Recombinant Protein Technical Manual

Recombinant Human GAD67/GAD1 Protein (His Tag)

RPES4882

Product Data:

Product SKU: RPES4882

Size: 20µg

Species: Human

Expression host: Baculovirus-Insect Cells

Uniprot: Q99259

Protein Information:

Molecular Mass: 68.3 kDa

AP Molecular Mass: 64 kDa

Tag: C-His

Bio-activity:

Purity: > 92 % as determined by reducing SDS-PAGE.

Endotoxin: < 1.0 EU per µg 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℃ 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, 500mM NaCl, 10% gly, pH 8.5

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

Application:

Synonyms: CPSQ1;GAD;SCP

Immunogen Information:

Sequence: Met 1-Leu 594

Background:

Glutamate decarboxylase 1, also known as 67 kDa glutamic acid decarboxylase, Glutamate decarboxylase 67 kDa isoform and GAD1, is a member of the group II decarboxylase family. GAD1 is expressed in benign and malignant prostatic tissue and may serve as a highly prostate-specific tissue biomarker. GAD1 isoform 3 is expressed in pancreatic islets, testis, adrenal cortex, and perhaps other endocrine tissues, but not in brain. Tissue-specific markers are useful for identification of tumour type in advanced cancers of unknown origin. In plants, as in most eukaryotes, glutamate decarboxylase catalyses the synthesis of GABA. Root-specific calcium/calmodulin-regulated GAD1 plays a major role in GABA synthesis in plants under normal growth conditions and in response to stress. Defects in GAD1 are the cause of cerebral palsy spastic quadriplegic type 1 (CPSQ1)which is a non-progressive disorder of movement and/or posture resulting from defects in the developing central nervous system. Affected individuals manifest symmetrical, non-progressive spasticity and no adverse perinatal history or obvious underlying alternative diagnosis.