Revision 1

Store at -20C

#13357

ARD1A (E1J2B) Rabbit mAb

Cell Signaling TECHNOLOGY®

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Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W, IP	H M R Mk	Endogenous	28	Rabbit IgG	#P41227	8260

Product Usage Information

Application	Dilution
Western Blotting	1:1000
Immunoprecipitation	1:50

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.

Specificity/Sensitivity

ARD1A (E1J2B) Rabbit mAb recognizes endogenous levels of total ARD1A protein.

Species predicted to react based on 100% sequence homology

Hamster, Horse

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Asp204 of human ARD1A protein.

Background

Protein acetylation is a common modification that occurs both at lysine residues within proteins (ε-amino acetylation) and multiple amino acid residues at the amino terminus of proteins (α-amino acetylation). The N-α-acetyltransferase ARD1 homolog A protein (ARD1A, also known as NAA10) and the highly homologous N-α-acetyltransferase ARD1 homolog B protein (ARD1B, also known as ARD2 or NAA11) are mutually exclusive catalytic subunits of the amino-terminal acetyltransferase complex (NatA) (1-3). This complex, which consists of either ARD1A or ARD1B and the N-α-acetyltransferase 15 (NAA15) auxiliary protein, localizes to ribosomes where it functions to acetylate Ser-, Ala-, Gly-, Thr-, Cys-, Pro-, and Val- amino termini after initiator methionine cleavage during protein translation (1-5). Like ε-amino acetylation, amino-terminal α-amino acetylation functions to regulate protein stability, activity, cellular localization, and protein-protein interactions (4,5). Defects in ARD1A have been shown to cause amino-terminal acetyltransferase deficiency (NATD), which results in severe delays and defects in postnatal growth (6).

In addition to functioning as amino-terminal acetyltransferases in the NatA complex, free ARD1A and ARD1B proteins regulate cell growth and differentiation through ε-amino acetylation of lysine residues in multiple target proteins, including the HIF-1α, β-catenin, and AP-1 transcription factors (7-9). ARD1A-mediated acetylation of HIF-1α at Lys532 under normoxic conditions enhances binding of VHL, leading to increased ubiquitination and degradation of HIF-1α and down-regulation of HIF-1α target genes involved in angiogenesis, apoptosis, cellular proliferation, and glucose metabolism (7). Decreased expression of ARD1A under hypoxic conditions contributes to the stabilization of HIF-1α and upregulation of target genes (7). ARD1A also promotes cell proliferation and tumorigenesis by acetylating and activating β-catenin and AP-1 transcription factors, leading to the stimulation of cyclin D1 expression (8,9). Interestingly, the acetyltransferase activity of ARD1A is regulated by autoacetylation at Lys136, which is required for the ability of ARD1A to promote proliferation and tumorigenesis (9). Research studies have shown that ARD1 proteins are over-expressed in multiple cancers, including breast, prostate, lung, and colorectal cancers (10-13).

Background References

1. Arnesen, T. et al. (2005) *Biochem J* 386, 433-43.
2. Arnesen, T. et al. (2006) *BMC Biochem* 7, 13.
3. Pang, A.L. et al. (2009) *Biol Reprod* 81, 302-9.
4. Van Damme, P. et al. (2011) *FEBS J* 278, 3822-34.
5. Polevoda, B. and Sherman, F. (2000) *J Biol Chem* 275, 36479-82.
6. Rope, A.F. et al. (2011) *Am J Hum Genet* 89, 28-43.
7. Jeong, J.W. et al. (2002) *Cell* 111, 709-20.
8. Lim, J.H. et al. (2006) *Cancer Res* 66, 10677-82.
9. Seo, J.H. et al. (2010) *Cancer Res* 70, 4422-32.
10. Arnesen, T. et al. (2005) *Thyroid* 15, 1131-6.
11. Ren, T. et al. (2008) *Cancer Lett* 264, 83-92.
12. Yu, M. et al. (2009) *Oncol Rep* 21, 909-15.

13. Yu, M. et al. (2009) *Cancer Invest* 27, 978-83.

Species Reactivity

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

Western Blot Buffer

IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

Applications Key

W: Western Blotting **IP:** Immunoprecipitation

Cross-Reactivity Key

H: Human **M:** Mouse **R:** Rat **Mk:** Monkey

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Limited Uses