

**DESCRIPTION**

<b>Source</b>	<i>E. coli</i> -derived human IL-4 protein His25-Ser153, with an N-terminal Met Accession # P05112.1
<b>N-terminal Sequence Analysis</b>	Met
<b>Predicted Molecular Mass</b>	15.1 kDa

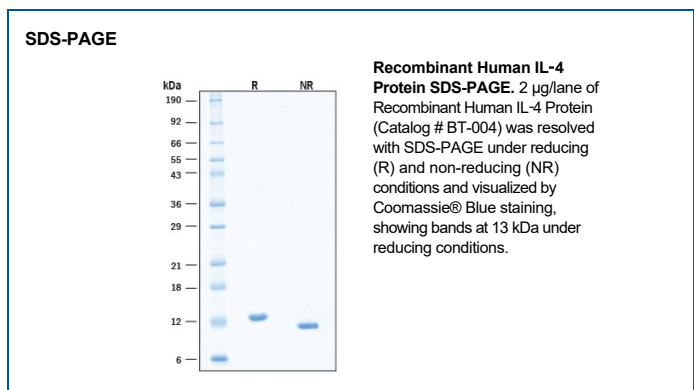
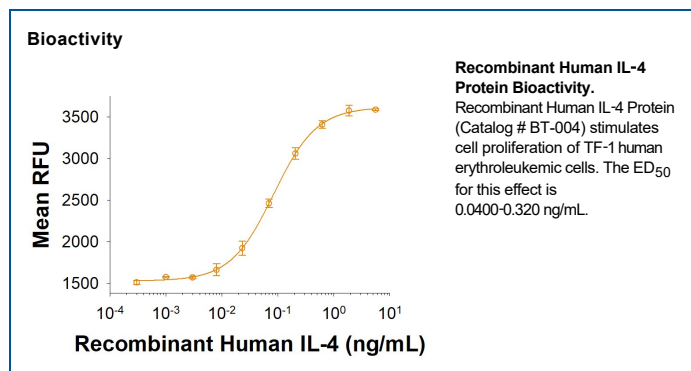
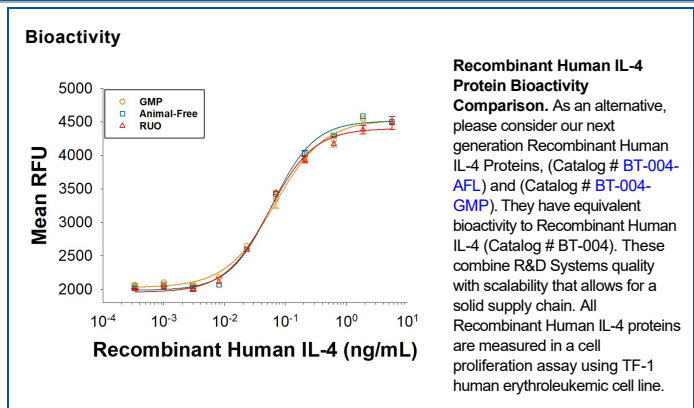
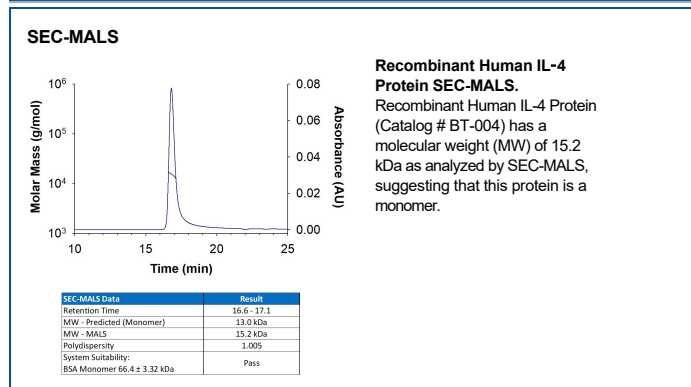
**SPECIFICATIONS**

<b>SDS-PAGE</b>	13 kDa, under reducing conditions.
<b>Activity</b>	Measured in a cell proliferation assay using TF-1 human erythroleukemic cells. Kitamura, T. <i>et al.</i> (1989) J. Cell Physiol. <b>140</b> :323. The ED <sub>50</sub> for this effect is 0.0400-0.320 ng/mL.
<b>Endotoxin Level</b>	<0.10 EU per 1 µg of the protein by the LAL method.
<b>Purity</b>	>97%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

**PREPARATION AND STORAGE**

<b>Reconstitution</b>	Reconstitute the 10 µg size at 100 µg/mL in PBS. Reconstitute all other sizes at 500 µg/mL in 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>

**DATA**



**BACKGROUND**

Interleukin-4 (IL-4), also known as B cell-stimulatory factor-1, is a monomeric, approximately 13 kDa-18 kDa Th2 cytokine that shows pleiotropic effects during immune responses (1-3). It is a glycosylated polypeptide that contains three intrachain disulfide bridges and adopts a bundled four  $\alpha$ -helix structure (4). Human IL-4 is synthesized with a 24 aa signal sequence. Alternate splicing generates an isoform with a 16 aa internal deletion. Mature human IL-4 shares 55%, 39% and 43% aa sequence identity with bovine, mouse, and rat IL-4, respectively. Human, mouse, and rat IL-4 are species-specific in their activities (5-7). IL-4 exerts its effects through two receptor complexes (8, 9). The type I receptor, which is expressed on hematopoietic cells, is a heterodimer of the ligand binding IL-4 Ra and the common  $\gamma$  chain (a shared subunit of the receptors for IL-2, -7, -9, -15, and -21). The type II receptor on nonhematopoietic cells consists of IL-4 Ra and IL-13 Ra1. The type II receptor also transduces IL-13 mediated signals. IL-4 is primarily expressed by Th2-biased CD4<sup>+</sup> T cells, mast cells, basophils, and eosinophils (1, 2). It promotes cell proliferation, survival, and immunoglobulin class switch to IgG4 and IgE in human B cells, acquisition of the Th2 phenotype by naïve CD4<sup>+</sup> T cells, priming and chemotaxis of mast cells, eosinophils, and basophils, and the proliferation and activation of epithelial cells (10-13). IL-4 plays a dominant role in the development of allergic inflammation and asthma (12, 14).

Due to its role in the differentiation of certain immune cell types, IL-4 is commonly used in combination with other growth factors to transform induced pluripotent stem cells into dendritic cells in high numbers. These dendritic cells can then be used for research or clinical applications to improve disease modeling, for screening and cell therapies (15). Study of IL-4 signaling has led to the development of monoclonal antibodies that can block the signaling pathway at various steps to mitigate the inflammatory response in certain autoimmune diseases (16, 17). While IL-4 has the capacity to improve immune functions, treatments involving IL-4 have not been utilized due to the dangerous side effects that may result from IL-4 signaling in non-immune cells (16). Blockade of IL-4 signaling also has been studied as a therapeutic target to suppress inflammation in the tumor microenvironment (18). Use of IL-4 suppressors can also improve the efficacy of anti-tumor immunotherapies, as blocking IL-4 enhances activity of tumor-specific T lymphocytes (19, 20).

**References:**

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