

ZC3H12A Antibody

Catalog # ASC11098

Product Information

Application WB, E **Primary Accession** Q5D1E8

Other Accession NP_079355, 156151383

Reactivity
Human
Rabbit
Clonality
Polyclonal
Isotype
IgG
Calculated MW
65699
Concentration (mg/ml)
1 mg/mL
Conjugate
Unconjugated

Application Notes ZC3H12A antibody can be used for detection of ZC3H12A by Western blot at 1

- 2 □g/mL.

Additional Information

Gene ID 80149

Other Names Ribonuclease ZC3H12A, 3.1.-.-, MCP-induced protein 1, Zinc finger CCCH

domain-containing protein 12A, ZC3H12A {ECO:0000312 | EMBL:EAX07346.1}

Target/Specificity ZC3H12A; This ZC3H12A antibody is predicted to have no cross-reactivity to

other members of the ZC3H12 protein family.

Reconstitution & Storage ZC3H12A antibody can be stored at 4°C for three months and -20°C, stable for

up to one year. As with all antibodies care should be taken to avoid repeated freeze thaw cycles. Antibodies should not be exposed to prolonged high

temperatures.

Precautions ZC3H12A Antibody is for research use only and not for use in diagnostic or

therapeutic procedures.

Protein Information

Name ZC3H12A (<u>HGNC:26259</u>)

Function Endoribonuclease involved in various biological functions such as cellular

inflammatory response and immune homeostasis, glial differentiation of neuroprogenitor cells, cell death of cardiomyocytes, adipogenesis and angiogenesis. Functions as an endoribonuclease involved in mRNA decay (PubMed:19909337). Modulates the inflammatory response by promoting the

degradation of a set of translationally active cytokine-induced

inflammation-related mRNAs, such as IL6 and IL12B, during the early phase of inflammation (PubMed:<u>26320658</u>). Prevents aberrant T-cell-mediated immune reaction by degradation of multiple mRNAs controlling T-cell

activation, such as those encoding cytokines (IL6 and IL2), cell surface receptors (ICOS, TNFRSF4 and TNFR2) and transcription factor (REL) (By similarity). Inhibits cooperatively with ZC3H12A the differentiation of helper T cells Th17 in lungs. They repress target mRNA encoding the Th17 cell-promoting factors IL6, ICOS, REL, IRF4, NFKBID and NFKBIZ. The cooperation requires RNA-binding by RC3H1 and the nuclease activity of ZC3H12A (By similarity). Together with RC3H1, destabilizes TNFRSF4/OX40 mRNA by binding to the conserved stem loop structure in its 3'UTR (By similarity). Self regulates by destabilizing its own mRNA (By similarity). Cleaves mRNA harboring a stem-loop (SL), often located in their 3'-UTRs, during the early phase of inflammation in a helicase UPF1-dependent manner (PubMed: 19909337, PubMed: 22561375, PubMed: 26134560, PubMed: 26320658). Plays a role in the inhibition of microRNAs (miRNAs) biogenesis (PubMed:22055188). Cleaves the terminal loop of a set of precursor miRNAs (pre-miRNAs) important for the regulation of the inflammatory response leading to their degradation, and thus preventing the biosynthesis of mature miRNAs (PubMed:22055188). Also plays a role in promoting angiogenesis in response to inflammatory cytokines by inhibiting the production of antiangiogenic microRNAs via its anti-dicer RNase activity (PubMed: <u>24048733</u>). Affects the overall ubiquitination of cellular proteins (By similarity). Positively regulates deubiquitinase activity promoting the cleavage at 'Lys-48'- and 'Lys-63'-linked polyubiquitin chains on TNF receptor-associated factors (TRAFs), preventing JNK and NF-kappa-B signaling pathway activation, and hence negatively regulating macrophage-mediated inflammatory response and immune homeostasis (By similarity). Also induces deubiquitination of the transcription factor HIF1A, probably leading to its stabilization and nuclear import, thereby positively regulating the expression of proangiogenic HIF1A-targeted genes (PubMed:24048733). Involved in a TANK-dependent negative feedback response to attenuate NF-kappaB activation through the deubiquitination of IKBKG or TRAF6 in response to interleukin-1-beta (IL1B) stimulation or upon DNA damage (PubMed: <u>25861989</u>). Prevents stress granule (SGs) formation and promotes macrophage apoptosis under stress conditions, including arsenite- induced oxidative stress, heat shock and energy deprivation (By similarity). Plays a role in the regulation of macrophage polarization; promotes IL4-induced polarization of macrophages M1 into anti- inflammatory M2 state (By similarity). May also act as a transcription factor that regulates the expression of multiple genes involved in inflammatory response, angiogenesis, adipogenesis and apoptosis (PubMed: 16574901, PubMed: 18364357). Functions as a positive regulator of glial differentiation of neuroprogenitor cells through an amyloid precursor protein (APP)-dependent signaling pathway (PubMed: 19185603). Attenuates septic myocardial contractile dysfunction in response to lipopolysaccharide (LPS) by reducing I-kappa-B-kinase (IKK)-mediated NF-kappa-B activation, and hence myocardial pro-inflammatory cytokine production (By similarity).

