

Mouse anti-TLR2, clone T2.5 (Monoclonal)

Clone no.      T2.5

MONOSAN

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Product name	Mouse anti-TLR2, clone T2.5 (Monoclonal)
Host	Mouse
Applications	IHC-fr,FC,FUNC,ELISA,IF,IP
Species reactivity	human, mouse
Conjugate	-
Immunogen	Unknown or proprietary to MONOSAN and/or its suppliers
Isotype	IgG1
Clonality	Monoclonal
Clone number	T2.5
Size	1 ml
Concentration	100 ug/ ml
Format	-
Storage buffer	PBS with 0.1% BSA and 0.02% sodium azide
Storage until expiry date	2-8°C

FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC PROCEDURES

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**Additional info**

The monoclonal antibody T2.5 recognizes human Toll-like receptor 2 (TLR2). Toll-like receptors (TLR) are highly conserved throughout evolution and have been implicated in the innate defense to many pathogens. At present, ligands for several of the TLR's, such as TLR2-6,9, have been identified, confirming their role in first line defense against invading microorganism. In mammals, TLRs are identified as type I transmembrane signaling receptors with an extracellular portion containing leucine-rich repeats with pattern recognition capabilities. Pathogen recognition by TLRs provokes rapid activation of innate immunity by inducing proliferation of proinflammatory cytokines and upregulation of costimulatory molecules and eventually to initiation of adaptive immunity. TLR2 has been identified as a receptor that is central to the innate immune response to lipoproteins of Gram-negative bacteria, several whole Gram-positive bacteria, as well as a receptor for peptidoglycan and lipoteichoic acid and other bacterial cell membrane products. It is suggested that TLR2 is able to recognize such a wide variety of PAMPs (pathogen-specific molecular patterns) by forming heterodimers with other TLRs like e.g. TLR6. TLR2 is essential for recognizing lipopeptides and lipoproteins from several microorganisms and also peptidoglycans derived from gram-positive bacteria. Bacterial species as diverse as mycobacteria, spirochetes, mycoplasma, Staphylococcus aureus, and Streptococcus pneumoniae have all been shown to mediate cellular activation via TLR2.

**References**

1. Meng; G et al. J Clin Invest 2004; 113: 1473
2. Roura-Mir, C et al J Immunol 2005, 175: 1758
3. Leemans; J et al. J Clin Invest 2005 115: 2894
4. Spiller S et al. JEM 2008; 205: 1747
5. Suttmuller R et al. J Clin Invest 2006; 116:485

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