Mouse OSMR / IL-31RB Protein (His Tag)

Catalog Number: 50500-M08H



General Information

Gene Name Synonym:

OSMRB

Protein Construction:

A DNA sequence encoding the mouse OSMR (O70458-1) extracellular domain (Met 1-Leu 738) was expressed, fused with a polyhistidine tag at the C-terminus.

Source: Mouse

Expression Host: HEK293 Cells

QC Testing

Purity: > 97 % 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 $\,$ $^{\circ}$ C

Predicted N terminal: Glu 24

Molecular Mass:

The secreted recombinant mouse OSMR consists of 726 amino acids and has a predicted molecular mass of 83.7 kDa. In SDS-PAGE under reducing conditions, the apparent molecular mass of rmOSMR is approximately 120-130 kDa due to glycosylation.

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

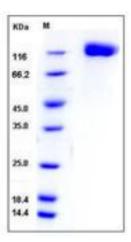
Storage:

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.

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