Mouse TNF RII/TNFRSF1B Alexa Fluor® 532-conjugated Antibody

Monoclonal Hamster IgG Clone # TR75-89.29
Catalog Number: FAB4262X
100 µg

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse TNF RII/TNFRSF1B in direct ELISAs and Western blots.
Source	Monoclonal Hamster IgG Clone # TR75-89.29
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	*E. coli*-derived recombinant mouse TNF RII/TNFRSF1B
Conjugate	Alexa Fluor 532 Excitation Wavelength: 534 nm Emission Wavelength: 553 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

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.*

Western Blot	Optimal dilution of this antibody should be experimentally determined.
Immunoprecipitation	Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

Two types of soluble TNF receptors have been identified in human serum and urine which can neutralize the biological activities of TNF-α and TNF-β. These binding proteins represent truncated forms of the two types of high-affinity cell surface receptors for TNF (TNFR-p60 Type B and TNFR-p80 Type A). Soluble TNF RII corresponds to TNFR-p80 Type A. In the new TNF superfamily nomenclature, TNF RII is referred to as TNFRSF1B. These apparent soluble forms of the receptors appear to arise as a result of shedding of the extracellular domains of the membrane-bound receptors. Normal concentrations as high as 4 ng/mL are found in the serum of healthy individuals, and even higher levels may be found in some pathological conditions. Although the physiological role of these proteins is not known, it has been speculated that shedding of the soluble receptors in response to TNF release could serve as a mechanism to scavenge the TNF not immediately bound and thus localize the inflammatory response. It is also possible that the pool of TNF bound to soluble receptors could represent a reservoir for the controlled release of TNF.

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