Elabscience®

Recombinant Human IL-19 protein (His Tag)

Catalog Number: PDMH100393

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

Description	
Species	Human
Source	HEK293 Cells-derived Human IL-19 protein Met1-Ala177, with an C-terminal His
Calculated MW	19.4 kDa
Observed MW	30 kDa
Accession	Q9UHD0
Bio-activity	Not validated for activity
Properties	
Purity	> 95% as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU/mg of the protein as determined by the LAL method
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80
	°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at ≤ 20 °C for 3 months
Shinning	This product is provided as wonhilized powder which is shipped with ice packs
	Lyan hiliard from a 0.2 um filtered solution in DDS with 50/ Trabalase and 50/
Formulation	Lyophilized from a 0.2 µm liftered solution in PBS with 5% Frenalose and 5%
	Mannitol.
Reconstitution	It is recommended that sterile water be added to the vial to prepare a stock solution of
	0.5 mg/mL. Concentration is measured by UV-Vis.

Data



> 95 % as determined by reducing SDS-PAGE.

Background

The molecular features at the IL19 locus may modestly alter the establishment of HIV-1 infection. Interleukin (IL) 19, IL-20, and IL-24 belong to the IL-10 cytokine family and have been identified to play a role in the regulation of epidermal functions and inflammation. The expression of IL19 in biopsies of patients with active ulcerative colitis was increased compared with patients with quiescent ulcerative colitis and that colitis was attenuated in IL-19-deficient mice. The disruption of the epithelial barrier with dextran sodium sulfate leads to increased IL-19 expression. Attenuated colitis in IL-19-deficient animals was associated with reduced numbers of IL-6-producing macrophages in the inflamed colonic lamina propria. Microbial-driven expression of IL-19 by intestinal macrophages may contribute to the pathogenesis of inflammatory bowel disease.