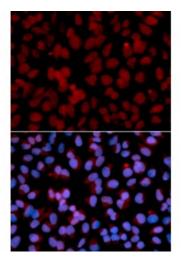


Anti-TNFAIP3 Antibody





Description This gene was identified as a gene whose expression is rapidly induced by

the tumor necrosis factor (TNF). The protein encoded by this gene is a zinc finger protein and ubiqitin-editing enzyme, and has been shown to inhibit NF-kappa B activation as well as TNF-mediated apoptosis. The encoded protein, which has both ubiquitin ligase and deubiquitinase activities, is involved in the cytokine-mediated immune and inflammatory responses. Several transcript variants encoding the same protein have been

found for this gene.

Model STJ115596

Host Rabbit

Reactivity Human, Rat

Applications IF, WB

Immunogen Recombinant fusion protein containing a sequence corresponding to amino

acids 1-190 of human TNFAIP3 (NP_006281.1).

Gene ID <u>7128</u>

Gene Symbol TNFAIP3

Dilution range WB 1:500 - 1:2000

IF 1:50 - 1:200

Purification Affinity purification

Note For Research Use Only (RUO).

Protein Name Tumor necrosis factor alpha-induced protein 3 TNF alpha-induced protein 3

Molecular Weight 89.614 kDa

Clonality Polyclonal

Conjugation Unconjugated

Isotype IgG

Formulation PBS with 0.02% sodium azide, 50% glycerol, pH7.3.

Storage Instruction Store at -20C. Avoid freeze / thaw cycles.

Database Links HGNC:11896OMIM:191163Reactome:R-HSA-168638

Alternative Names Tumor necrosis factor alpha-induced protein 3 TNF alpha-induced protein 3

Function 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

Cellular Localization Cytoplasm, Nucleus, Lysosome

Post-translational

Modifications

Proteolytically cleaved by MALT1 upon TCR stimulation

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