Human GDF-15 / GDF15 Protein (His Tag)  
(Mature Form)

Catalog Number: 10936-H07Y

General Information

Gene Name Synonym:
GDF-15; MIC-1; MIC1; NAG-1; PDF; PLAB; PTGFB

Protein Construction:
A DNA sequence encoding the mature form of human GDF15 (NP_004855.2) (Ala197-Ile308) was expressed with a polyhistidine tag at the N-terminus.

Source: Human

Expression Host: Yeast

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 °C

Predicted N terminal: His

Molecular Mass:
The recombinant mature form of human GDF15 consists of 130 amino acids and predicts a molecular mass of 14.2 kDa.

Formulation:
Lyophilized from sterile 35% CAN, 0.1% TFA.

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

Growth-differentiation factor 15 (GDF15), also known as MIC-1, is a secreted member of the transforming growth factor (TGF)-β superfamily, as a novel antihypertrophic regulatory factor in the heart. GDF-15 / GDF15 is not expressed in the normal adult heart but is induced in response to conditions that promote hypertrophy and dilated cardiomyopathy and it is expressed highly in liver. GDF-15 / GDF15 has a role in regulating inflammatory and apoptotic pathways in injured tissues and during disease processes. GDF-15 / GDF15 is synthesized as precursor molecules that are processed at a dibasic cleavage site to release C-terminal domains containing a characteristic motif of 7 conserved cysteines in the mature protein. GDF-15 / GDF15 overexpression arising from an expanded erythroid compartment contributes to iron overload in thalassemia syndromes by inhibiting hepcidin expression.

References