# IL-17RE Protein, Human (HEK293, Fc)

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| **Cat. No.:** | HY-P72576 |
| **Synonyms:** | IL-17 RE; IL-17 receptor E; IL17RE; Interleukin 17 receptor E |
| **Species:** | Human |
| **Source:** | HEK293 |
| **Accession:** | Q8NFR9 (T155-H454) |
| **Gene ID:** | 132014 |
| **Molecular Weight:** | 60-75 kDa |

## PROPERTIES

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| **AA Sequence** | T Q P S D P E T W E S L P R L D S Q R H G G P E F S F D L L P E A R A I R V T I<br>S S G P E V S V R L C H Q W A L E C E E L S S P Y D V Q K I V S G G H T V E L P<br>Y E F L L P C L C I E A S Y L Q E D T V R R K K C P F Q S W P E A Y G S D F W K<br>S V H F T D Y S Q H T Q M V M A L T L R C P L K L E A A L C Q R H D W H T L C K<br>D L P N A T A R E S D G W Y V L E K V D L H P Q L C F K F S F G N S S H V E C P<br>H Q T G S L T S W N V S M D T Q A Q Q L I L H F S S R M H A T F S A A W S L P G<br>L G Q D T L V P P V Y T V S Q A R G S S P V S L D L I I P F L R P G C C V L V W<br>R S D V Q F A W K H L L C P D V S Y R H |
| **Appearance** | Lyophilized powder |
| **Formulation** | Lyophilized from a 0.2 μm filtered solution of PBS, pH 7.4. |
| **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 $ddH_2O$. For long term storage it is recommended to add a carrier protein (0.1% BSA, 5% HSA, 10% FBS or 5% Trehalose). |
| **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

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| **Background** | IL-17RE is an orphan receptor of the IL-17 receptor family. IL-17RE is a receptor specific to IL-17C and has an essential role in host mucosal defense against infection. Also, no interactions were found between IL-17RE and any of the other IL-17 cytokine family members. IL-17C activated downstream signaling through IL-17RE-IL-17RA complex for the induction of genes encoding antibacterial peptides as well as proinflammatory molecules. IL-17RE is required for IL-17C-induced |

production of antibacterial peptides, proinflammatory cytokines and chemokines both ex vivo and in vivo[1][3].
The amino acid sequence of human IL-17RE protein has low homology between mouse and rat IL-17E protein.
IL-17C is a homodimeric cytokine that is expressed by non-hematopoietic-mainly epithelial-cells. It binds to its heterodimeric receptor complex IL-17RA/RE that is expressed on both a variety of epithelial cells and TH17 cells. Compared to other IL-17 cytokine family members, IL17C is upregulated at early stages of the diseases and plays two roles. (a) In an autocrine manner it sustains barrier integrity of epithelial cell layers and thus supports the innate immune system to keep infections in check. (b) By binding to IL-17RE on TH17 cells, IL-17C also stimulates the adaptive immune response to potently fight off invading pathogens. Binding of IL-17C to the IL-17RA/RE complex on the epithelial IL-17C-source cells forms an autocrine loop in the epithelium. Like IL-17A, IL-17C signaling through IL-17RA/RE employs the adaptor molecule ACT1. The signaling cascade then activates the MAPK pathway by phosphorylation of p38, ERK, and JNK as well as the NF-κB pathway by phosphorylation of the p65 subunit and the NF-κB inhibitor IκBα. Also, signaling through L-17RA/RE on epithelial cells reinforces the mechanical epithelial barrier by expressing the tight-junction proteins occludin, claudin-1, and claudin-4. Intracellular signaling of IL-17C/RE involves anti-apoptotic Bcl-2 and Bcl-XL as well as the NF-κB and MAPK pathways to promote proliferation and host defense[2].
IL-17RE meditates T cell activation, including the expression of effector cytokines (e.g. IL-17A), and the IL-17C/IL-17RE-axis enhances the expression of cytokine by effector Th17 cells in a in a model of autoimmune disease. IL-17RE is also highly expressed by liver resident $CD4^+$ T cells and natural killer T cells and augments T cell function in autoimmune hepatitis together with IL-17C. Deficiency of IL-17RE also provides protection in a model of crescentic nephrotoxic nephritis[1]. IL-17C/IL-17RE-axis assuming a crucial role in protection against bacteria-driven DSS-induced colitis. IL-17C/IL-17RE-axis also plays a role in the defense against airway infections with Pseudomonas aeruginosa and Haemophilus influenza. In addition, the IL-17C/IL-17RE-axis might be involved in the pathogenesis of COPD exacerbations of mixed upper airway infections. Also, IL-17C/IL-17RE signaling aggravates the course of experimental autoimmune encephalitis (EAE)[2].

## REFERENCES


[1]. Giovanna Vella, et al. The IL-17 receptor IL-17RE mediates polyIC-induced exacerbation of experimental allergic asthma. Respir Res. 2020 Jul 8;21(1):176.

[2]. Jasper F Nies, et al. IL-17C/IL-17RE: Emergence of a Unique Axis in TH17 Biology. Front Immunol. 2020 Feb 26;11:341.

[3]. Xinyang Song, et al. IL-17RE is the functional receptor for IL-17C and mediates mucosal immunity to infection with intestinal pathogens. Nat Immunol. 2011 Oct 12;12(12):1151-8.

[4]. Caini Liu, et al. The flavonoid cyanidin blocks binding of the cytokine interleukin-17A to the IL-17RA subunit to alleviate inflammation in vivo. Sci Signal. 2017 Feb 21;10(467):eaaf8823.





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