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Recombinant Human CAMK1G/CaMKI gamma Protein (His & GST Tag)

Catalog No. PKSH030332

Note: Centrifuge before opening to ensure complete recovery of vial contents.

Description	
Synonyms	CLICK3;CLICKIII;dJ272L16.1;RP1-272L16.2;VWS1
Species	Human
Expression Host	Baculovirus-Insect Cells
Sequence	Met 1-Met 476
Accession	Q96NX5-1
Calculated Molecular Weight	81.0 kDa
Observed molecular weight	75 kDa
Tag	N-His-GST
Bioactivity	Not validated for activity
Properties	
Purity	> 85 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per μ g of the protein as determined by the LAL method.
Storage	Store at $< -20^{\circ}$ C, stable for 6 months. Please minimize freeze-thaw cycles.
Shipping	This product is provided as liquid. It is shipped at frozen temperature with blue ice/gel packs. Upon receipt, store it immediately at $< -20^{\circ}$ C.
Formulation	Supplied as sterile solution of 50mM Tris, 100mM NaCl, pH 8.0, 20% glycerol, 0.3mM DTT
Reconstitution	Not Applicable
Data	

KDa	MK	R
116		
66.2	-	
45.0	-	
35.0	-	
25.0	-	
18.4 14.4	=	

> 85 % as determined by reducing SDS-PAGE.

Background

Calmodulin-Dependent Protein Kinase (CaM Kinase) is a kind of protein phosphorylate multiple downstream targets. Concentration of cytosolic calcium functions as a second messenger that mediates a wide range of cellular responses. Calcium binds to calcium binding proteins (calmodulin/CaM) and stimulates the activity of a variety of enzymes, including CaM kinases referred to as CaM-kinases (CaMKs), such as CaMKI, CaMKII, CaMKIV and CaMKK. Calmodulin-dependent protein kinase CL3/CaMKIY is a memberane-anchored CaMK belonging to the CaM kinase

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family. Its C-terminal region is uniquely modified by two sequential lipidification steps: prenylation followed by a kinaseactivity-regulated palmitoylation. These modifications are essential for CaMKIγ membrane anchoring and targeting into detergent-resistant lipid microdomains in the dendrites. It has been found that CaMKIγ critically contributed to BDNFstimulated dendritic growth. Raft insertion of CaMKIγ specifically promoted dendritogenesis of cortical neurons by acting upstream of RacGEF STEF and Rac, both present in lipid rafts. Thus, CaMKIγ may represent a key element in the Ca2+-dependent and lipid-raft-delineated switch that turns on extrinsic activity-regulated dendrite formation in developing cortical neurons.

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