SANTA CRUZ BIOTECHNOLOGY, INC.

p-CaMKIV (Thr 196)-R: sc-28443-R

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

The Ca^{2+}/calmodulin-dependent protein kinases (CaM kinases) comprise a structurally related subfamily of serine/threonine kinases which include CaMKI, CaMKII and CaMKIV. CaMKII is a ubiquitously expressed serine/threonine protein kinase that is activated by Ca^{2+} and calmodulin (CaM) and has been implicated in regulation of the cell cycle and transcription. There are four CaMKII isozymes designated α, β, γ and δ which may or may not be co-expressed in the same tissue types. CaMKIV is stimulated by Ca^{2+} and CaM but also requires phosphorylation on threonine 196 by a CaMK for full activation. Stimulation of the T cell receptor CD3 signaling complex with an anti-CD3 monoclonal antibody leads to a 10-40 fold increase in CaMKIV activity. An additional kinase, CaMKK, functions to activate CaMKI through the specific phosphorylation of the regulatory threonine residue at position 177.

REFERENCES

1. Tombes, R.M., et al. 1995. G_1 cell cycle arrest apoptosis are induced in NIH/3T3 cells by KN-93, an inhibitor of CaMK-II (the multifunctional Ca^{2+}/CaM kinase). Cell Growth Differ. 6: 1063-1070.
2. Hama, N., et al. 1995. Calcium/calmodulin-dependent protein kinase II downregulates both calcineurin and protein kinase c-mediated pathways for cytokine gene transcription in human T cells. J. Exp. Med. 181: 1217-1222.
3. Baltas, L.G., et al. 1995. The cardiac sarcoplasmic reticulum phospholamban kinase is a distinct δ-CaM kinase isozyme. FEBS Lett. 373: 71-75.
4. Tokumitsu, H., et al. 1995. Characterization of a CaM-kinase cascade: molecular cloning and expression of calcium/calmodulin-dependent protein kinase kinase. J. Biol. Chem. 270: 19320-19324.
5. Park, I.K., et al. 1995. Activation of Ca^{2+}/calmodulin-dependent protein kinase (CaM-kinase) IV by CaM-kinase kinase in Jurkat T lymphocytes. J. Biol. Chem. 270: 30464-30469.

CHROMOSOMAL LOCATION

Genetic locus: CAMK4 (human) mapping to 5q22.1; Camk4 (mouse) mapping to 18 B1.

SOURCE

p-CaMKIV (Thr 196)-R is a rabbit polyclonal antibody raised against a short amino acid sequence containing Thr 196 phosphorylated CaMKIV of mouse origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-28443 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

APPLICATIONS

p-CaMKIV (Thr 196)-R is recommended for detection of Thr 196 phosphorylated CaMKIV of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

p-CaMKIV (Thr 196)-R is also recommended for detection of correspondingly phosphorylated CaMKIV in additional species, including equine, canine, bovine, porcine and avian.

Suitable for use as control antibody for CaMKIV siRNA (h): sc-29902, CaMKIV siRNA (m): sc-29903, CaMKIV shRNA Plasmid (h): sc-29902-SH, CaMKIV shRNA Plasmid (m): sc-29903-SH, CaMKIV shRNA (h) Lentiviral Particles: sc-29902-V and CaMKIV shRNA (m) Lentiviral Particles: sc-29903-V.

Molecular Weight of p-CaMKIV: 60 kDa.

Positive Controls: Jurkat whole cell lysate: sc-2204 or A-431 whole cell lysate: sc-2201.

SELECT PRODUCT CITATIONS

1. Tai, Y., et al. 2008. TRPC6 channels promote dendritic growth via the CaMKIV-CREB pathway. J. Cell Sci. 121: 2301-2307.
2. Kong, H., et al. 2009. Requirement of AQP4 for antidepressive efficiency of fluoxetine: implication in adult hippocampal neurogenesis. Neuropsychopharmacology 34: 1263-1276.
3. Xu, J., et al. 2010. Memory impairment in cognitively impaired aged rats associated with decreased hippocampal CREB phosphorylation: reversal by procyanidins extracted from the lotus seedpod. J. Gerontol. A Biol. Sci. Med. Sci. 65: 933-940.
4. Aonurm-Helm, A., et al. 2010. NCAM-mimetic, FGL peptide, restores disrupted fibroblast growth factor receptor (FGFR) phosphorylation and FGFR mediated signaling in neural cell adhesion molecule (NCAM)-deficient mice. Brain Res. 1309: 1-8.
5. Zheng, Z., et al. 2010. Oligomeric amyloid-β inhibits the proteolytic conversion of brain-derived neurotrophic factor (BDNF), AMPA receptor trafficking, and classical conditioning. J. Biol. Chem. 285: 34708-34717.
6. Song, B., et al. 2010. Inhibitory phosphorylation of GSK-3 by CaMKII couples depolarization to neuronal survival. J. Biol. Chem. 285: 41122-41134.
7. Tinsley, C.J., et al. 2011. A role for the CAMKK pathway in visual object recognition memory. Hippocampus 22: 466-476.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.