Purified Anti-Mouse IL-17A Antibody[TC11-18H10.1]

catalog number: **E-AB-F1199A**

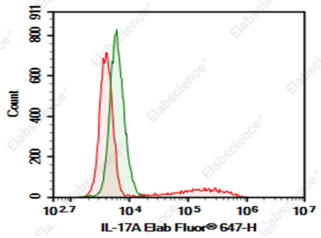
Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

Description

Reactivity	Mouse
Immunogen	Recombinant Mouse IL-17A protein
Host	Rat
Isotype	Rat IgG1, κ
Clone	TC11-18H10.1
Purification	>98%, Protein A/G purified
Buffer	Phosphate-buffered solution, pH 7.2, containing 0.05% non-protein stabilizer. Dialyze to completely remove the stabilizer prior to labeling.

Applications	Recommended Dilution
FCM	≤ 0.2 μg per million cells in 100 μL volume

Data



HEK293T cells transfected with pcDNA3.1 plasmid encoding Mouse IL17A gene were stained with 0.2 μg Purified Anti-Mouse IL-17A Antibody[TC11-18H10.1] (Right) and 0.2 μg Rat IgG1, κ Isotype Control (Left), followed by Elab Fluor® 647-conjugated Goat Anti-Rat IgG Secondary Antibody.

Preparation & Storage

Storage	Store at 4°C valid for 12 months or -20°C valid for long term storage, avoid freeze / thaw cycles.
Shipping	Ice bag

Background

Toll-free: 1-888-852-8623
Tel: 1-832-243-6086
Fax: 1-832-243-6017
Web: www.elabscience.com
Email: techsupport@elabscience.com

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. Mature rat IL-17A shares 60% and 89% amino acid sequence identity with human and mouse IL-17A, respectively. IL-17A is secreted by Th17 cells, gamma /δ T cells, iNKT cells, NK cells, LTi cells, neutrophils, and intestinal Paneth cells. It forms disulfide-linked homodimers as well as disulfide-linked heterodimers with IL-17F. IL-17A exerts its effects through the transmembrane IL-17RA in complex with IL-17RC or IL-17RD. Both IL-17RA and IL-17RC are required for responsiveness to heterodimeric IL-17A/F. IL-17A promotes protective mucosal and epidermal inflammation in response to microbial infection. It induces chemokine production, neutrophil influx, and the production of antibacterial peptides. IL-17A/F likewise induces neutrophil migration, but IL-17F does not. IL-17A additionally enhances the production of inflammatory mediators by rheumatoid synovial fibroblasts and contributes to TNF-alpha induced shock. In contrast, it can protect against the progression of colitis by limiting chronic inflammation. IL-17A encourages the formation of autoreactive germinal centers and exacerbates the onset and progression of experimental models of autoimmunity. IL-17A has been shown to exert either tumorigenic or anti-tumor effects.

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