IL-17RC Protein, Human (sf9, His)

Cat. No.:	HY-P75837
Synonyms:	Interleukin-17 receptor C; IL-17 receptor C; IL17RC; IL17Rhom; IL-17RL; ZcytoR14
Species:	Human
Source:	Sf9 insect cells
Accession:	Q8NAC3-3 (M1-A454)
Gene ID:	84818
Molecular Weight:	Approximately 60 kDa

PROPERTIES

Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 μm filtered solution of 20 mM Tris, 500 mM NaCl, pH 7.4, 10% Glycerol. Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH_2O.
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US; may vary elsewhere.

DESCRIPTION

Background

IL-17RC, is the receptor for IL17A and IL17F homodimers as part of a heterodimeric complex with IL17RA. IL-17 cytokine family members IL-17A and IL-17F mediate inflammatory activities via the IL-17R complex, comprised of the IL-17RA and IL-17RC subunits. The expression profile and tissue distribution of IL-17RC suggest that the gene regulation of IL-17RC differs considerably from IL-17RA. Specifically, epithelial cells of the prostate, kidney, and joints express high levels of IL-17RC mRNA, while low levels of expression are detected in the hematopoietic cell compartments[1].

The amino acid sequence of human IL-17RC protein has low homology with mouse IL-17C protein. The differences between the human and murine systems extend to IL-17A and IL-17F cytokine binding affinities. hIL-17RA binds preferentially to IL-17A and has a relatively low binding affinity to IL-17F. In contrast, hIL-17RC binds IL-17A and IL-17F with the same affinity. In the murine system, the inverse is true: mIL-17RA binds IL-17A and IL-17F with equal affinities, but mIL-17RC binds preferentially to IL-17F. Therefore, in both humans and mice, IL-17RC appears to serve as a contact point for IL-17F[1].

The IL-17R subfamily includes IL-17RA, IL-17RB, IL-17RC, IL-17RD, and IL-17RE. The best-characterized IL-17R molecules are the IL-17RA and IL-17RC subunits, in part because of their interaction to form a receptor complex capable specific for IL-17A and IL-17F. IL-17RA co-immunoprecipitates with IL-17RC in a ligand-dependent manner, raising the possibility that the ligand-dependent loss of FRET between IL-17RA subunits results from oligomerization with IL-17RC. Consistent with this, IL-17RC also forms large, multimeric complexes consistent with oligomerization with IL-17RA. IL-17RC forms heterodimers

with IL-17RA to mediate IL-17A and IL-17F signals in mouse stromal cells and human gastric adenocarcinoma AGS cells and synoviocytes. Although The IL-17RA and IL-17RC subunits operate in concert to mediate IL-17 signaling, IL-17RC possesses a number of features that differentiate it from IL-17RA. IL-17RC bears only 22% sequence homology with IL-17RA. Alignment against the human genome indicates that the il17rc gene contains 19 exons on chromosome 3 and spans 16,550 base pairs within the chromosomal region 3p25.3 to 3.24.1. The murine il17rc gene contains 18 exons on chromsome 6 and spans 11,565 base pairs on the chromosomal arm 6q. The full-length human IL-17RC (hIL-17RC) contains 720 amino acids, and the murine IL-17RC (mIL-17RC) contains 698 amino acids. In both species, il17rc encodes a single pass type I transmembrane protein where the transmembrane domain is encoded in exon 17[1][2].

The initial discovery of IL-17RC was based on its high levels of expression in human prostate cancer cells. Specific overexpression of IL-17RC protects prostate cancer cell lines from TNFα-induced apoptosis. IL-17RC also contributes to autoimmune disease pathogenesis. In rheumatoid arthritis (RA) models have high levels of IL-17A, IL-17F, IL-17RA, and IL-17RC in sera and inflamed synovium. Furthermore, based on RNAi blocking experiments, both IL-17RA and IL-17RC are required for the pro-inflammatory factors secreted by RA synoviocytes. The gene transcript analyses of psoriatic lesions revealed an impairment of IL-17RC mRNA expression. Perhaps this defect in IL-17RC expression leads to a compensatory effect, which could result in overactive Th17 cells and an inflammatory program[1][2].

REFERENCES

[1]. Allen W Ho, et al. IL-17RC: a partner in IL-17 signaling and beyond. Semin Immunopathol. 2010 Mar;32(1):33-42.

[2]. Dongxia Ge, et al. Expression of interleukin-17RC protein in normal human tissues. Int Arch Med. 2008 Oct 17;1(1):19.

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