

**DESCRIPTION**

**Source** Mouse myeloma cell line, NS0-derived  
Gly25-Val481 (Ser102Arg, Tyr284His), with a C-terminal 6-His tag  
Accession # NP\_032515

**N-terminal Sequence Analysis** Gly25

**Predicted Molecular Mass** 51.5 kDa

**SPECIFICATIONS**

**SDS-PAGE** 60-65 kDa, reducing conditions

**Activity** 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 0.5-3 ng/mL.

**Endotoxin Level** <0.10 EU per 1 µg of the protein by the LAL method.

**Purity** >95%, by SDS-PAGE under reducing conditions and visualized by silver stain.

**Formulation** Lyophilized from a 0.2 µm filtered solution in MES, NaCl, PEG and CHAPS. 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.

**BACKGROUND**

LBP (Lipopolysaccharide binding protein) is a 58 - 62 kDa, single-chain glycoprotein member of the BPI/LBP family, BPI/PLUNC/PSP superfamily of lipid-binding proteins (1 - 3). It is secreted by a number of mammalian cell types, including hepatocytes (4), gingival keratinocytes (5), intestinal Paneth cells (6), and type II Greater alveolar cells (7). LBP is considered to be a class 1 APR (acute phase reactant) that is induced upon exposure to both IL-1 and IL-6 (8). These two cytokines appear upon immune cell exposure to pathogenic microbes. Following its synthesis and release, LBP is known to interact with bacterial wall components, lipopolysaccharide/LPS/Lipid A from Gram<sup>-</sup> (Gm<sup>-</sup>) bacteria, and lipoteichoic acid/LTA from Gm<sup>+</sup> bacteria (9 - 13). In the case of LPS, this interaction appears to occur both in the bacterial cell wall, and within the intercellular space, where LPS micelles naturally form following bacterial death and cell wall dissolution (14 - 17). LBP is posited to induce 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 (15, 18). This CD14:LPS complex activates a TLR4:MD2 membrane complex, resulting in the production of NO and TNF- $\alpha$  (19). TNF- $\alpha$  serves as a chemoattractant for PMNs, and an initiator of coagulation that helps to wall-off and localize microbial elements (16). Notably, increased concentrations of LBP are also associated with parasitic infections (Trypanosoma), and may contribute to the immune response towards parasites (20). In addition to the above, LBP is also reported to transfer LPS to lipoproteins, particularly HDL and LDL (19, 21 - 23). For LDL, this transfer appears to be inhibitory to monocyte activation; for HDL, the effect may be either stimulatory or inhibitory, depending upon the circumstances (19). Mouse 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) (24). It contains an N-terminal LPS binding region plus a likely C-terminal LPS transfer region (24 - 25). Mature mouse LBP shares 68% and 88% aa identity with human and rat LBP, respectively (11, 25).

**References:**

1. Beamer, L.J. *et al.* (1998) *Protein Sci.* **7**:906.
2. Schroder, N.W.J. & R.R. Schumann (2005) *J. Endotoxin Res.* **11**:237.
3. Miyake, K. (2006) *J. Endotoxin Res.* **12**:195.
4. Grube, B.J. *et al.* (1994) *J. Biol. Chem.* **269**:8477.
5. Ren, L. *et al.* (2004) *J. Periodont. Res.* **39**:242.
6. Hansen, G.H. *et al.* (2009) *Histochem. Cell Biol.* **131**:727.
7. Dentener, M.A. *et al.* (2000) *Am. J. Respir. Cell Mol. Biol.* **23**:146.
8. Schumann, R.R. *et al.* (1996) *Mol. Cell. Biol.* **16**:3490.
9. Weber, J.R. *et al.* (2003) *Immunity* **19**:269.
10. Schroder, N.W.J. *et al.* (2004) *J. Immunol.* **173**:2683.
11. Su, G.L. *et al.* (1994) *J. Immunol.* **153**:743.
12. Schroder, N.W.J. *et al.* (2003) *J. Biol. Chem.* **178**:15587.
13. Wright, S.D. *et al.* (1989) *J. Exp. Med.* **170**:1231.
14. Hallatschek, W. *et al.* (2004) *Eur. J. Immunol.* **34**:1441.
15. Schumann, R.R. & E. Latz (2000) *Chem. Immunol.* **74**:42.
16. Mannel, D.N. & B. Echtenacher (2000) *Chem. Immunol.* **74**:141.
17. Tsukamoto, H. *et al.* (2010) *Int. Immunol.* **22**:271.
18. Heinzelmann, M. & H. Bosshart (2005) *J. Immunol.* **174**:2280.
19. Gallay, P. *et al.* (1993) *Infect. Immun.* **61**:378.
20. Ngure, R.M. *et al.* (2009) *Res. Vet. Sci.* **86**:394.
21. Levels, J.H.M. *et al.* (2005) *Infect. Immun.* **73**:2321.
22. Hubacek, J.A. *et al.* (1997) *Biochem. Biophys. Res. Commun.* **236**:427.
23. Thompson, P.A. & R.L. Kitchens (2006) *J. Immunol.* **177**:4880.
24. Lengacher, S. *et al.* (1995 - 1996) *J. Inflamm.* **47**:165.
25. Schumann, R.R. *et al.* (1990) *Science* **249**:1429.