

Mouse anti-CD120b, TNF-RII (p75/p80), clone 80M2 (Monoclonal)

Clone no. 80M2

MONOSAN

Product name	Mouse anti-CD120b, TNF-RII (p75/p80), clone 80M2 (Monoclonal)
Host	Mouse
Applications	FC, FUNC, ELISA, IF, IP
Species reactivity	human, rat
Conjugate	-
Immunogen	Unknown or proprietary to MONOSAN and/or its suppliers
Isotype	IgG1
Clonality	Monoclonal
Clone number	80M2
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 80M2 recognizes the extracellular part of membrane-bound TNF-RII as well as the soluble form of TNF-RII which is generated by proteolytic cleavage of the extracellular domain. The soluble form can still bind TNF-alpha with high affinity and functions as a TNF-alpha antagonist. TNF-alpha is an important signaling protein in the immune system which can activate inflammatory responses, induce apoptosis, regulate cellular proliferation, and may even promote cancer progression. TNF-alpha can bind to two structurally distinct membrane receptors, TNF-RI and TNF-RII, which have both distinct and overlapping downstream signaling cascades. TNFRI is believed to be expressed on nearly all cell types, whereas TNFRII exhibits more restricted expression, being found on certain subpopulations of immune cells and several other cell types. A dominant role of TNF-RII has been shown in thymocyte activation by TNF-alpha, whereas induction of cytotoxicity and other functions are mediated largely by TNF-RI. TNF-RI is equally well activated by both the 17 kDa soluble and 26 kDa membrane-bound form, whereas TNF-RII is activated only by the membrane bound form of TNF-alpha. The antibody is a non-agonistic receptor modulating antibody. It enhances in vitro TNF alpha responses by increasing the affinity of the soluble form of TNF-alpha for TNF-RII.

References

1. Grell; M et al. Cell 1995; 83: 793
2. Till, A et al J Biol Chem 2005, 280: 5994
3. Scherubl C Journal of Inflammation 2005; 2: 7
4. Krippner A et al. J Immunol 2008; 180: 8176
5. Fairfax B et al. J Immunol 2011; 186: 3058

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