

DESCRIPTION

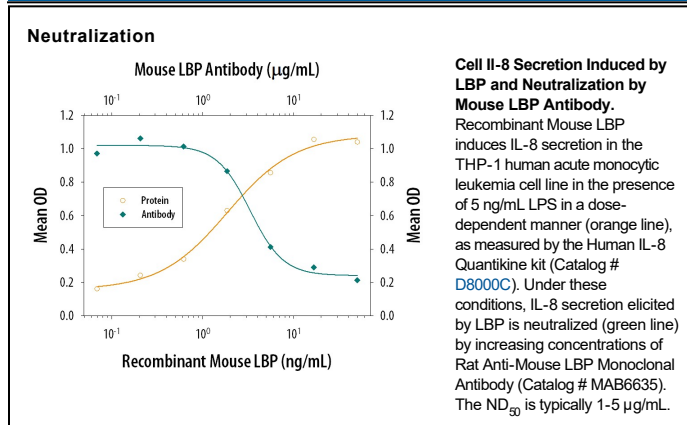
Species Reactivity	Mouse
Specificity	Detects mouse LBP in direct ELISAs. In direct ELISAs, no cross-reactivity with recombinant human LBP is observed.
Source	Monoclonal Rat IgG ₁ Clone # 749405
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse LBP Gly25-Val481 (Ser102Arg, Tyr284His) Accession # Q61805
Endotoxin Level	<0.10 EU per 1 µg of the antibody by the LAL method.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 µm filtered solution in PBS.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

Neutralization	Measured by its ability to neutralize LBP-induced IL-8 secretion in the THP-1 human acute monocytic leukemia cell line treated with LPS. The Neutralization Dose (ND ₅₀) is typically 1-5 µg/mL in the presence of 50 ng/ml Recombinant Mouse LBP.
-----------------------	--

DATA



PREPARATION AND STORAGE

Reconstitution	Sterile PBS to a final concentration of 0.5 mg/mL.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 6 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⁻ (Gm⁻) bacteria, and lipoteichoic acid/LTA from Gm⁺ 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- α (19). TNF- α 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.