

HIRA (D2A5E) Rabbit mAb

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Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W	H M Mk	Endogenous	112	Rabbit IgG	#P54198	7290

Product Usage Information**Application**

Western Blotting

Dilution

1:1000

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

HIRA (D2A5E) Rabbit mAb recognizes endogenous levels of total HIRA protein.

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Leu593 of human HIRA protein.

Background

Histone cell cycle regulation defective homolog A (HIRA), also known as TUP1-like enhancer of split protein 1 (TUPLE1), is the mammalian homolog of the yeast HIR1 and HIR2 transcriptional repressor proteins (1). HIRA interacts with UBN1, CABIN, and ASF1A in the cell nucleus to form the evolutionarily conserved HUCA histone chaperone complex that deposits the variant histone H3.3 into chromatin in a DNA-replication independent manner (2). HIRA is required for deposition of histone H3.3 at the transcription start sites of genes, where incorporation of histone H3.3 facilitates nucleosome destabilization and contributes to transcriptional activation (3-5). Histone H3.3 is also linked to gene silencing and is incorporated into regions of the genome thought to be transcriptionally inactive (5-7). While some incorporation of H3.3 into heterochromatin is facilitated by a different histone chaperone complex that contains ATRX and DAXX (ie. telomeric incorporation of H3.3), HIRA is required for incorporation of histone H3.3 and formation of senescence-associated heterochromatin foci (SAHF) during cellular senescence (5-8). HIRA is ubiquitously expressed during mouse embryonic development (9). In the adult mouse, HIRA is expressed at high levels in the kidney, skeletal muscle, and pancreas, but it is expressed at lower levels in the heart, lung, placenta, brain, and liver (9). A missing copy of the HIRA gene on human chromosome region 22q11.2 is a common characteristic of DiGeorge syndrome patients and insufficient production of the HIRA protein may disrupt normal embryonic development (9).

Background References

1. Lamour, V. et al. (1995) *Hum Mol Genet* 4, 791-9.
2. Rai, T.S. et al. (2011) *Mol Cell Biol* 31, 4107-18.
3. Jin, C. et al. (2009) *Nat Genet* 41, 941-5.
4. Jin, C. and Felsenfeld, G. (2007) *Genes Dev* 21, 1519-29.
5. Goldberg, A.D. et al. (2010) *Cell* 140, 678-91.
6. Wong, L.H. et al. (2010) *Genome Res* 20, 351-60.
7. Wong, L.H. et al. (2009) *Genome Res* 19, 404-14.
8. Zhang, R. et al. (2007) *Mol Cell Biol* 27, 2343-58.
9. Wilming, L.G. et al. (1997) *Hum Mol Genet* 6, 247-58.

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

Cross-Reactivity Key

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

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