Cellular Location

Nucleus. Cytoplasm. Cytoplasm, P-body. Rough endoplasmic reticulum membrane {ECO:0000250|UniProtKB:Q5D1E7}; Peripheral membrane protein {ECO:0000250|UniProtKB:Q5D1E7}; Cytoplasmic side {ECO:0000250|UniProtKB:Q5D1E7}. Cytoplasmic granule {ECO:0000250|UniProtKB:Q5D1E7}. Note=Predominantly localized in the cytoplasm. Colocalizes with GW182 on many granule-like structures, probably corresponding to cytoplasmic GW bodies (GWBs), also called processing bodies (P bodies). Colocalizes with calnexin on the surface of the rough endoplasmic reticulum (RER) membrane and with translationally active polysomes (By similarity). Colocalizes with ZC3H12D in cytoplasmic mRNA processing P-body, also known as GW bodies (GWBs) (PubMed:22055188, PubMed:26134560)

Tissue Location

Expressed in heart, placenta, spleen, kidney, liver and lung

Background

ZC3H12A Antibody: ZC3H12A, also known as MCPIP, is an essential member of a family of novel CCCH-zinc finger proteins that regulate macrophage activation and may be involved in host immunity and inflammatory diseases. ZC3H12A has RNase activity that prevents some immune disorders by directly affecting the mRNA stability of interleukins such as IL-6 and IL12p40. Mice lacking the ZC3H12A gene suffered from severe anemia, and most dies within 12 weeks. Overexpression of ZC3H12A causes ER stress and induces a number of genes involved in apoptosis and autophagy, including JNK, PUMA, and beclin-1 in cardiac myoblasts, leading to cell death. ZC3H12A can also induce adipogenesis in 3T3-L1 pre-adipocytes in the absence of PPAPgamma, demonstrating the complex roles ZC3H12A plays.

References

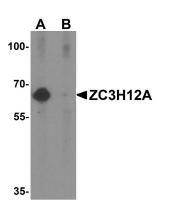
Zhou L, Azfer A, Niu J, et al. Monocyte chemoattractant protein-1 induces a novel transcription factor that causes cardiac myocyte apoptosis and ventricular dysfunction. Circ. Res. 2006; 98:1177-85.

Liang J, Wang J, Azfer A, et al. A novel CCCH-zinc finger protein family regulates proinflammatory activation of macrophages. J. Biol. Chem. 2008; 283:6337-46.

Matsushita K, Takeuchi O, Standley DM, et al. Zc3h12a is an RNase essential for controlling immune responses by regulating mRNA decay. Nature 2009; 458:1185-90.

Younce CW and Kolattukudy PE. MCP-1 causes cardiomyoblast death via autophagy resulting from ER stress caused by oxidative stress generated by inducing a novel zinc-finger protein, MCPIP. Biochem. J. 2010; 426:43-53.

Images



Western blot analysis of ZC3H12A in K562 cell lysate with ZC3H12A antibody at 1 μ g/mL in (A) the absence and (B) the presence of blocking peptide.

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