

**DESCRIPTION**

<b>Source</b>	Mouse myeloma cell line, NS0-derived Ala26-Val481 with a C-terminal 6-His tag Accession # CAA67226
<b>N-terminal Sequence Analysis</b>	Ala26
<b>Structure / Form</b>	Monomer
<b>Predicted Molecular Mass</b>	51.7 kDa

**SPECIFICATIONS**

<b>SDS-PAGE</b>	60-65 kDa, reducing conditions
<b>Activity</b>	Measured by its ability to enhance LPS-stimulated IL-8 secretion by THP-1 human acute monocytic leukemia cells. The ED <sub>50</sub> for this effect is 0.25-1.5 ng/mL.
<b>Endotoxin Level</b>	<0.10 EU per 1 µg of the protein by the LAL method.
<b>Purity</b>	>95%, by SDS-PAGE under reducing conditions and visualized by silver stain.
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

**PREPARATION AND STORAGE**

<b>Reconstitution</b>	Reconstitute at 100 µg/mL in PBS.
<b>Shipping</b>	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
<b>Stability &amp; Storage</b>	<p><b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b></p> <ul style="list-style-type: none"> <li>● 12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>● 1 month, 2 to 8 °C under sterile conditions after reconstitution.</li> <li>● 3 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

**BACKGROUND**

LBP (Lipopolysaccharide binding protein) is a 58 - 62 kDa, glycoprotein member of the BPI/LBP family of lipid-binding proteins (1 - 4). It is secreted by hepatocytes, gingival keratinocytes, intestinal paneth cells, type II Greater alveolar cells, lacrimal and submandibular gland epithelium, and caudal epididymal epithelium (5 - 10). LBP is a class 1 APR (acute phase reactant) that is induced upon exposure to both IL-1 and IL-6 (11). Following its synthesis and release, LBP interacts with bacterial wall components, lipopolysaccharide/LPS/Lipid A from Gram- (Gm-) bacteria, and lipoteichoic acid/LTA from Gm+ bacteria (12 - 16). In the case of LPS, this interaction occurs both in the bacterial cell wall, and within the intercellular space, where LPS micelles naturally form following bacterial death and cell wall dissolution (17 - 20). LBP induces disassembly of LPS micelles, allowing for LPS binding to LBP, and a heparin-mediated transfer of LPS from LBP to membrane-bound CD14 on the surface of monocytes/macrophages (18, 21). This CD14:LPS complex activates a TLR4:MD2 membrane complex, resulting in the production of NO and TNF $\alpha$  (22). TNF $\alpha$  serves as a chemoattractant for PMNs, and an initiator of coagulation that helps to wall-off and localize microbial elements (19). In addition to the above, LBP is also reported to transfer LPS to lipoproteins, particularly HDL and LDL (22 - 25). For LDL, this transfer inhibits monocyte activation; for HDL, the effect may be either stimulatory or inhibitory, depending upon the circumstances (22). Human LBP is synthesized as a 481 amino acids (aa) precursor that contains a 25 aa signal sequence and a 456 aa mature region (aa 26 - 481) (26). It contains an N-terminal LPS binding region (aa 29 - 252) plus a likely C-terminal LPS transfer region (aa 276 - 477) (26, 27). There are two potential splice variants. One shows a four aa substitution for aa 154 - 157 coupled to an Ala substitution for aa 266 - 270, while another contains a seven aa substitution for aa 468 - 481. Mature human LBP shares 68% and 69% aa identity with mouse and rat LBP, respectively (14, 22).

**References:**

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