels in the pancreas, followed by placenta, kidney and lung. Isoform 2 is strongly expressed in brain and pancreas, weaker in heart, placenta, lung, liver and skeletal muscle.

SGK Antibody (N-term) Blocking Peptide - Protocols

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

- Blocking Peptides

SGK Antibody (N-term) Blocking Peptide - Images

SGK Antibody (N-term) Blocking Peptide - Background

SGK is a serine/threonine protein kinase that is highly similar to the rat serum-and glucocorticoid-induced protein kinase (SGK). This gene was identified in a screen of hepatocellular genes regulated in response to cellular hydration or swelling. Cellular hydration is a catabolic signal, stimulating glycogenolysis and proteolysis, and inhibiting protein and glycogen synthesis. This kinase has been shown to be important in activating certain potassium, sodium, and chloride channels. Expression of this gene in hepatocytes is stimulated by transforming growth factor-beta

(TGF-beta) which participates in the pathophysiology of diabetic complications. Since both TGF-beta expression and SGK expression are elevated in diabetic nephropathy, this suggests an involvement of SGK in the development of this condition.

SGK Antibody (N-term) Blocking Peptide - References

Hong, G., et al., FASEB J. 17(13):1966-1968 (2003).Boehmer, C., et al., Biochem. Biophys. Res. Commun. 306(1):156-162 (2003).Embark, H.M., et al., Pflugers Arch. 445(5):601-606 (2003).Farman, N., et al., J. Clin. Invest. 110(9):1233-1234 (2002).Yun, C.C., et al., J. Am. Soc. Nephrol. 13(12):2823-2830 (2002).