

**DESCRIPTION**

**Source** *E. coli*-derived  
Val17-Ala169, with and without an N-terminal Met  
Accession # NP\_542767

**N-terminal Sequence Analysis** Val17 and Met

**Structure / Form** Disulfide-linked homodimer

**Predicted Molecular Mass** 17.6 kDa (monomer)

**SPECIFICATIONS**

**Activity** Measured by its ability to induce CXCL1/GRO $\alpha$  secretion in HT-29 human colon adenocarcinoma cells.  
The ED<sub>50</sub> for this effect is 0.25-1.5 ng/mL.

**Endotoxin Level** <0.01 EU per 1  $\mu$ g of the protein by the LAL method.

**Purity** >97%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.

**Formulation** Lyophilized from a 0.2  $\mu$ m filtered solution in Acetonitrile and TFA. See Certificate of Analysis for details.

**PREPARATION AND STORAGE**

**Reconstitution** Reconstitute at 100  $\mu$ g/mL in sterile 4 mM HCl.

**Shipping** The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

**Stability & Storage** Use a manual defrost freezer and avoid repeated freeze-thaw cycles.

- 12 months from date of receipt, -20 to -70 °C as supplied.
- 1 month, 2 to 8 °C under sterile conditions after reconstitution.
- 3 months, -20 to -70 °C under sterile conditions after reconstitution.

**BACKGROUND**

The Interleukin-17 (IL-17) family of proteins are immunoregulatory cytokines that share a conserved cysteine-rich region. IL-17E, which is also known as IL-25, promotes Th2-biased immune responses. This is in contrast to other IL-17 family members which promote Th1- and Th17-biased inflammation. IL-25 is an important mediator of allergic reactions and protection against intestinal parasites (1, 2). Mature mouse IL-25 shares 80% and 91% amino acid sequence identity with human and rat IL-25, respectively (3, 4). During helminth infections and allergic reactions, IL-25 is locally up-regulated in intestinal and airway epithelial cells, atopic dermatitis skin lesions, and local Th2 cells, eosinophils, and basophils (4-9). It binds to IL-17 RB but also requires IL-17 RA to exert its activity (3, 8, 10). IL-25 acts on a variety of cell types which respond with increased production of Th2 cytokines (e.g. IL-4, IL-5, IL-13) and reduced production of Th1 and Th17 cytokines (e.g. IFN- $\gamma$ , IL-12, IL-23, IL-17A, IL-17F) (4-6, 8, 9, 11-15). Airway IL-25 can be activated by MMP-7, a protease that is up-regulated in airway epithelium in response to allergen exposure (16). Cleaved IL-25 shows enhanced binding to IL-17 RB and stronger induction of Th2 cytokines (16). The Th2 cytokines, in turn, trigger expansion of Th2 memory cells and anti-inflammatory M2 macrophages, increased eosinophil mobilization and activation, and dendritic cell migration (4, 6, 9, 13). These actions promote protective anti-helminth immune responses (4, 5) as well as allergic inflammation and airway hyperreactivity (11). The IL-25 induced suppression of Th1 and Th17 cytokines limits Th17 cell expansion and disease pathology in autoimmunity and colitis (12, 15). IL-25 also promotes vascular endothelial cell proliferation and assembly into tubular structures (7). It supports the integrity of the blood-brain barrier and limits CD4<sup>+</sup> T cell infiltration into the brain (17).

**References:**

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