

Human GALE / UDP galactose-4-epimerase Protein (His Tag)

Catalog Number: 14810-H07E



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Biological Solution Specialist

General Information

Gene Name Synonym:

SDR1E1; Z310002A12Rik; AI323962

Protein Construction:

A DNA sequence encoding the mature form of human GALE (Q14376) (Met1-Ala348) was expressed with a polyhistidine tag at the N-terminus.

Source: Human

Expression Host: E. coli

QC Testing

Purity: > 95 % 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 human GALE consists of 363 amino acids and predicts a molecular mass of 40.1 KDa. It migrates as an approximately 36 KDa band in SDS-PAGE under reducing conditions.

Formulation:

Lyophilized from sterile 20mM HEPES, 150mM NaCl, 10% Glycerol, pH 7.5.

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:

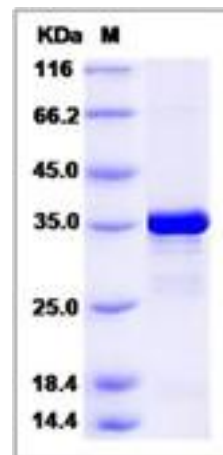
Store it under sterile conditions at -20°C to -80°C upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:

Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:



Protein Description

UDP galactose-4'-epimerase, also known as GALE, enables the body to process a simple sugar called galactose, which is present in small amounts in many foods. Galactose is primarily part of a larger sugar called lactose, which is found in all dairy products and many baby formulas. UDP galactose-4'-epimerase catalyzes two distinct but analogous reactions: the epimerization of UDP-glucose to UDP-galactose, and the epimerization of UDP-N-acetylglucosamine to UDP-N-acetylgalactosamine. Defects in GALE causes epimerase-deficiency galactosemia (EDG), also known as galactosemia type 3. Clinical features include early-onset cataracts, liver damage, deafness and mental retardation.

References

1. Kim W. et al., 2011, Mol Cell. 44 (2): 325-40.
2. Lee KA. et al., 2011, J Biol Chem. 286 (48): 41530-8.
3. McCorvie TJ. et al., 2012, Biochim Biophys Acta. 1822 (10): 1516-26.

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