SANTA CRUZ BIOTECHNOLOGY, INC.

p-CaMKIIα (Thr 286)-R: sc-12886-R

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 an 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 type. CaMKIIα is autophosphorylated on Thr 286 upon the binding of the Ca^{2+}/CaM complex to the autoinhibitory domain of CaMKII. This process is called Ca^{2+}/CaM trapping, which is thought to be involved in the synaptic encoding of information.

CHROMOSOMAL LOCATION

Genetic locus: CAMK2A (human) mapping to 5q32; Camk2a (mouse) mapping to 18 E1.

SOURCE

p-CaMKIIα (Thr 286)-R is is a rabbit polyclonal antibody raised against a short amino acid sequence containing phosphorylated Thr 286 of CaMKIIα of human 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-12886 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

p-CaMKIIα (Thr 286)-R is recommended for detection of Thr 286 phosphorylated CaMKIIα of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), immunohistochemistry (including paraffin-embedded sections) (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-CaMKIIα (Thr 286)-R is also recommended for detection of correspondingly phosphorylated Thr on CaMKIIα in additional species, including equine, canine, bovine, porcine and avian.

Suitable for use as control antibody for CaMKIIα siRNA (h): sc-29900, CaMKIIα siRNA (m): sc-29901, CaMKIIα shRNA Plasmid (h): sc-29900-SH, CaMKIIα shRNA Plasmid (m): sc-29901-SH, CaMKIIα shRNA (h) Lentiviral Particles: sc-29900-V and CaMKIIα shRNA (m) Lentiviral Particles: sc-29901-V.

Molecular Weight of p-CaMKIIα: 50 kDa.

Positive Controls: mouse brain extract: sc-2253 or human lung tumor.

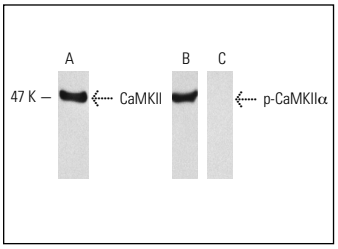
STORAGE

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

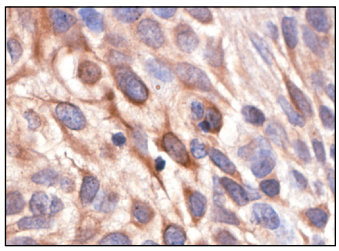
RESEARCH USE

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

DATA



Western blot detection of CaMKII phosphorylation in mouse brain extracts. Blots were probed with CaMKII (M-176): sc-9035 (**A**) and p-CaMKIIα (Thr 286)-R: sc-12886-R (**B**,**C**). Antibody was preincubated with cognate non-phosphorylated (**B**) or phosphorylated (**C**) peptide.



p-CaMKIIα (Thr 286)-R: sc-12886-R. Immunoperoxidase staining of formalin-fixed, paraffin-embedded human lung tumor showing cytoplasmic staining.

SELECT PRODUCT CITATIONS

1. Fatima, S., et al. 2003. CaM kinase IIα mediates norepinephrine-induced translocation of cytosolic phospholipase A2 to the nuclear envelope. J. Cell Sci. 116: 353-365.
2. Rashid, A.J., et al. 2007. D_1-D_2 Dopamine receptor heterooligomers with unique pharmacology are coupled to rapid activation of $G_{q/11}$ in the striatum. Proc. Natl. Acad. Sci. USA 104: 654-659.
3. Tai, Y., et al. 2008. TRPC6 channels promote dendritic growth via the CaMKIV-CREB pathway. J. Cell Sci. 121: 2301-2307.
4. Najdi, R., et al. 2009. A Wnt kinase network alters nuclear localization of TCF-1 in colon cancer. Oncogene 28: 4133-4146.
5. Liraz, O., et al. 2009. CAMKII activation is not required for maintenance of learning-induced enhancement of neuronal excitability. PLoS ONE 4: e4289.
6. Kojundzic, S.L., et al. 2010. Depression of Ca^{2+}/calmodulin-dependent protein kinase II in dorsal root ganglion neurons after spinal nerve ligation. J. Comp. Neurol. 518: 64-74.
7. Gomez-Pinilla, F., et al. 2011. Exercise impacts brain-derived neurotrophic factor plasticity by engaging mechanisms of epigenetic regulation. Eur. J. Neurosci. 33: 383-390.
8. Blenn, C., et al. 2011. Poly(ADP-ribose)glycohydrolase is an upstream regulator of Ca^{2+} fluxes in oxidative cell death. Cell. Mol. Life Sci. 68: 1455-1466.