Human IL7RA / CD127 Protein (His Tag)

Catalog Number: 10975-H08H

Sino Biological
Biological Solution Specialist

General Information

Gene Name Synonym:

CD127; CDW127; IL-7R; IL-7R-alpha; IL7RA; ILRA

Protein Construction:

A DNA sequence encoding the human IL7Rα IL7R (P16871-1) (Met1-Gly236, with mutations I66T, V138I) was expressed with a polyhistidine tag at the C-terminus.

Source: Human

Expression Host: HEK293 Cells

QC Testing

Purity: ≥ 92 % as determined by SDS-PAGE. ≥ 95 % as determined by SEC-HPLC.

Bio Activity:

Measured by its ability to bind biotinylated mouse TSLP-his in functional ELISA.

Endotoxin:

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

Predicted N terminal: Glu 21

Molecular Mass:

The recombinant human IL7Rαconsists of 227 amino acids and has a predicted molecular mass of 26.3 kDa. In SDS-PAGE under reducing conditions, the apparent molecular mass of human IL7Rα is approximately 55.4 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

Stability & Storage:
Samples are stable for twelve months from date of receipt at -20℃ to -80℃.

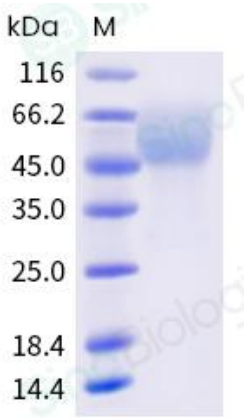
Store it under sterile conditions at -20℃ to -80℃ upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:

Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:



Protein Description

Interleukin 7 Receptor alpha (IL-7RA), also known as CD127, is a 75 kDa hematopoietin receptor superfamily member that plays an important role in lymphocyte differentiation, proliferation, and survival. IL-7 receptor alpha (CD127) signaling is essential for T-cell development and regulation of naive and memory T-cell homeostasis. IL-7RA is critically required for the proper development and function of lymphoid cells. Therefore, the IL-7RA is critically required for the proper development and function of lymphoid cells. Studies from both pathogenic and controlled HIV infection indicate that the containment of immune activation and preservation of CD127 expression are critical to the stability of CD4(+) T cells in infection. A better understanding of the factors regulating CD127 expression in HIV disease, particularly on T(CM) cells, might unveil new approaches exploiting the IL-7/IL-7R receptor pathway to restore T cell homeostasis and promote immune reconstitution in HIV infection. Factors relevant to HIV infection that could potentially decrease CD127 expression on human CD8(+) T cells. CD127 down-regulation may be an important contributor to HIV-associated T-cell dysfunction. In addition to IL-7, IL-7RA also associates with TSLPR to form the functional receptor for thymic stromal lymphopoietin (TSLP) which indirectly regulates T cell development by modulating dendritic cell activation. Mutations in the human IL-7RA gene cause a type of severe combined immunodeficiency in which the major deficiencies are in T cell development, whereas B and NK cells are relatively normal in number. Variation in the IL7RA gene was recently found associated with multiple sclerosis (MS). The polymorphisms in the IL7RA gene is involved in MS pathogenesis and suggest that IL7RA variation may primarily affect chronic disease courses. Soluble CD127 (sCD127) appears to play an important role in the immunopathogenesis of several chronic infections, multiple sclerosis, and various cancers.

References

1.Vranjkovic A, et al. (2007) IL-7 decreases IL-7 receptor alpha (CD127) expression and induces the shedding of CD127 by human CD8+ T cells. Int Immunol. 19(12): 1329-39.
2.Kiazyk SA, et al. (2008) Loss of CD127 expression links immune activation and CD4(+) T cell loss in HIV infection. Trends Microbiol. 16(12): 567-73.
3.Akkad DA, et al. (2009) Variation in the IL7RA and IL2RA genes in German multiple sclerosis patients. J Autoimmun. 32(2): 110-5.