Epoto Biotech 南京艾璞拓生物科技有限公司

Recombinant Mouse IL-6, Tag Free

Catalog Number: MF-1006

General Information			
Synonyms	BSF2; BSF-2; CDF; CTL differentiation factor ; HSF; IFNB2	; IFN-beta-2; IL6; IL-6	
Accession #	P08505		
Source	Human embryonic kidney cell, HEK293-derived mouse IL-6	S protein	
	Phe25-Thr211		
Predicted Moleucular we	ight 21.8 kDa		
Components and Sto	prage		
Formulation	Solution protein.		
	Dissolved in sterile PBS buffer.		
	This solution can be diluted into other aqueous buffers. Centrifuge th	e 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 0.01–0.05 ng/mL.		
SDS-PAGE	Gel filtration	Bioactivity	

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Background

Interleukin-6 (IL-6) plays important roles in the acute phase reaction, inflammation, hematopoiesis, bone metabolism, and cancer progression (1 – 5). Mature mouse IL-6 is 187 amino acids (aa) in length and shares 39% and 85% aa sequence identity with human and rat IL-6, respectively (6 - 8). IL-6 induces signaling through a cell surface heterodimeric receptor complex composed of a ligand binding subunit (IL-6 R alpha) and a signal transducing subunit (gp130). IL-6 binds to IL-6 R alpha, triggering IL-6 R alpha association with gp130 and gp130 dimerization (9). Soluble forms of IL-6 R alpha are

generated by both alternative splicing and proteolytic cleavage (5). 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 (5). 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, 5). 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 - 5). When dysregulated, it contributes to chronic inflammation in obesity, insulin resistance, inflammatory bowel disease, arthritis, sepsis, and atherosclerosis (1, 2, 5). IL-6 can also function as an anti-inflammatory molecule, as in skeletal muscle where it is secreted in response to exercise (2). In addition, it enhances hematopoietic stem cell proliferation and the differentiation of Th17 cells, memory B cells, and plasma cells (1, 10). Reference

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3. Erta, M. et al. (2012) Int. J. Biol. Sci. 8:1254.		8. Van Snick, J. et al. (1988) Eur. J. Immunol. 18:193.	
4. Garbers, C. et al. (2012) Cytokine Growth Factor Rev. 23:85.		9. Murakami, M. et al. (1993) Science 260:1808.	
5. Mihara, M. et al. (2012) Clin. Sci. (Lond.) 122:143.		10. Cerutti, A. et al. (1998) J. Immunol. 160:2145.	

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