Human ENSA / Endosulfine alpha Protein (His Tag)

Catalog Number: **14321-H07E**

General Information

Gene Name Synonym:

ARPP-19e

Protein Construction:

A DNA sequence encoding the mature form of human ENSA (O43768-1) (Met1-Glu121) was expressed with a polyhistide tag at the N-terminus.

Source: Human

Expression Host: E. coli

QC Testing

Purity: > 90 % as determined by SDS-PAGE

Endotoxin:

Please contact us for more information.

Stability:

Samples are stable for up to twelve months from date of receipt at -70 ℃

Predicted N terminal: His

Molecular Mass:

The recombinant human ENSA consists of 136 amino acids and predicts a molecular mass of 15.2 KDa. It migrates as an approximately 18 KDa band in SDS-PAGE under reducing conditions.

Formulation:

Lyophilized from sterile PBS, pH 7.4.

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:

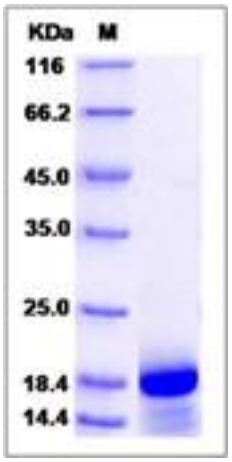
Store it under sterile conditions at -20℃ to -80℃ upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:

Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:



Protein Description

Endosulfine alpha, also known as ENSA, belongs to the endosulfine family. It is a highly conserved cAMP-regulated phosphoprotein (ARPP) family. Endosulfine alpha is widely expressed with high levels in skeletal muscle and brain and lower levels in the pancreas. As a protein phosphatase inhibitor, ENSA specifically inhibits protein phosphatase 2A (PP2A) during mitosis. When phosphorylated at Ser-67 during mitosis, specifically interacts with PPP2R2D (PR55-delta) and inhibits its activity, leading to inactivation of PP2A, an essential condition to keep cyclin-B1-CDK1 activity high during M phase By similarity. Endosulfine alpha also acts as a stimulator of insulin secretion by interacting with sulfonylurea receptor (ABCC8), thereby preventing sulfonylurea from binding to its receptor and reducing K(ATP) channel currents.

References

1.Ye M. et al., 2001, Genome Res. 10 (10): 1546-60. 2.Apiou F. et al., 1999, Diabetes 48 (9): 1873-6. 3.Lennon G. et al., 1997, Genome Res. 6 (9): 791-806.