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Zuschläge

- Mindermengenzuschlag
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Recombinant Human IL-19 protein (His tag)

Catalog Number:PDMH100076



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

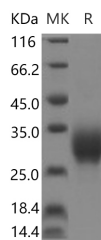
Description

Synonyms	IL-10C;Interleukin-19;MDA1;NG.1;ZMDA1
Species	Human
Expression Host	HEK293 Cells
Sequence	Met1-Ala177
Accession	Q9UHD0
Calculated Molecular Weight	19.4 kDa
Observed molecular weight	30 kDa
Tag	C-His

Properties

Purity	> 95 % as determined by reducing SDS-PAGE.
Endotoxin	Please contact us for more information.
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.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from sterile PBS, pH 7.4. Normally 5 % - 8 % trehalose, mannitol and 0.01 % Tween80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
Reconstitution	Please refer to the printed manual for detailed information.

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.

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