

Recombinant Human CAMK1D Protein (GST Tag)

Catalog Number: PKSH033740

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

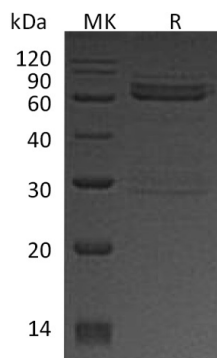
Description

Species	Human
Source	E.coli-derived Human CAMK1D protein Met1-Lys385, with an N-terminal GST
Calculated MW	69.2 kDa
Observed MW	60-70 kDa
Accession	Q8IU85
Bio-activity	Not validated for activity

Properties

Purity	> 90 % as determined by reducing SDS-PAGE.
Concentration	Subject to label value.
Endotoxin	< 1.0 EU per µg of the protein as determined by the LAL method.
Storage	Store at < -20°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°C.
Formulation	Supplied as a 0.2 µm filtered solution of 20mM PB, 150mM NaCl, pH 7.4.

Data



> 90 % as determined by reducing SDS-PAGE.

Background

For Research Use Only

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Calcium/calmodulin-dependent protein kinase or CaM kinases are serine/threonine-specific protein kinases that are primarily regulated by the Calcium/calmodulin complex. These kinases show a memory effect on activation. CaM kinases activity can outlast the intracellular calcium transient that is needed to activate it. In neurons, this property is important for the induction of synaptic plasticity. Pharmacological inhibition of CaM kinases II blocks the induction of long-term potentiation. Upon activation, CaM kinases II phosphorylates postsynaptic glutamate receptors and changes the electrical properties of the synapse. Calcium/calmodulin-dependent protein kinase type 1D, also known as CaM kinase I delta, CaM kinase ID, CaMKI-like protein kinase, CKLIK and CAMK1D, is a member of the protein kinase superfamily and CaMK subfamily. It contains one protein kinase domain. CAMK1D is broadly expressed. It is highly and mostly expressed in polymorphonuclear leukocytes (neutrophilic and eosinophilic granulocytes) while little or no expression is observed in monocytes and lymphocytes. Engineered overexpression of CAMK1D in non-tumorigenic breast epithelial cells led to increased cell proliferation, and molecular and phenotypic alterations indicative of epithelial-mesenchymal transition (EMT), including loss of cell-cell adhesions and increased cell migration and invasion. CAMK1D is a potential therapeutic target with particular relevance to clinically unfavorable basal-like tumors.