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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 MW69.2 kDaObserved MW60-70 kDaAccessionQ8IU85

Bio-activity Not validated for activity

Properties

Purity > 90 % as determined by reducing SDS-PAGE.

Concentration Subject to label value.

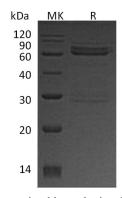
Endotoxin $\leq 1.0 \text{ EU per } \mu \text{g of the protein as determined by the LAL method.}$ **Storage** Storage Stor

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

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Calcium/calmodulin-dependent protein kinase or CaM kinases are serine/threonine-specific protein kinasesthat 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. Inneurons, this property is important for the induction of synaptic plasticity. Pharmacological inhibition of CaM kinases II blocks the induction oflong-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 CAMKID, is a member of the protein kinase superfamily and CaMK subfamily. It contains one protein kinase domain. CAMKID 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 CAMKID 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. CAMKID is a potential therapeutic target with particular relevance to clinically unfavorable basal-like tumors.