Recombinant Human GRK5/GPRK5 Protein (His Tag)

Catalog Number: PKSH031399

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

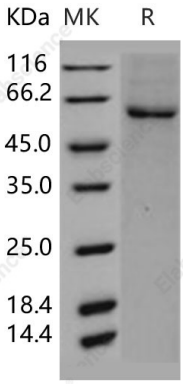
Description

Species	Human
Source	Baculovirus-Insect Cells-derived Human GRK5/GPRK5 protein Met 1-Ser 590, with an C-terminal His
Calculated MW	69 kDa
Observed MW	58 kDa
Accession	NP_005299.1
Bio-activity	Not validated for activity

Properties

Purity	> 90% as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per μg of the protein as determined by the LAL method.
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from sterile 50 mM Tris, 100 mM NaCl, 0.5 mM PMSF, 1 mM DTT, 0.5 mM EDTA, 10% glycerol, pH 7.4 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



> 90 % as determined by reducing SDS-PAGE.

Background

G protein-coupled receptor kinase 5, also known as G protein-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 G protein-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 G protein-coupled receptors. GRK5 contains oneAGC-kinase C-terminal domain, oneprotein kinase domain and oneRGS domain. GRK5 specifically phosphorylates the activated forms of G protein-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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