

Mouse ACO2 / Aconitase 2 Protein (His & GST Tag)

Catalog Number: 51015-M20B



Sino Biological Inc.

Biological Solution Specialist

General Information

Gene Name Synonym:

Aco-2, Aco3, D10Wsu183e

Protein Construction:

A DNA sequence encoding the mouse ACO2 (Q99KI0) (Gln 28-Gln 780) was fused with the N-terminal polyhistidine-tagged GST tag at the N-terminus.

Source: Mouse

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 secreted recombinant mouse ACO2/GST chimera consists of 990 amino acids and has a calculated molecular mass of 110 kDa. The recombinant protein migrates as an approximately 100 kDa band in SDS-PAGE under reducing conditions.

Formulation:

Lyophilized from sterile 50mM Tris, 100mM NaCl, 10% gly, 0.5mM GSH, pH 8.0

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.

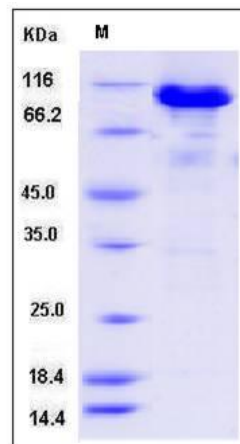
Usage Guide

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

Aconitase employs a cluster to catalyze the stereospecific dehydration-rehydration of citrate to isocitrate via cis-aconitase in the second and third steps of the Krebs cycle. The protein contains four domains. Residues from all four domains contribute to the active site region, which is defined by the Fe[BOND]S cluster. Three of these side chains reside on a three turn 310 helix in the first domain. Diseases relevant to aconitase are aconitase deficiency, Friedreich's ataxia (FRDA), and diabetes. Aconitase deficiency is caused by mutation in the gene for iron-sulfur cluster scaffold protein, which helps to form the Fe-S cluster. FRDA is caused when the Fe-S proteins in aconitase and succinate dehydrogenase have decreased activity. ACO2 is the mitochondrial form of aconitase different from the soluble form ACO1.

References

1. Robbins AH. et al., 1989, Proteins. 5 (4): 289-312.
2. Lauble H. et al., 1992, Biochemistry. 31 (10): 2735-48.
3. Robbins AH. et al., 1989, Proc Natl Acad Sci. 86 (10): 3639-43.

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