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Datasheet for ABIN7173775
anti-TNFAIP3 antibody (AA 97-183) (HRP)

Overview

Quantity:	100 µg
Target:	TNFAIP3
Binding Specificity:	AA 97-183
Reactivity:	Human
Host:	Rabbit
Clonality:	Polyclonal
Conjugate:	This TNFAIP3 antibody is conjugated to HRP
Application:	ELISA

Product Details

Immunogen:	Recombinant Human Tumor necrosis factor alpha-induced protein 3 protein (97-183AA)
Isotype:	IgG
Cross-Reactivity:	Human
Purification:	>95%, Protein G purified

Target Details

Target:	TNFAIP3
Alternative Name:	TNFAIP3 (TNFAIP3 Products)
Background:	Background: Ubiquitin-editing enzyme that contains both ubiquitin ligase and deubiquitinase activities. Involved in immune and inflammatory responses signaled by cytokines, such as TNF-

alpha and IL-1 beta, or pathogens via Toll-like receptors (TLRs) through terminating NF-kappa-B activity. Essential component of a ubiquitin-editing protein complex, comprising also RNF11, ITCH and TAX1BP1, that ensures the transient nature of inflammatory signaling pathways. In cooperation with TAX1BP1 promotes disassembly of E2-E3 ubiquitin protein ligase complexes in IL-1R and TNFR-1 pathways, affected are at least E3 ligases TRAF6, TRAF2 and BIRC2, and E2 ubiquitin-conjugating enzymes UBE2N and UBE2D3. In cooperation with TAX1BP1 promotes ubiquitination of UBE2N and proteasomal degradation of UBE2N and UBE2D3. Upon TNF stimulation, deubiquitinates 'Lys-63'-polyubiquitin chains on RIPK1 and catalyzes the formation of 'Lys-48'-polyubiquitin chains. This leads to RIPK1 proteasomal degradation and consequently termination of the TNF- or LPS-mediated activation of NF-kappa-B. Deubiquitinates TRAF6 probably acting on 'Lys-63'-linked polyubiquitin. Upon T-cell receptor (TCR)-mediated T-cell activation, deubiquitinates 'Lys-63'-polyubiquitin chains on MALT1 thereby mediating disassociation of the CBM (CARD11:BCL10:MALT1) and IKK complexes and preventing sustained IKK activation. Deubiquitinates NEMO/IKBKG, the function is facilitated by TNIP1 and leads to inhibition of NF-kappa-B activation. Upon stimulation by bacterial peptidoglycans, probably deubiquitinates RIPK2. Can also inhibit I-kappa-B-kinase (IKK) through a non-catalytic mechanism which involves polyubiquitin, polyubiquitin promotes association with IKBKG and prevents IKK MAP3K7-mediated phosphorylation. Targets TRAF2 for lysosomal degradation. In vitro able to deubiquitinate 'Lys-11', 'Lys-48' and 'Lys-63' polyubiquitin chains. Inhibitor of programmed cell death. Has a role in the function of the lymphoid system. Required for LPS-induced production of proinflammatory cytokines and IFN beta in LPS-tolerized macrophages.

Aliases: A20 antibody, AISBL antibody, MGC104522 antibody, MGC138687 antibody, MGC138688 antibody, OTU domain containing protein 7C antibody, OTU domain-containing protein 7C antibody, OTUD7C antibody, Putative DNA binding protein A20 antibody, Putative DNA-binding protein A20 antibody, TNAP3_HUMAN antibody, TNF alpha-induced protein 3 antibody, TNFA1P2 antibody, TNFAIP 3 antibody, TNFAIP3 (A20) antibody, TNFAIP3 antibody, Tumor necrosis factor alpha induced protein 3 antibody, Tumor necrosis factor alpha-induced protein 3 antibody, Tumor necrosis factor induced protein 3 antibody, Tumor necrosis factor inducible protein A20 antibody, tumor necrosis factor, alpha-induced protein 3 antibody, Zinc finger protein A20 antibody

UniProt: [P21580](#)

Pathways: [TLR Signaling](#), [Activation of Innate immune Response](#), [Cellular Response to Molecule of Bacterial Origin](#), [Production of Molecular Mediator of Immune Response](#)

Application Details

Application Notes:	Optimal working dilution should be determined by the investigator.
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Restrictions:	For Research Use only
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Handling

Format:	Liquid
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Buffer:	Preservative: 0.03 % Proclin 300 Constituents: 50 % Glycerol, 0.01M PBS, PH 7.4
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Preservative:	ProClin
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Precaution of Use:	This product contains ProClin: a POISONOUS AND HAZARDOUS SUBSTANCE which should be handled by trained staff only.
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Storage:	-20 °C,-80 °C
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Storage Comment:	Upon receipt, store at -20°C or -80°C. Avoid repeated freeze.
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