

ARID5A Antibody (Center)
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP17617c**Specification**

ARID5A Antibody (Center) - Product Information

Application	WB,E
Primary Accession	Q03989
Other Accession	NP_997646.1
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	64074
Antigen Region	382-411

ARID5A Antibody (Center) - Additional Information**Gene ID** 10865**Other Names**

AT-rich interactive domain-containing protein 5A, ARID domain-containing protein 5A, Modulator recognition factor 1, MRF-1, ARID5A, MRF1

Target/Specificity

This ARID5A antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 382-411 amino acids from the Central region of human ARID5A.

Dilution

WB~~1:1000

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

ARID5A Antibody (Center) is for research use only and not for use in diagnostic or therapeutic procedures.

ARID5A Antibody (Center) - Protein Information**Name** ARID5A**Synonyms** MRF1

Function Binds to AT-rich stretches in the modulator region upstream of the human cytomegalovirus major intermediate early gene enhancer. May act as repressor and down-regulate enhancer-dependent gene expression (PubMed:[8649988](#)). May positively regulate chondrocyte-specific transcription such as of COL2A1 in collaboration with SOX9 and positively regulate histone H3 acetylation at chondrocyte-specific genes. May stimulate early-stage chondrocyte differentiation and inhibit later stage differentiation (By similarity). Can repress ESR1- mediated transcriptional activation; proposed to act as corepressor for selective nuclear hormone receptors (PubMed:[15941852](#)). As RNA-binding protein involved in the regulation of inflammatory response by stabilizing selective inflammation-related mRNAs, such as IL6, STAT3 and TBX21. Binds to stem loop structures located in the 3'UTRs of IL6, STAT3 and TBX21 mRNAs; at least for STAT3 prevents binding of ZC3H12A to the mRNA stem loop structure thus inhibiting its degradation activity. Contributes to elevated IL6 levels possibly implicated in autoimmunity processes. IL6-dependent stabilization of STAT3 mRNA may promote differentiation of naive CD4+ T-cells into T-helper Th17 cells. In CD4+ T-cells may also inhibit RORC-induced Th17 cell differentiation independently of IL6 signaling. Stabilization of TBX21 mRNA contributes to elevated interferon-gamma secretion in Th1 cells possibly implicated in the establishment of septic shock (By similarity). Stabilizes TNFRSF4/OX40 mRNA by binding to the conserved stem loop structure in its 3'UTR; thereby competing with the mRNA-destabilizing functions of RC3H1 and endoribonuclease ZC3H12A (By similarity).

Cellular Location

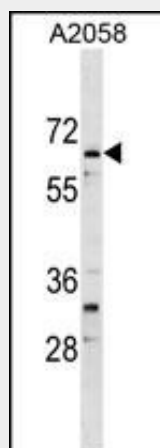
Nucleus {ECO:0000255|PROSITE-ProRule:PRU00355, ECO:0000269|PubMed:8649988}

ARID5A Antibody (Center) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

ARID5A Antibody (Center) - Images



ARID5A Antibody (Center) (Cat. #AP17617c) western blot analysis in A2058 cell line lysates (35ug/lane). This demonstrates the ARID5A antibody detected the ARID5A protein (arrow).

ARID5A Antibody (Center) - Background

Members of the ARID protein family, including ARID5A, have diverse functions but all appear to play important roles in development, tissue-specific gene expression, and regulation of cell growth (Patsialou et al., 2005 [PubMed 15640446]).[supplied by OMIM].

ARID5A Antibody (Center) - References

Lim, J., et al. Cell 125(4):801-814(2006)
Patsialou, A., et al. Nucleic Acids Res. 33(1):66-80(2005)
Clark, H.F., et al. Genome Res. 13(10):2265-2270(2003)
Huang, T.H., et al. Nucleic Acids Res. 24(9):1695-1701(1996)