

human IL-6, His-tag

Interleukin 6

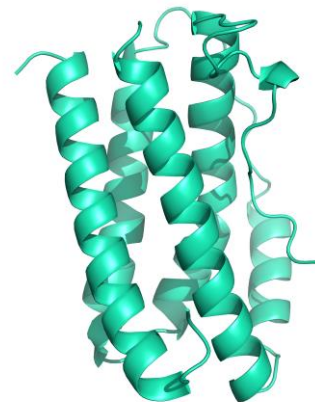
Cat. no. P2020-140

Product Information

Protein:	human IL-6, His-tag (~ 22.6 kDa)
Uniprot#:	P05231
Sequence:	MVPPGEDSKDVAAPHRQPLTSSERIDKQIRYILDGISALRKETCNKSNMCESSKEALAEN NLNLPKMAEKDGCFSQGFNEETCLVKIITGLLEFEVYLEYLQNRFESSEEQARAVQMSTK VLIQFLQKKAKNLDAITTPDPTTASLLTKLQAQNQWLQDMTTHLILRSFKEFLQSSLRA LRQM
	Methionine at pos. 1 might be present due to cloning constraints, C-terminal His-tag not shown in sequence.
Source:	Recombinantly expressed in HEK293 cells.
Tag(s):	His-tag, C-terminal
Purification:	Purified by affinity chromatography and subsequent buffer exchange.
Formulation:	PBS; pH 7.4. Liquid, stored and shipped at -80 °C.
Purity:	> 85% (will be determined by densitometry of Coomassie stained gel, example next page)
Concentration:	Will be determined by BCA-Assay.
Long-term storage:	No recommendations.
Comment:	Protein migrates at higher molecular weight during SDS-PAGE due to posttranslational modifications.

Background Information:

Interleukin-6 (IL-6) is an important pro-inflammatory cytokine, which belongs to the type I cytokine family and is produced by a variety of cell types, including mononuclear phagocytes, dendritic cells, vascular endothelial cells and fibroblasts. Signaling is induced by binding of IL-6 to the cytokine-binding polypeptide chain IL-6R alpha, leading to association of IL-6R alpha with the signal-transducing subunit gp130, together constituting the IL-6 receptor. Expression of the classical IL-6 receptor is restricted to hepatocytes, monocytes, some resting lymphocytes and some epithelial cells. Remarkably, alternative splicing and proteolytic cleavage generate soluble forms of IL-6R alpha allowing IL-6 trans-signaling by binding of IL-6/IL-6R alpha complexes to gp130-expressing cells. Thereby, the range of IL-6 responsive cells is markedly increased as gp130 is ubiquitously expressed. Both pathogen-associated molecular patterns (PAMPs) and damage-associated molecular patterns (DAMPs) induce the secretion of IL-6, which has both local and systemic effects. Next to IL-6, tumor necrosis factor (TNF)-alpha and IL-1 beta are produced, which promote further synthesis of IL-6. IL-6 is a crucial mediator of the acute



Structural model of human IL-6, His-tag

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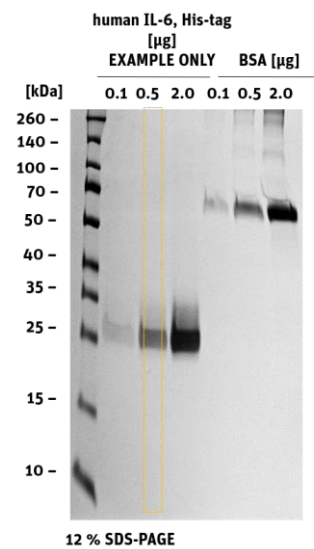
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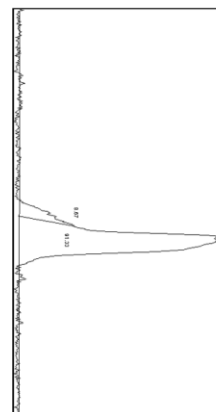
phase response and fever. It stimulates the synthesis of acute phase reactants by hepatocytes, such as the C-reactive protein (CRP), serum amyloid A (SAA) and fibrinogen, and inhibits production of albumin, transferrin and fibronectin. Furthermore, IL-6 secretion leads to increased production of neutrophils in the bone marrow. The differentiation of naïve CD4+ T cells into IL-17 producing helper T cells is stimulated by IL-6, whereas differentiation into regulatory T cells is concomitantly inhibited. Additionally, IL-6 promotes differentiation of activated B cells into antibody-producing plasma cells, all of which contributes to acquired immunity. However, dysregulation of IL-6

production causes several chronic inflammatory diseases and autoimmunity. Overproduction of IL-6 has been detected in the synovial cells of rheumatoid arthritis, swollen lymph nodes of the Castleman's disease, myeloma cells, and peripheral blood cells. With tocilizumab, a humanized monoclonal antibody, an effective therapeutic drug targeting IL-6 signaling cascades has been developed to treat immune-mediated diseases. In contrast to its pro-inflammatory properties, IL-6 also acts as an anti-inflammatory myokine, which is a cytokine produced by contracting muscle cells.

Quality Information (provided for each lot):



SDS-PAGE/Coll.Coomassie



	Area	Percent
1	1294.719	8.673
2	13634.104	91.327

Histogram (of marked lane in gel picture)