Rat anti-mouse TNF-RII (p75/p80), clone HM102 (Monoclonal)

Clone no. HM102

MONOSAN

Product name	Rat anti-mouse TNF-RII (p75/p80), clone HM102 (Monoclonal)
Host	Rat
Applications	IHC-fr, FC, FUNC, ELISA, IP, WB
Species reactivity	mouse
Conjugate	-
Immunogen	Unknown or proprietery to MONOSAN and/or its suppliers
Isotype	IgG2a
Clonality	Monoclonal
Clone number	HM102
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 HM102 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 signalling 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 TNFRII, 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 TNFRII 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 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

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2. Brekke et al. J of Lipid res 1999;40:2223
3. Marchetti et al. J Biol Chem 2004; 279:32869
4. Taoufik et al. J Neurosci 2007;27:6633
5. Veroni et al. Mol Cel Neurosci 2010;45:234