

Mouse IL2RA / CD25 Protein (ECD, His Tag)



Sino Biological
Biological Solution Specialist

Catalog Number: 50292-M08H

General Information

Gene Name Synonym:

CD25; IL2r; Ly-43

Protein Construction:

A DNA sequence encoding the mouse IL2RA (NP_032393.3) extracellular domain (Met 1-Lys 236) was expressed, with a C-terminal polyhistidine tag.

Source: Mouse

Expression Host: HEK293 Cells

QC Testing

Purity: ≥ 98 % as determined by SDS-PAGE. ≥ 95 % as determined by SEC-HPLC.

Bio Activity:

Immobilized Recombinant Human IL2 Protein, Low Endotoxin (Cat: 11848-HNAH1-E) at 2 µg/ml (100 µl/well) can bind Recombinant Mouse IL2RA / CD25 Protein (ECD, His Tag) (Cat: 50292-M08H). The EC₅₀ is 410-900 ng/mL.

Endotoxin:

< 1.0 EU per µg of the protein as determined by the LAL method

Predicted N terminal: Glu 22

Molecular Mass:

The secreted recombinant mouse IL2RA comprises 226 amino acids with a predicted molecular mass of 26 kDa. As a result of glycosylation, the apparent molecular mass of the rm IL2RA is approximately 50-55 kDa 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

Stability & Storage:

Samples are stable for twelve months from date of receipt at -20°C to -80°C.

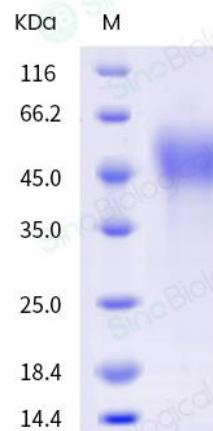
Store it under sterile conditions at -20°C to -80°C upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:

Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:



Protein Description

CD25 (alpha-chain of the IL-2 receptor, or IL2RA), is a type I transmembrane glycoprotein with a signal peptide, an extracellular region, a transmembrane region, and a cytoplasmic domain. IL2RA is expressed on activated T cells and regulatory T cells and is capable of binding IL2 with low affinity by itself. However, a ligand-induced high-affinity heterotrimeric receptor complex is produced when IL2RA is associated non-covalently with the IL2 receptor beta and gamma chain, and subsequently initiates the intracellular signal pathways such as MAPK or JAK/STAT. On dendritic cells (DC), CD25 has been previously regarded as an activation marker, while both murine and human DC can express CD25, they do not express the beta-chain of the IL-2 receptor, which is indispensable for the execution of IL-2 signaling. The IL2RA (CD25) gene is a substantial component of the high-affinity receptor molecule highly expressed by activated T lymphocytes. Recently, a piece of strong evidence was obtained for the involvement of IL-2RA in conferring susceptibility to type 1 diabetes (T1D). Cancer growth and development are associated with the stimulation of the innate immune system, including enhanced interleukin 2 receptor (IL-2R) expression in immune cells and its shedding into the circulation in a soluble form of sIL-2Ralpha. In most hematological malignancies, including different types of leukemias and lymphomas, sIL-2Ralpha is released directly from the surface of neoplastic cells thus reflecting the tumor bulk, turnover, and activity. Several studies have proved that not only lymphoid cancer cells but also some non-lymphoid cancer cells, express IL-2R on their surface. They include malignant melanoma and carcinomas of the kidney, head and neck, esophagus, and lung. Thus, sIL-2Ralpha is elevated in most proliferative disturbances of the hematopoietic system and many solid tumors.

References

1. Driesen J, *et al.* (2008) CD25 as an immune regulatory molecule expressed on myeloid dendritic cells. *Immunobiology*. 213(9-10): 849-58.
2. Olejniczak K, *et al.* (2008) Biological properties of interleukin 2 and its role in pathogenesis of selected diseases--a review. *Med Sci Monit*. 14(10): RA179-89.
3. Chistiakov DA, *et al.* (2008) The crucial role of IL-2/IL-2RA-mediated immune regulation in the pathogenesis of type 1 diabetes, an evidence coming from genetic and animal model studies. *Immunol Lett*. 118(1): 1-5.