Product datasheet MON9058

Mouse anti-Tumor Necrosis Factor Receptor, clone H398 (Monoclonal)

Clone no. H398

MONOSAN

Product name	Mouse anti-Tumor Necrosis Factor Receptor, clone H398 (Monoclonal)
Host	Mouse
Applications	IHC-fr,FC,FUNC,ELISA,IP,WB
Species reactivity	human, rat
Conjugate	-
Immunogen	Unknown or proprietery to MONOSAN and/or its suppliers
Isotype	IgG2a
Clonality	Monoclonal
Clone number	H398
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

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Additional info

The monoclonal antibody H398 recognizes the extracellular part of the Tumor Necrosis Factor Receptor type I (TNF-RI) of the membrane-bound as well as the soluble receptor. TNF-RI (~55-60 kDa) is present on most cell types and is considered to play a prominent role in cell stimulation by TNF-alpha. TNF-alpha activates inflammatory responses, induces apoptosis, regulates cellular proliferation, and may even promote cancer progression. The effects of TNF-alpha are mediated by TNF-RI and TNF-RII, which have both distinct and overlapping downstream signaling cascades. Induction of cytotoxicity and other functions are mediated largely via TNF-RI. TNF-RI is equally well activated by both the 17 kDa soluble and 26 kDa membrane-bound form, whereas TNF-RII is efficiently activated only by the membrane bound form of TNF-alpha. TNF-RI signaling is initiated when trimeric TNF-alpha binds TNF-RI receptors. Subsequent TNF-RI trimerization promotes the recruitment of a proximal signaling complex composed of TNF Receptor Associated protein with a Death Domain (TRADD), Receptor Interacting Protein (RIP), cellular Inhibitor of Apoptosis Protein 1 (cIAP1), TNF Receptor Associated Factor 2 (TRAF2), and likely TRAF5. Studies with TNF-RI-deficient mice indicate that TNF-RI mediates most of the proliferation, pro-inflammatory, and apoptosis-activating pathways.

References

1. Thoma; B et al. J Exp Med 1990; 172: 1019
2. Grell, M et al Lymphokine Cytokine Res 1993, 12: 143
3. Scheurich; P et al. Tumor Necrosis factor 1993; 4: 52
4. Grell M et al. Proc Natl Acad Sci USA 1998; 95: 570
5. Krippner-Heidenreich A et al. J Immunol 2008; 180: 8176

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