

Human LIFR Protein (Fc Tag)

Catalog Number: 10628-H02H

General Information

Gene Name Synonym:

CD118; LIF-R; SJS2; STWS; SWS

Protein Construction:

A DNA sequence encoding the human LIFR (NP_002301.1) (Met1-Ser833) was expressed with the Fc region of human IgG1 at the C-terminus.

Source: Human

Expression Host: HEK293 Cells

QC Testing

Purity: > 95 % as determined by SDS-PAGE.

Endotoxin:

< 1.0 EU per µg 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: Gln 45

Molecular Mass:

The recombinant human LIFR consists of 1027 amino acids and predicts a molecular mass of 116.1 kDa.

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:

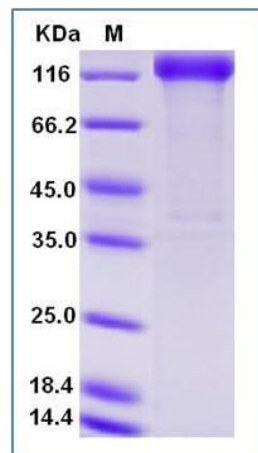
Store it under sterile conditions at -20°C to -80°C upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:

Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:



Protein Description

LIFR (leukemia inhibitory factor receptor) belongs to the family of cytokine receptors. LIFR forms a high-affinity receptor complex with gp13, which mediates the activity of LIF (leukemia inhibitory factor) and thus affects the differentiation, proliferation, and survival of a wide variety of cells in the adult and the embryo. Besides LIF, LIFR can also bind to and activate CNTF (ciliary neurotrophic factor) and CLC (cardiotrophin like cytokine). Evidence showed that in the retina, LIFR activating LIF, CT-1 and cardiotrophin like cytokine (CLC) are strongly upregulated in response to preconditioning with bright cyclic light leading to robust activation of signal transducer and activator of transcription-3 (STAT3) in a time-dependent manner. Further, blocking LIFR activation during preconditioning using a LIFR antagonist (LIF5) attenuated the induced STAT3 activation and also resulted in reduced preconditioning-induced protection of the retinal photoreceptors. These data demonstrate that LIFR and its ligands play an essential role in endogenous neuroprotective mechanisms triggered by preconditioning-induced stress. LIFR was newly found to be a suppressor of hepatocellular carcinoma (HCC), one of the world's top five causes of cancer-related deaths.

References

1.Gearing, D.P. et al.,1991, EMBO J. 10 (10): 2839-2848. 2.Gearing, D.P. et al.,1992, New Biol. 4 (1): 61-65. 3.Mosley, B. et al.,1996, J. Biol. Chem. 271 (51): 32635-32643.

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