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BRSK1 Antibody (C-term) Blocking Peptide

Synthetic peptide
Catalog # BP8168b

Specification

BRSK1 Antibody (C-term) Blocking Peptide - Product Information

Primary Accession	Q8TDC3
Other Accession	NP_115806

BRSK1 Antibody (C-term) Blocking Peptide - Additional Information

Gene ID 84446

Other Names
Serine/threonine-protein kinase BRSK1, Brain-selective kinase 1, Brain-specific serine/threonine-protein kinase 1, BR serine/threonine-protein kinase 1, Serine/threonine-protein kinase SAD-B, Synapses of Amphids Defective homolog 1, SAD1 homolog, hSAD1, BRSK1, KIAA1811, SAD1, SADB

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP8168b was selected from the C-term region of human KIAA1811. 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.

BRSK1 Antibody (C-term) Blocking Peptide - Protein Information

Name BRSK1

Synonyms KIAA1811, SAD1, SADB

Function
Serine/threonine-protein kinase that plays a key role in polarization of neurons and centrosome duplication. Phosphorylates CDC25B, CDC25C, MAPT/TAU, RIMS1, TUBG1, TUBG2 and WEE1. Following phosphorylation and activation by STK11/LKB1, acts as a key regulator of polarization of cortical neurons, probably by mediating phosphorylation of microtubule-associated proteins such as MAPT/TAU at 'Thr-529' and 'Ser-579'. Also regulates neuron polarization by mediating phosphorylation of WEE1 at 'Ser-642' in postmitotic neurons, leading to down-regulate WEE1

activity in polarized neurons. In neurons, localizes to synaptic vesicles and plays a role in neurotransmitter release, possibly by phosphorylating RIMS1. Also acts as a positive regulator of centrosome duplication by mediating phosphorylation of gamma-tubulin (TUBG1 and TUBG2) at 'Ser-131', leading to translocation of gamma-tubulin and its associated proteins to the centrosome. Involved in the UV-induced DNA damage checkpoint response, probably by inhibiting CDK1 activity through phosphorylation and activation of WEE1, and inhibition of CDC25B and CDC25C.

Cellular Location
Cytoplasm. Nucleus. Cytoplasm, cytoskeleton, microtubule organizing center, centrosome. Synapse {ECO:0000250|UniProtKB:B2DD29}. Presynaptic active zone {ECO:0000250|UniProtKB:B2DD29}. Cytoplasmic vesicle, secretory vesicle, synaptic vesicle {ECO:0000250|UniProtKB:B2DD29}. Note=Nuclear in the absence of DNA damage. Translocated to the nucleus in response to UV- or MMS-induced DNA damage (By similarity).

Tissue Location
Widely expressed, with highest levels in brain and testis. Protein levels remain constant throughout the cell cycle

BRSK1 Antibody (C-term) Blocking Peptide - Protocols

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

- Blocking Peptides

BRSK1 Antibody (C-term) Blocking Peptide - Images

BRSK1 Antibody (C-term) Blocking Peptide - Background

BRSK1 may be involved as a checkpoint kinase in the regulation of G2/M arrest in response to UV- or methyl methane sulfonate (MMS)-induced, but not IR-induced, DNA damage. This protein phosphorylates WEE1 and CDC25B in vitro and CDC25C in vitro and in vivo. BRSK1 is partitioned between cytoplasmic and nuclear locations in the absence of DNA damage, but translocates to the nucleus in response to Uv- or MMS-induced DNA damage. BRSK1 shares significant homology with the fission yeast Cdr2, a mitosis-regulatory kinase, and Caenorhabditis elegans SAD1, a neuronal cell polarity regulator. The BRSK1 transcript is expressed ubiquitously with the highest levels of expression in brain and testis.

BRSK1 Antibody (C-term) Blocking Peptide - References

Lu, R., et al., J. Biol. Chem. 279(30):31164-31170 (2004).Lizcano, J.M., et al., EMBO J. 23(4):833-843 (2004).