Recombinant Human 14-3-3 tau/14-3-3 theta/YWHAQ Protein (GST Tag)

Catalog Number: PKSH031392

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

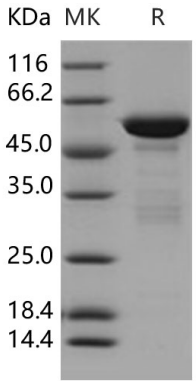
Description

Species	Human
Source	E.coli-derived Human 14-3-3 tau/14-3-3 theta/YWHAQ protein Met 1-Asn 245, with an N-terminal GST
Calculated MW	54.6 kDa
Observed MW	53 kDa
Accession	P27348
Bio-activity	Not validated for activity

Properties

Purity	> 88 % as determined by reducing SDS-PAGE.
Endotoxin	Please contact us for more information.
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80 ℃. Reconstituted protein solution can be stored at 4-8℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from sterile 20mM Tris, 0.15m NaCl, 20mM GSH, pH 7.5 Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
Reconstitution	Please refer to the printed manual for detailed information.

Data



> 88 % as determined by reducing SDS-PAGE.

Background

Gprotein-coupled receptor kinase 5, also known as Gprotein-coupled receptor kinase GRK5 and GRK5, is a member of the protein kinase superfamily, AGC Ser/Thr protein kinase family and GPRK subfamily. GRKs specifically phosphorylate agonist-occupied Gprotein-coupled receptors at the inner surface of the plasma membrane (PM), leading to receptor desensitization. GRKs utilize a variety of mechanisms to bind tightly, and sometimes reversibly, to cellular membranes. GRKs play an important role in mediating agonist-specific desensitization of numerous Gprotein-coupled receptors. GRK5 contains oneAGC-kinase C-terminal domain, oneprotein kinase domain and oneRGS domain. GRK5 specifically phosphorylates the activated forms of Gprotein-coupled receptors. Phospholipid-stimulated autophosphorylation may represent a novel mechanism for membrane association and regulation of GRK5 activity. GRK5 deficiency significantly exaggerates microgliosis and astrogliosis in the presence of an inflammatory initiator, such as the excess fibrillar Abeta and the subsequent active inflammatory reactions. GRK5 deficiency has been linked to early Alzheimer's disease in humans and mouse models of the disease.

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