Human Placental Lactogen / CSH1 Protein (His Tag)

Catalog Number: **11596-H08H**

General Information

Gene Name Synonym:

CS-1; CSA; CSMT; hCS-1; hCS-A; PL

Protein Construction:

A DNA sequence encoding the human CSH1 (NP_001308.1) (Met 1-Phe 217) was expressed, fused with a polyhistidine tag at the C-terminus.

Source: Human

Expression Host: HEK293 Cells

QC Testing

Purity: > 87 % 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 ℃

Predicted N terminal: Val 27

Molecular Mass:

The secreted recombinant human CSH1 consists of 202 amino acids and has a calculated molecular mass of 23.7 kDa. It migrates as an approximately 25 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:

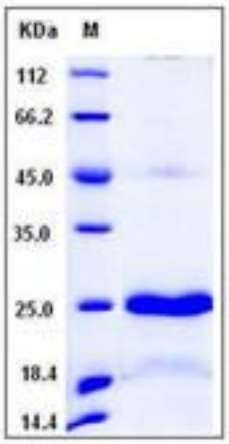
Store it under sterile conditions at -20℃ to -80℃ upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:

Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:



Protein Description

Chorionic somatomammotropin hormone, also known as Choriomammotropin, Lactogen, Placental lactogen and CSH1, is a secreted protein which belongs to thesomatotropin / prolactin family. CSH1 is produced only during pregnancy and is involved in stimulating lactation, fetal growth and metabolism. Does not interact with GHR but only activates PRLR through zinc-induced dimerization. The CSH1 gene is member of the GH gene cluster on 17q, which consists of two growth hormone genes and three CSH genes. Genomic alterations in the GH cluster are well known, causing different phenotypes depending on the size of the deletion and the genes involved. The increased prevalence of hemizygosity of CSH1 in population in comparison to controls indicates a role for CSH1 haploinsufficiency in the etiology of growth retardation. Investigation of CSH1 deletions in further SRS and growth retarded patients will enable us to establish under which circumstances haploinsufficiency of CSH1 is likely to result in clinical changes.

References

1.Prager,S. et al., 2003,Genet Test. 7 (3):259-63. 2.Singleton, DR. et al., 2004, Microbiology. 150 (Pt 2): 285-92. 3.Chen,Y. et al., 2008, Cancer Res. 68 (23):9729-34.