

**STK29 Antibody (C-term) Blocking Peptide**  
**Synthetic peptide**  
**Catalog # BP7191b****Specification**

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**STK29 Antibody (C-term) Blocking Peptide - Product Information**Primary Accession [Q8IWQ3](#)**STK29 Antibody (C-term) Blocking Peptide - Additional Information****Gene ID** 9024**Other Names**

Serine/threonine-protein kinase BRSK2, Brain-selective kinase 2, Brain-specific serine/threonine-protein kinase 2, BR serine/threonine-protein kinase 2, Serine/threonine-protein kinase 29, Serine/threonine-protein kinase SAD-A, BRSK2, C11orf7, PEN11B, SADA, STK29

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP7191b](/product/products/AP7191b) was selected from the C-term region of human STK29. 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.

**STK29 Antibody (C-term) Blocking Peptide - Protein Information****Name** BRSK2**Synonyms** C11orf7, PEN11B, SADA, STK29**Function**

Serine/threonine-protein kinase that plays a key role in polarization of neurons and axonogenesis, cell cycle progress and insulin secretion. Phosphorylates CDK16, CDC25C, MAPT/TAU, PAK1 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. Plays a role in the regulation of the mitotic cell cycle progress and the onset of mitosis. Plays a role in the regulation of insulin secretion in response to elevated

glucose levels, probably via phosphorylation of CDK16 and PAK1. While BRSK2 phosphorylated at Thr- 174 can inhibit insulin secretion (PubMed:<a href="http://www.uniprot.org/citations/22798068" target="\_blank">22798068</a>), BRSK2 phosphorylated at Thr-260 can promote insulin secretion (PubMed:<a href="http://www.uniprot.org/citations/22669945" target="\_blank">22669945</a>). Regulates reorganization of the actin cytoskeleton. May play a role in the apoptotic response triggered by endoplasmic reticulum (ER) stress.

#### **Cellular Location**

Cytoplasm, cytoskeleton, microtubule organizing center, centrosome. Cytoplasm, perinuclear region. Endoplasmic reticulum. Note=Detected at centrosomes during mitosis. Localizes to the endoplasmic reticulum in response to stress caused by tunicamycin

#### **Tissue Location**

Detected in pancreas islets (at protein level).

### **STK29 Antibody (C-term) Blocking Peptide - Protocols**

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

- [Blocking Peptides](#)

### **STK29 Antibody (C-term) Blocking Peptide - Images**

### **STK29 Antibody (C-term) Blocking Peptide - Background**

BRSK2 expressed in insect cells specifically phosphorylates WEE1A, CDC25C, and CDC25B in an in vitro assay, but a kinase-dead mutant does not. Overexpression of BRSK2 in HeLa cells results in increased phosphorylation of CDC25C. DNA damage induced by ultraviolet (UV) irradiation or methyl methane sulfonate, but not by ionizing radiation, enhances endogenous BRSK2 kinase activity in a caffeine-sensitive manner and causes translocation of BRSK2 from the cytoplasm to the nucleus. Overexpression of BRSK2 induces G2/M arrest in HeLa cells. Small interfering RNA against BRSK2 partly abrogates UV-induced G2/M arrest. BRSK2 may act as a checkpoint kinase upon DNA damage induced by UV irradiation or methyl methane sulfonate.

### **STK29 Antibody (C-term) Blocking Peptide - References**

J. Biol. Chem. 279: 31164-31170, 2004. J. Hum. Genet. 44:1-9(1999).J. Hum. Genet. 43: 283-284, 1998.