Datasheet

Cat. No. PM2013

Recombinant Mouse IL-17A, Tag Free

Information

Accession #	Q62386
Alternate Names	IL17; IL-17; IL17A; IL-17A; CTLA8; CTLA-8; Cytotoxic T-lymphocyte-associated antigen 8
Source	Human embryonic kidney cell, HEK293-derived mouse IL-17/IL-17A protein
Protein sequence	Ala26-Ala158
M.Wt	15.0 kDa
Appearance	Solution protein
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. 3 years from date of receipt, -20 to -70°C as supplied.
Concentration	0. 2 mg/mL
Formulation	Dissolved in sterile PBS buffer.
Reconstitution	We recommend that this vial be briefly centrifuged prior to opening to bring the contents to the bottom. This solution can be diluted into other aqueous buffers.
Biological Activity	The EC50 for this effect is 0.12-1.25 ng/mL. Measured by its ability to induce IL-6 secretion by NIH-3T3 mouse embryonic fibroblast cells.
Shipping Condition	Shipping with dry ice.
Handling	Centrifuge the vial prior to opening.
Usage	For Research Use Only! Not to be used in humans.

Quality Control

Purity	> 95%, determined by SDS-PAGE.
Endotoxin	<0.010 EU per 1 ug of the protein by the LAL method.

Description

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 [13, 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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