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Recombinant Mouse IL-17A, Tag Free

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Catalog Number: MF-1017A

General Information		
Synonyms	IL17; IL-17; IL17A; IL-17A; CTLA8; CTLA-8; Cytotoxic T-lymphocyte-associated antigen 8	
Accession #	Q62386	
Source	Irce Human embryonic kidney cell, HEK293-derived mouse IL-17/IL-17A protein	
	Ala26-Ala158	
Predicted Moleucular w	eight 15.0 kDa	
Components and St	orage	
Formulation	on 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	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 by its ability to induce IL-6 secretion by NIH-3T3 mouse embryonic fibroblast cells.	
	The EC50 for this effect is 0 12–1 25 ng/ml	

SDS-PAGE

Bioactivity





Recombinant mouse IL–17A (Catalog # MF–1017A) induces IL–6 secretion by NIH–3T3 mouse embryonic fibroblast cells.

Background

Interleukin–17A(IL–17A), also known as CTLA–8, is a 15–20 kDa glycosylated cytokine that plays an important role in anti–microbial and chronic inflammation. The six IL–17 cytokines (IL–17A–F) are encoded by separate genes but adopt a conserved cystine knot fold (1, 2). Mature mouse IL–17A shares 61% and 89% amino acid sequence identity with human and rat IL–17A, respectively (3, 4). IL–17A is secreted by Th17 cells, gamma / δ T cells, iNKT cells, NK cells, LTi cells, neutrophils, and intestinal Paneth cells (2). It forms disulfide–linked homodimers as well as disulfide–linked heterodimers with IL–17F (5, 6). IL–17A exerts its effects through the transmembrane IL–17RA in complex with IL–17RC or IL–17RD (7, 8). Both IL–17RA and IL–17RC are required for responsiveness to heterodimeric IL–17A/F (7). IL–17A promotes protective mucosal and epidermal inflammation in response to microbial

infection (9–12). IL–17A/F likewise induces neutrophil migration, but IL–17F does not (11). IL–17A additionally enhances the production of inflammatory mediators by rheumatoid synovial fibroblasts and contributes to TNF–alpha induced shock (Fossiez, 14). In contrast, it can protect against the progression of colitis by limiting chronic inflammation (12). IL–17A encourages the formation of autoreactive germinal centers and exacerbates the onset and progression of experimental models of autoimmunity (15,16). IL–17A has been shown to exert either tumorigenic or anti–tumor effects (17, 18).

Reference

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