

Anti-CaM Kinase II (Thr286) Antibody

Our Anti-CaM Kinase II (Thr286) rabbit polyclonal phosphospecific primary antibody from PhosphoSolut
Catalog # AN1325

Specification**Anti-CaM Kinase II (Thr286) Antibody - Product Information**

Application	WB
Primary Accession	P11275
Reactivity	Bovine, Chicken
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Calculated MW	54115

Anti-CaM Kinase II (Thr286) Antibody - Additional Information

Gene ID **25400**

Other Names

Calcium/calmodulin dependent protein kinase II alpha antibody, Calcium/calmodulin dependent protein kinase II beta antibody, Calcium/calmodulin dependent protein kinase II delta antibody, Calcium/calmodulin dependent protein kinase II gamma antibody, Calcium/calmodulin-dependent protein kinase type II subunit alpha antibody, CaM kinase II alpha antibody, CaM kinase II antibody, CaM kinase II beta antibody, CaM kinase II delta antibody, CaM kinase II gamma antibody, CaM kinase II subunit alpha antibody, CaMK-II subunit alpha antibody, CAMK2 antibody, Camk2a antibody, CAMK2B antibody, CAMK2D antibody, CAMK2G antibody, CAMKA antibody, KCC2A_HUMAN antibody

Target/Specificity

Ca²⁺/calmodulin-dependent protein kinase II (CaM Kinase II) is a multi-functional calcium and calmodulin-dependent protein kinase that mediates cellular responses to a wide variety of intercellular signals (Kennedy, 1998; Schulman and Hanson, 1993). CaM Kinase II has been shown to regulate diverse cellular functions including synaptic plasticity, neurotransmitter synthesis and release, gene expression, ion channel function, carbohydrate metabolism, cytoskeletal function, and Ca²⁺-homeostasis (Gleason et al., 2003; Soderling, 2000; Hudmon and Schulman, 2002). Phosphorylation of Thr-286 on the kinase produces an autonomously active form of CaM Kinase II (Meng et al., 2003; Picciotto et al., 1993). Autophosphorylation of Thr-305 inhibits the activity CaM Kinase II. Phosphorylation at this site appears to reduce the association of CaM Kinase II with the PSD and reduce LTP and learning (Elgersma et al., 2002).

Dilution

WB~~1:1000

Format

Antigen Affinity Purified from Pooled Serum

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

Anti-CaM Kinase II (Thr286) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

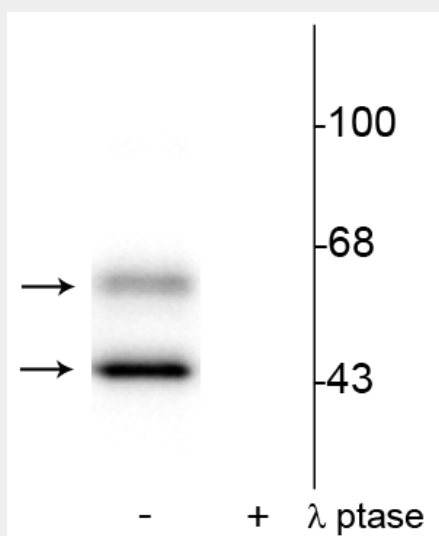
Shipping

Blue Ice

Anti-CaM Kinase II (Thr286) Antibody - Protocols

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

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

Anti-CaM Kinase II (Thr286) Antibody - Images

Western blot of rat brain lysate showing specific immunolabeling of the ~50 kDa α - and the ~60 kDa β -CaM Kinase II phosphorylated at Thr286 in the first lane (-). Phosphospecificity is shown in the second lane (+) where the immunolabeling is completely eliminated by blot treatment with lambda phosphatase (λ -Ptase, 1200 units for 30 minutes).

Anti-CaM Kinase II (Thr286) Antibody - Background

Ca²⁺/calmodulin-dependent protein kinase II (CaM Kinase II) is a multi-functional calcium and calmodulin-dependent protein kinase that mediates cellular responses to a wide variety of intercellular signals (Kennedy, 1998; Schulman and Hanson, 1993). CaM Kinase II has been shown to regulate diverse cellular functions including synaptic plasticity, neurotransmitter synthesis and release, gene expression, ion channel function, carbohydrate metabolism, cytoskeletal function, and Ca²⁺-homeostasis (Gleason et al., 2003; Soderling, 2000; Hudmon and Schulman, 2002). Phosphorylation of Thr-286 on the kinase produces an autonomously active form of CaM Kinase II (Meng et al., 2003; Picciotto et al., 1993). Autophosphorylation of Thr-305 inhibits the activity CaM

Kinase II. Phosphorylation at this site appears to reduce the association of CaM Kinase II with the PSD and reduce LTP and learning (Elgersma et al., 2002).