



Recombinant Protein Technical Manual  
Recombinant Mouse GAD65/GAD2/GAD-2 Protein  
RPES0429

### Product Data:

**Product SKU:** RPES0429

**Size:** 10µg

**Species:** Mouse

**Expression host:** Baculovirus-Insect Cells

**Uniprot:** NP\_032104.2

### Protein Information:

**Molecular Mass:** 65.4 kDa

**AP Molecular Mass:** 58 kDa

**Tag:**

**Bio-activity:**

**Purity:** > 90 % as determined by SDS-PAGE

**Endotoxin:** < 1.0 EU per µg of the protein as determined by the LAL method.

**Storage:** 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, 10% gly, 3mM DTT, pH 8.0

**Reconstitution:** Please refer to the printed manual for detailed information.

**Application:**

**Synonyms:** 6330404F12Rik;GAD(65);Gad-2;GAD65

## Immunogen Information:

**Sequence:** Met 1-Leu 585

## Background:

Glutamate decarboxylase 2, also known as glutamate decarboxylase 65 kDa isoform, 65 kDa glutamic acid decarboxylase, GAD2 and GAD65, is a member of the group 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.