Mouse GAD65 / GAD2 Protein

Catalog Number: 50653-MNCB



General Information

Gene Name Synonym:

6330404F12Rik; GAD(65); Gad-2; GAD65

Protein Construction:

A DNA sequence encoding the mouse GAD2 (NP_032104.2) (Met 1-Leu 585) was expressed and purified with two additional amino acids (Gly & Pro) at the N-terminus.

Source: Mouse

Expression Host: Baculovirus-Insect Cells

QC Testing

Purity: > 90 % as determined by SDS-PAGE

Endotoxin:

< 1.0 EU per µg of the protein as determined by the LAL method

Stability:

Samples are stable for up to twelve months from date of receipt $\,$ at -70 $\,$ $^{\circ}$ C

Predicted N terminal: Gly

Molecular Mass:

The recombinant mouse GAD2 consists of 587 amino acids and predicts a molecular mass of 65.4 KDa. It migrates as an approximately 58 KDa band in SDS-PAGE under reducing conditions.

Formulation:

Lyophilized from sterile 50mM Tris, 100mM NaCl, 10% gly, 3mM DTT, pH 8.0.

Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Specific concentrations are included in the hardcopy of COA. Please contact us for any concerns or special requirements.

Usage Guide

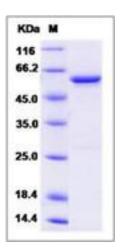
Storage:

Avoid repeated freeze-thaw cycles.

Reconstitution:

Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:



Protein Description

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.

References

1.Karlsen A.E., et al.,(1991), Cloning and primary structure of a human islet isoform of glutamic acid decarboxylase from chromosome 10. Proc. Natl. Acad. Sci. U.S.A. 88:8337-8341. 2.Bu D.-F., et al., (1992), Two human glutamate decarboxylases, 65-kDa GAD and 67-kDa GAD, are each encoded by a single gene.Proc. Natl. Acad. Sci. U.S.A. 89:2115-2119. 3.Bu D.-F., et al.,(1994), The exon-intron organization of the genes (GAD1 and GAD2) encoding two human glutamate decarboxylases (GAD67 and GAD65) suggests that they derive from a common ancestral GAD.Genomics 21:222-228.

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