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CAMK1G, Active recombinant protein

CAMK1g, Calcium/calmodulin-dependent protein kinase type I gamma
Catalog # PBV11311r

Specification

CAMK1G, Active recombinant protein - Product info

Primary Accession	Q96NX5
Concentration	0.1
Calculated MW	60.0 kDa KDa

CAMK1G, Active recombinant protein - Additional Info

Gene ID	57172
Gene Symbol	CAMK1G

Other Names
CAMK1g, Calcium/calmodulin-dependent protein kinase type I gamma, CaMK-like CREB kinase III

Source	Baculovirus (Sf9 insect cells)
Assay&Purity	SDS-PAGE; ≥90%
Assay2&Purity2	HPLC;
Recombinant	Yes

Format
Liquid

Storage
-80°C; Recombinant protein in storage buffer (50 mM Tris-HCl, pH 7.5, 150 mM NaCl, 0.25 mM DTT, 0.1 mM EGTA, 0.1 mM EDTA, 0.1 mM PMSF, 25% glycerol).

CAMK1G, Active recombinant protein - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

CAMK1G, Active recombinant protein - Images

CAMK1G, Active recombinant protein - Background

CLICK-III/CaMKIG is a novel membrane-anchored neuronal Ca2+/calmodulin-dependent protein kinase (CaMK), an isoform of the CaMKI family with an extended C-terminal domain ending with CAAX motif (where AA is aliphatic acid). As expected from the similarity of its kinase domain with the other CaMKI isoforms, full activation of CLICK-III/CaMKIG required both Ca(2+)/CaM and

phosphorylation by CaMKK. Ca(2+)/cAMP-response element-binding protein (CREB) was a good substrate for CLICK-III/CaMKIG, at least in vitro. Interestingly enough, CLICK-III/CaMKIG transcripts were most abundant in neurons, with the highest levels in limited nuclei such as the central nucleus of the amygdala (CeA) and the ventromedial hypothalamus. Consistent with the presence of the CAAX motif, CLICK-III/CaMKIG was found to be anchored to various membrane compartments, especially to Golgi and plasma membranes. Both point mutation in the CAAX motif and treatment with compactin, a HMG-CoA reductase inhibitor, disrupted such membrane localization, suggesting that membrane localization of CLICK-III/CaMKIG occurred in a prenylation-dependent way. These findings provide a novel mechanism by which neuronal CaMK activity could be targeted to specific membrane compartments.