Human OSMR / IL31RB Protein (His Tag)

Catalog Number: 11226-H08H

General Information

Gene Name Synonym:

IL-31RB; OSMRB; PLCA1

Protein Construction:

A DNA sequence encoding the human OSMR (NP_003990.1) extracellular domain (Met 1-Met 740) was expressed, fused with a polyhistidine tag at the C-terminus.

Source: Human

Expression Host: HEK293 Cells

QC Testing

Purity: ≥ 96 % as determined by SDS-PAGE. ≥ 90 % as determined by SEC-HPLC.

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: Glu 28

Molecular Mass:

The secreted recombinant human OSMR comprises 724 amino acids and has a predicted molecular mass of 82.6 kDa. As a result of glycosylation, rh OSMR migrates as an approximately 130-140 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:

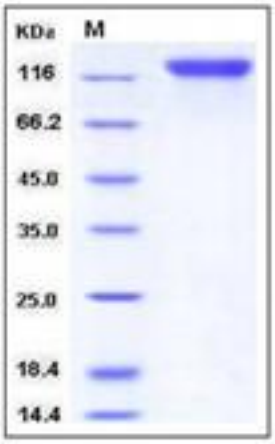
Store it under sterile conditions at -20℃ to -80℃ upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:

Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:



Protein Description

Oncostatin-M specific receptor subunit beta also known as the oncostatin M receptor (OSMR) and Interleukin-31 receptor subunit beta (IL-31RB), is one of the receptor proteins for oncostatin M. OSMR is a member of the type I cytokine receptor family. IL-31RB/OSMR heterodimerizes with interleukin 6 signal transducer to form the type II oncostatin M receptor and with interleukin 31 receptor A to form the interleukin 31 receptor, and thus transduces oncostatin M and interleukin 31 induced signaling events. Mutations in IL-31RB/OSMR have been associated with familial primary localized cutaneous amyloidosis. Defects in IL-31RB/OSMR are the cause of amyloidosis primary localized cutaneous type 1 (PLCA1), also known as familial lichen amyloidosis or familial cutaneous lichen amyloidosis. PLCA1 is a hereditary primary amyloidosis characterized by localized cutaneous amyloid deposition. This condition usually presents with itching (especially on the lower legs) and visible changes of skin hyperpigmentation and thickening (lichenification) that may be exacerbated by chronic scratching and rubbing. The amyloid deposits probably reflect a combination of degenerate keratin filaments, serum amyloid P component, and deposition of immunoglobulins.

References

1.Arita K, *et al*.. (2008) Oncostatin M Receptor-β Mutations Underlie Familial Primary Localized Cutaneous Amyloidosis. Am J Hum. Genet. 82 (1): 73-80. 2.Malaval L, *et al*.. (2005) GP130/OSMR is the only LIF/IL-6 family receptor complex to promote osteoblast differentiation of calvaria progenitors. J Cell Physiol. 204(2): 585-93. 3.Lin MW, *et al*.. (2010) Novel IL31RA gene mutation and ancestral OSMR mutant allele in familial primary cutaneous amyloidosis. Eur J Hum Genet. 18(1): 26-32.