**DESCRIPTION**

**Source**  
E. coli-derived  
Ser2-Asp192  
Accession # Q9NZH6-2

**N-terminal Sequence Analysis**  
Ser2

**Predicted Molecular Mass**  
21 kDa

**SPECIFICATIONS**

<table>
<thead>
<tr>
<th>SDS-PAGE</th>
<th>22 kDa, reducing conditions</th>
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| Activity | Measured by its ability to inhibit LPS-stimulated IL-8 secretion by HK-2 human kidney epithelial cells.  
The ED_{50} for this effect is 0.25-1.25 μg/mL. |
| Endotoxin Level | <0.10 EU per 1 μg of the protein by the LAL method. |
| Purity | >95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining. |
| Formulation | Lyophilized from a 0.2 μm filtered solution in PBS and DTT with BSA as a carrier protein. See Certificate of Analysis for details. |

**PREPARATION AND STORAGE**

**Reconstitution**  
Reconstitute at 100 μg/mL in PBS.

**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.

**DATA**

**Bioactivity**  
Recombinant Human IL-37/IL-1F7 (Catalog # 9225-IL) inhibits LPS-stimulated IL-8 secretion by HK-2 human kidney epithelial cells. The ED_{50} for this effect is 0.25-1.25 μg/mL.

Recombinant Human IL-37/IL-1F7 (μg/mL)
Human Interleukin-1 family member 7 (IL-1F7), also named FIL-1 zeta, IL-37, IL-1H4, IL-1HL and IL-1RP1, is an anti-inflammatory member of the IL-1 cytokine family (1-3). Alternative splicing generates multiple isoforms (IL-37a through e) with deletions in the N-terminal region of the molecule (4-7). IL-37b shares approximately 21%, 24%, and 30% aa sequence identity with mature IL-1 alpha, IL-1 beta, and IL-1ra, respectively. Mouse IL-37 has not been reported, but human IL-37b is active on mouse cells. Like IL-1 alpha, IL-1 beta and IL-18, all of the IL-37 variants lack a typical signal peptide. IL-37b is up-regulated by inflammatory stimuli in peripheral blood mononuclear cells (8). Experimental over-expression of IL-37b in vivo limits the inflammatory response and protects mice from colitis and LPS-induced shock (8, 9). Both unprocessed and mature IL-37b can form homodimers in solution (8, 10). Although IL-37b will bind to IL-18 R alpha with low affinity, this has no effect on IL-18 receptor activity (7, 10, 11). Alternatively, IL-37b will also bind to IL-18 BP, generating a complex that interacts with IL-18 R beta. This has the effect of attenuating IL-18 activity via the IL-18 receptor (11). IL-37b can also function intracellularly. Following LPS induced cleavage of its propeptide, IL-37b associates with Smad3 and translocates to the nucleus. This results in a reduction of pro-inflammatory cytokine secretion (12).

References: