

## ±11969

## Mouse TNF-α Neutralizing (D2H4) Rabbit mAb



Orders: 877-616-CELL (2355)

orders@cellsignal.com

Support: 877-678-TECH (8324)

Web: info@cellsignal.com

cellsignal.com

3 Trask Lane | Danvers | Massachusetts | 01923 | USA

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<b>Applications:</b> N	<b>Reactivity:</b> M	<b>Sensitivity:</b> Endogenous	<b>Source/Isotype:</b> Rabbit IgG	UniProt ID: #P06804	Entrez-Gene Id: 21926
Product Usage Information		CST recommends incubation of the neutralizing antibody with the intended target for 1 hr at 37°C before addition to the experiment at an optimal concentration determined by the user.			
		Reconstitution: Add sterile 10 mM HEPES pH 7.0 to a final concentration of greater than 50 $\mu$ g/ml. Solubilize for 20 min at room temperature with occasional gentle vortexing.			
		<b>Application</b> Neutralizing		<b>Dilution</b> 1:1	
Formulation		Lyophilized from a 0.2 $\mu$ m filtered solution in 10mM HEPES with trehalose.			
Storage		Store lyophilized material at -20°C. After reconstitution, recommended storage at $4^{\circ}$ C for 1 month or -20°C for 6 months. Avoid repeated freeze/thawing.			
Specificity/Sensitivity		Mouse TNF- $\alpha$ Neutralizing (D2H4) Rabbit mAb binds to mouse TNF- $\alpha$ and neutralizes its cytotoxic effects. This antibody does not cross-react with human TNF- $\alpha$ or human LTA.			
Source / Purification		Monoclonal antibody is produced by immunizing animals with a recombinant mouse TNF- $\alpha$ protein.			
Description		Neutralizing antibodies can be used to inhibit normal biological function through their binding to biological molecules. These reagents can be used to determine the effects that a particular molecule has in biological systems. Mouse TNF- $\alpha$ Neutralizing (D2H4) Rabbit mAb has been shown to neutralize the cytotoxic effects of TNF- $\alpha$ in L-929 cells <i>in vitro</i> with an ND <sub>50</sub> in the range of 1-6 ng/ml.			
Background		TNF-α, the prototypical member of the TNF protein superfamily, is a homotrimeric type-II membrane protein (1,2). Membrane-bound TNF-α is cleaved by the metalloprotease TACE/ADAM17 to generate a soluble homotrimer (2). Both membrane and soluble forms of TNF-α are biologically active. TNF-α is produced by a variety of immune cells including T cells, B cells, NK cells, and macrophages (1). Cellular response to TNF-α is mediated through interaction with receptors TNF-R1 and TNF-R2, and results in activation of pathways that favor both cell survival and apoptosis depending on the cell type and biological context. Activation of kinase pathways (including JNK, Erk (p44/42), p38 MAPK, and NF-κB) promotes the survival of cells, while TNF-α-mediated activation of caspase-8 leads to programmed cell death (1,2). TNF-α plays a key regulatory role in inflammation and host defense against bacterial infection, notably <i>Mycobacterium tuberculosis</i> (3). The role of TNF-α in autoimmunity is underscored by research studies that show that blocking TNF-α action may be used to treat rheumatoid arthritis and Crohn's disease (1,2,4).			
Background References		1. Aggarwal, B.B. (2003) <i>Nat Rev Immunol</i> 3, 745-56. 2. Hehlgans, T. and Pfeffer, K. (2005) <i>Immunology</i> 115, 1-20. 3. Lin, P.L. et al. (2007) <i>J Investig Dermatol Symp Proc</i> 12, 22-5. 4. Brennan, F.M. and McInnes, I.B. (2008) <i>J Clin Invest</i> 118, 3537-45.			
Species Reactivity		Species reactivity is dete	rmined by testing in at le	ast one approved ap	plication (e.g., western blot).

Applications Key

N: Neutralizing

**Cross-Reactivity Key** 

M: Mouse

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