Recombinant Mouse GAD65/GAD2/GAD-2 Protein (His &GST Tag)

Catalog Number: PKSM040560

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

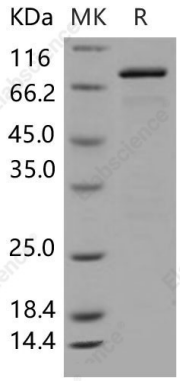
Description

Species	Mouse
Source	Baculovirus-Insect Cells-derived Mouse GAD65/GAD2/GAD-2 protein Met 1-Leu 585, with an N-terminal His & GST
Calculated MW	93.0 kDa
Observed MW	80 kDa
Accession	NP_032104.2
Bio-activity	Not validated for activity

Properties

Purity	> 92 % 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 50mM Tris, 100mM NaCl, 0.5mM PMSF, 0.5mM GSH, 3mM DTT, 10% gly, pH 8.0
Reconstitution	Please refer to the printed manual for detailed information.

Data



> 92 % as determined by reducing SDS-PAGE.

Background

Glutamate decarboxylase 2, also known as glutamate decarboxylase 65 kDa isoform, 65 kDa glutamic acid decarboxylase, GAD2 and GAD65, is a member of thegroup II decarboxylase family. GAD2 is identified as a major autoantigen in insulin-dependent diabetes. GAD2 is responsible for catalyzing the production of gamma-aminobutyric acid from L-glutamic acid. A pathogenic role for this enzyme has been identified in the human pancreas since it has been identified as an autoantibody and an autoreactive T cell target in insulin-dependent diabetes. GAD2 may also play a role in the stiff man syndrome. GAD2 is implicated in the formation of the gamma-aminobutyric acid (GABA), a neurotransmitter involved in the regulation of food intake. GABA is synthesized in brain by two isoforms of glutamic acid decarboxylase (Gad), GAD1 and GAD2. GAD1 provides most of the GABA in brain, but GAD2 can be rapidly activated in times of high GABA demand. Mice lacking GAD2 are viable whereas deletion of GAD1 is lethal. Deletion of GAD2 increased ethanol palatability and intake and slightly reduced the severity of ethanol-induced withdrawal.

Toll-free: 1-888-852-8623
Tel: 1-832-243-6086
Fax: 1-832-243-6017
Web: www.elabscience.com
Email: techsupport@elabscience.com