Mouse MBL-2 Antibody

Monoclonal Rat IgG_{2A} Clone # 272801

Catalog Number: MAB2208

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse MBL-2 in direct ELISAs and Western blots. In Western blots, no cross-reactivity with recombinant human MBL is observed.
Source	Monoclonal Rat IgG_{2A} Clone # 272801
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse MBL-2 Glu19-Asp244 Accession # P41317
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.*

	Recommended Concentration	Sample
Western Blot	1 µg/mL	Recombinant Mouse MBL-2 (Catalog # 2208-MB)

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 mg/mL in sterile PBS.
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.** ● 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

Mannan binding lectin (MBL) belongs to the collectin family of innate immune defense proteins, which binds to an array of carbohydrate patterns on pathogen surfaces (1, 2). Collectin family members share common structural features: a cysteine rich amino-terminal domain, a collagen-like region, an α-helical coiled-coil neck domain and a carboxy terminal C-type (Ca^{++}-dependent) lectin or carbohydrate recognition domain (CRD). MBL homotrimerizes to form a structural unit joined by N-terminal disulfide bridges. These homotrimers further associates into oligomeric structures of up to 6 units. Whereas two forms of MBL proteins (MBL-1, also known as S-MBP or MBL-A and MBL-2, also known as L-MBP or MBL-C) exist in rodents and other animals, only one functional MBL protein is present in humans. Mouse MBL-2 shares approximately 52% and 60% aa sequence identity with mouse MBL-1 and human MBL, respectively. In mouse, MBL-1 and MBL-2 are the only collectins that can activate complement via the lectin complement pathway (1, 2). Serum oligomeric MBL associates with MBL-associated serine protease (MASP) proenzymes. The MBL-MASP proenzyme complex preferentially interact with sugar patterns containing mannose, glucose, L-fucose, or N-acetyl-glucosamine present at a terminal nonreducing postion on the cell surface of various pathogens and certain tumor cells. This interaction induces pro-enzyme activation and the triggering of the complement cascade, resulting in opsonization and pathogen removal via humoral and cellular immune responses. MBL does not recognize self-components or glycoproteins from other higher animals due to the presence of terminal sialic acid or galactose that interrupts the repeating carbohydrate structures (3). A number of membrane receptors for MBL, including C1q phagocytic receptor (C1qRp), calreticulin (also known as C1qR), and CR1 (CD35), have been described. Interactions with these receptors may also be important in stimulating phagocytosis (1, 2). Mouse MBL-1 and MBL-2 are produced primarily in the liver and are secreted into the blood stream. In addition, mouse MBL-1 is also expressed in lung, kidney, and testis while MBL-2 is expressed in kidney, thymus, and small intestine (1, 4, 5).

References:

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2. Fujita, R. *et al.* (2004) Immunol. Rev. **198**:185.
3. Saevarsdottir, S. *et al.* (2004) Scand. J. Immnunol. **60**:23.
4. Uemura, K. *et al.* (2002) J. Immunol. **169**:6945.
5. Wagner, S. *et al.* (2003) J. Immunol. **170**:1462.

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