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## HMGA1 (D6A4) XP® Rabbit mAb



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Applications: Reactivity: Sensitivity: MW (kDa): Source/Isotype: **UniProt ID:** Entrez-Gene Id: WB, IP, IF-IC H Mk Endogenous 18 Rabbit IgG #P17096 3159 **Product Usage** Application Dilution Information Western Blotting 1:1000 1:200 Immunoprecipitation Immunofluorescence (Immunocytochemistry) 1:800 - 1:3200 Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 μg/ml BSA, 50% glycerol and less than **Storage** 0.02% sodium azide. Store at -20°C. Do not aliquot the antibody. HMGA1 (D6A4) XP® Rabbit mAb recognizes endogenous levels of total HMGA1 protein, isoforms 1a and Specificity / Sensitivity 1b. Based on sequence homology, this antibody is not predicted to cross-react with HMGA2. Species predicted to

react based on 100% sequence homology: **Bovine** 

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Gly68 of human HMGA1 protein.

**Background** 

HMGA1, formerly known as HMG-I/Y, belongs to a family of high mobility group proteins that contain an AThook DNA binding domain. HMGA proteins are considered architectural transcription factors; they do not have direct transcriptional activation capacity, but instead regulate gene expression by changing DNA conformation through binding to AT-rich regions in the DNA and/or direct interaction with other transcription factors (1,2). HMGA1 is highly expressed during embryogenesis and in embryonic stem cells, but not in fully differentiated adult tissues (2-4). Research studies have shown that HMGA1 is over-expressed in rapidly dividing neoplastic cells and a wide variety of aggressive cancers, including thyroid, colon, breast, pancreas, and prostate (2-4). Investigators have shown that forced expression of HMGA1 induces cellular transformation and an epithelial-to-mesenchymal transition (EMT), while inhibition of HMGA1 expression blocks anchorage-independent cell growth and proliferation of cancer cells, suggesting that HMGA1 contributes to carcinogenesis by inducing and maintaining a de-differentiated, highly proliferative cell state (5-8).

## **Background References**

- 1. Cleynen, I. and Van de Ven, W.J. (2008) Int J Oncol 32, 289-305.
- 2. Resar, L.M. (2010) Cancer Res 70, 436-9.
- 3. Chiappetta, G. et al. (1996) Oncogene 13, 2439-46.
- 4. Ben-Porath, I. et al. (2008) Nat Genet 40, 499-507.
- 5. Wood, L.J. et al. (2000) Mol Cell Biol 20, 5490-502.
- 6. Wood, L.J. et al. (2000) Cancer Res 60, 4256-61.
- 7. Xu, Y. et al. (2004) Cancer Res 64, 3371-5. 8. Scala, S. et al. (2000) Proc Natl Acad Sci U S A 97, 4256-61.

**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 Cross-Reactivity Key**  WB: Western Blotting IP: Immunoprecipitation IF-IC: Immunofluorescence (Immunocytochemistry)

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H: human M: mouse R: rat Hm: hamster Mk: monkey Vir: virus Mi: mink C: chicken Dm: D, melanogaster X: Xenopus Z: zebrafish B: bovine Dq: dog Pq: pig Sc: S. cerevisiae Ce: C. elegans Hr: horse GP: Guinea Pig Rab: rabbit All: all species expected

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