

**DESCRIPTION**

<b>Source</b>	Mouse myeloma cell line, NS0-derived		
	Mouse IL-17 RA/IL-17 R (Ser32-Trp322) Accession # Q60943	IEGRMDP	Mouse IgG <sub>2A</sub> (Glu98-Lys330)
	N-terminus		C-terminus
<b>N-terminal Sequence Analysis</b>	Ser32		
<b>Structure / Form</b>	Disulfide-linked homodimer		
<b>Predicted Molecular Mass</b>	60.4 kDa (monomer)		

**SPECIFICATIONS**

<b>SDS-PAGE</b>	80-95 kDa, reducing conditions
<b>Activity</b>	Measured by its ability to inhibit IL-17-induced IL-6 secretion by NIH-3T3 mouse embryonic fibroblast cells. The ED <sub>50</sub> for this effect is 0.015-0.075 µg/mL in the presence of 10 ng/mL of recombinant mouse IL-17.
<b>Endotoxin Level</b>	<0.01 EU per 1 µg of the protein by the LAL method.
<b>Purity</b>	>95%, by SDS-PAGE under reducing conditions and visualized by silver stain.
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

**PREPARATION AND STORAGE**

<b>Reconstitution</b>	Reconstitute at 100 µg/mL in sterile PBS.
<b>Shipping</b>	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
<b>Stability &amp; Storage</b>	<p><b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b></p> <ul style="list-style-type: none"> <li>● 12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>● 1 month, 2 to 8 °C under sterile conditions after reconstitution.</li> <li>● 3 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

**BACKGROUND**

IL-17 R, also known as IL-17 RA, is a 120 kDa type I transmembrane glycoprotein protein that plays a central role in inflammatory responses (1-3). Mature mouse IL-17 R consists of a 291 amino acid (aa) extracellular domain, a 21 aa transmembrane segment, and a 521 aa cytoplasmic domain (4). The cytoplasmic domain contains a region homologous to the TIR domain of the TLR/IL-1 R family (5). Mouse IL-17 R shares 84% and 72% aa sequence identity with rat and human IL-17 R, respectively. Within the extracellular domain, it shares 18%-25% sequence identity with mouse IL-17 RB, C, D, and E. While the expression of IL-17 is restricted to activated T cells, IL-17 R exhibits a broad tissue distribution (4). Even in the absence of ligand, IL-17 R exists on the cell surface as a multimer (6). IL-17 R can bind IL-17 but must associate with IL-17 RC to transduce signals (7, 8). Interestingly, human IL-17 R does not appear to form productive complexes with mouse IL-17 RC (8). The IL-17 R can also signal in response to IL-17F (9). IL-17 R ligation promotes T cell activation and the production of IL-6, G-CSF, SCF, and multiple pro-inflammatory chemokines (4, 7, 9, 10). IL-17A and IL-17F synergize with TNF-α in the induction of CXCL1, G-CSF, and IL-6 (9, 11). This effect requires the presence of both TNF RI and TNF RII (9). IL-17 interactions with IL-17 R also inhibit the TNF-α induced up-regulation of fibroblast CCL5 and VCAM-1 (11). CCL5 and VCAM-1 induced effects are differentially sensitive to blockade with IL-17 R specific antibodies, suggesting that IL-17 R triggers divergent intracellular signals (11). *In vivo*, IL-17 R activity is important for increased generation of neutrophils and their recruitment to sites of inflammation (10, 12, 13). IL-17 R is required for host defense against microbial infection and for the progression of arthritis from inflammation to destructive joint erosion (10, 13).

**References:**

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