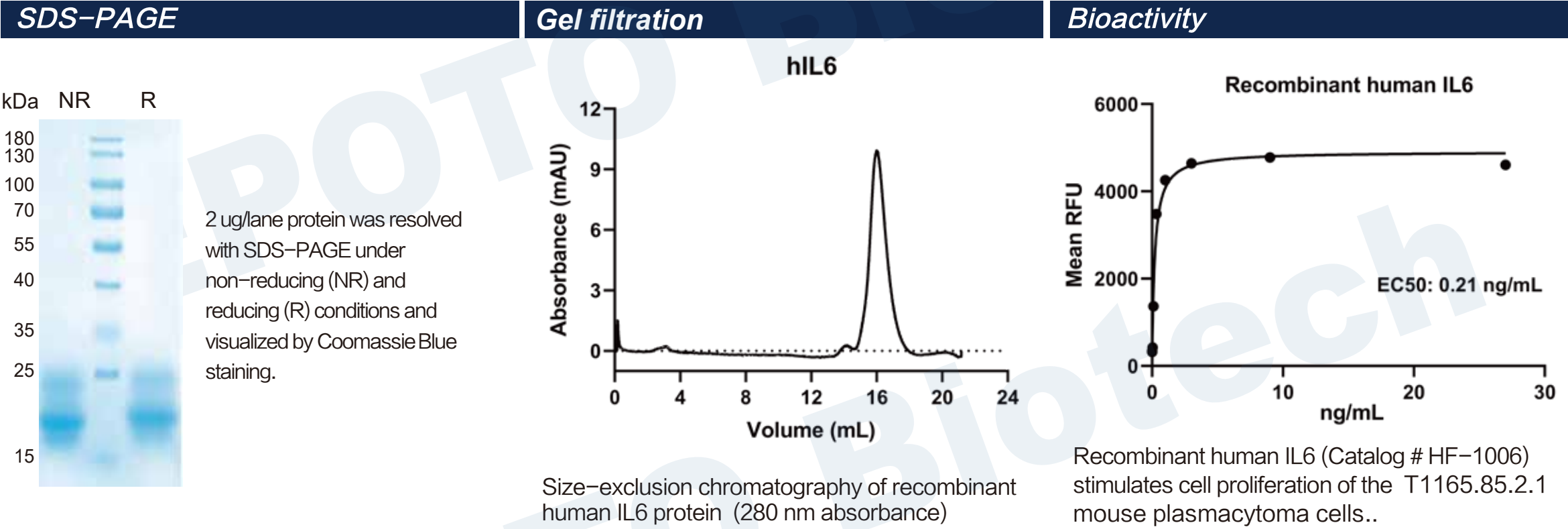


General Information	
Synonyms	IL6, B-cell differentiation factor, B-cell stimulatory factor 2, BSF2, CDF, CTL differentiation factor
Accession #	Q75MH2
Source	Human embryonic kidney cell, HEK293-derived human IL-6 protein
	Pro29-Met212
Predicted Moleucular weight	21.0 kDa
Components and Storage	
Formulation	Solution protein.
	Dissolved in sterile PBS buffer.
	This solution can be diluted into other aqueous buffers. Centrifuge the vial prior to opening.
Storage and Stability	Avoid repeated freeze-thaw cycles.
	It is recommended that the protein be aliquoted for optimal storage.
	12 months from date of receipt, -20 to -70 °C as supplied.
Shipping	Shipping with dry ice.
Quality	
Purity	> 95%, determined by SDS-PAGE.
Endotoxin Level	<0.010 EU per 1 ug of the protein by the LAL method.
Activity	Measured in a cell proliferation assay using T1165.85.2.1 mouse plasmacytoma cells.
	The EC50 for this effect is 0.1-0.5 ng/mL.



Background

Interleukin-6 (IL-6) is a multifunctional α -helical cytokine that regulates cell growth and differentiation of various tissues (1, 2). Mature human IL-6 is 183 amino acids in length and shares 39% sequence identity with mouse and rat IL-6 (3). Alternative splicing generates several isoforms with internal deletions, some of which exhibit antagonistic properties (4-7). IL-6 induces signaling through a cell surface heterodimeric receptor complex composed of a ligand binding subunit (IL-6R alpha) and a signal transducing subunit (gp130). IL-6 binds to IL-6R alpha, triggering IL-6R alpha association with gp130 (8). gp130 is also a component of the receptors for CLC, CNTF, CT-1, IL-11, IL-27, LIF, and OSM (9). Soluble forms of IL-6 R alpha are generated by both alternative splicing and proteolytic cleavage (2). In a mechanism known as trans-signaling, complexes of soluble IL-6 and IL-6 R alpha elicit responses from gp130-expressing cells that lack cell surface IL-6 R alpha (2). Trans-signaling enables a wider range of cell types to respond to IL-6, as the expression of gp130 is ubiquitous, while that of IL-6 R alpha is predominantly restricted to hepatocytes, monocytes, and resting lymphocytes (2). Soluble splice forms of gp130 block trans-signaling from IL-6/IL-6 R alpha but not from other cytokines that use gp130 as a co-receptor (2, 10). IL-6, along with TNF-alpha and IL-1, drives the acute inflammatory response and the transition from acute inflammation to either acquired immunity or chronic inflammatory disease (1, 2). When dysregulated, it contributes to chronic inflammation in obesity, insulin resistance, inflammatory bowel disease, arthritis, sepsis, and atherosclerosis (1, 2, 5).

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