

Human NMNAT2 / NMNAT-2 Protein (His Tag)



Sino Biological Inc.

Biological Solution Specialist

Catalog Number: 11399-H08B

General Information

Gene Name Synonym:

C1orf15; PNAT2; AI843915; D030041I09Rik; PNAT1; PNAT2

Protein Construction:

A DNA sequence encoding the human NMNAT2 (Q9BZQ4-1)(Met1-Gly307) was fused with a polyhistidine tag at the C-terminus.

Source: Human

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 °C

Predicted N terminal: Met

Molecular Mass:

The recombinant human NMNAT2 consists of 317 amino acids and has a calculated molecular mass of 35.8 kDa. The recombinant protein migrates as an approximately 35 kDa band in SDS-PAGE under reducing conditions.

Formulation:

Lyophilized from sterile 20mM Tris, 500mM NaCl, 5mM DTT, 10% glycerol, 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:

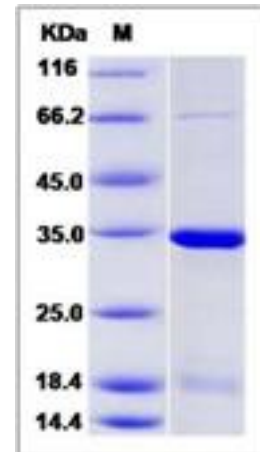
Store it under sterile conditions at -20°C to -80°C upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:

Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:



Protein Description

NMNAT2, also known as NMNAT-2, belongs to the nicotinamide mononucleotide adenylyltransferase (NMNAT) enzyme family. NMNAT is a central enzyme in NAD⁺ biosynthesis, transferring the adenylyl moiety of ATP to nicotinamide mononucleotide (NMN) or nicotinic acid mononucleotide (NaMN) resulting in the formation of NAD⁺ or NaAD⁺ and the release of pyrophosphate. NMNAT2 is predominantly expressed in human pancreas, insulinoma as well as in the brain, especially in the cerebrum, cerebellum, occipital lobe, frontal lobe, temporal lobe and putamen. Immunofluorescence microscopy localized endogenous NMNAT2 to the Golgi apparatus in human cell line. Endogenous NMNAT2 seem to be a labile axon survival factor, because specific depletion of NMNAT2 is sufficient to induce Wallerian-like degeneration of uninjured axons which endogenous NMNAT1 and NMNAT3 cannot prevent. Thus endogenous NMNAT2 represents an exciting new therapeutic target for axonal disorders.

References

1. Ljungberg MC. et al., 2012, Hum Mol Genet. 21 (2): 251-67.
2. Seki N. et al., 1998, DNA Res. 4 (5): 345-9.
3. Raffaelli N. et al., 2002, Biochem Biophys Res Commun. 297 (4): 835-40.

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