

Human AGA / ASRG / Aspartylglucosaminidase Protein (His Tag)

Catalog Number: 13705-H08H



Sino Biological Inc.

Biological Solution Specialist

General Information

Gene Name Synonym:

AGA

Protein Construction:

A DNA sequence encoding the human AGA (CAA39029.1) (Met1-Ile346) was expressed with a C-terminal polyhistidine tag.

Source: Human

Expression Host: Human Cells

QC Testing

Purity: (5.2+44.1+46.8+3.4) % 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: Ser 24

Molecular Mass:

The recombinant human AGA comprises 334 amino acids and has a predicted molecular mass of 36.1 kDa. The apparent molecular mass of the protein is approximately 47, 29, 23 and 20 kDa in SDS-PAGE under reducing conditions due to glycosylation.

Formulation:

Lyophilized from sterile PBS, pH 7.4.

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

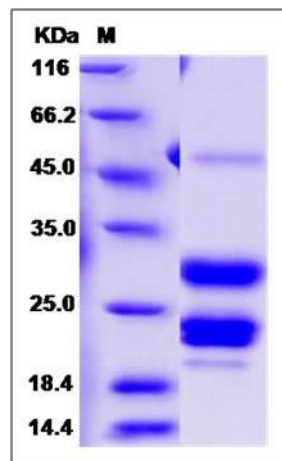
Usage Guide

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.

SDS-PAGE:



Reconstitution:

Detailed reconstitution instructions are sent along with the products.

Protein Description

ASRG, also known as aspartylglucosaminidase, is a lysosomal amidase that hydrolyzes the N- acetylglucosamine -asparagine linkage as one of the final steps in the breakdown of glycoproteins. ASRG cleaves asparagine from N-acetylglucosamines as one of the final steps in the lysosomal breakdown of glycoproteins. The lysosomal storage disease aspartylglucosaminuria is caused by a deficiency in ASRG. Aspartylglucosaminuria is an inborn lysosomal storage disease. Clinical features of aspartylglucosaminuria include mild to severe mental retardation manifesting from the age of 2, coarse facial features and mild connective tissue abnormalities.

References

1. Ikonen E. et al., 1991, EMBO J. 10 (1): 51-8.
2. Enomaa N. et al., 1992, Biochem J. 286 (2): 613-8.
3. Mononen I. et al., 1993, FASEB J. 7 (13): 1247-56.
4. Enomaa NE. et al., 1993, J Histochem Cytochem. 41 (7): 981-9.

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