

POLR1A (D6S6S) Rabbit mAb

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Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W, ChIP	H M Mk	Endogenous	200	Rabbit IgG	#O95602	25885

Product Usage Information

For optimal ChIP results, use 10 µl of antibody and 10 µg of chromatin (approximately 4 x 10⁶ cells) per IP. This antibody has been validated using SimpleChIP[®] Enzymatic Chromatin IP Kits.

Application

Western Blotting
Chromatin IP

Dilution

1:1000
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

POLR1A (D6S6S) Rabbit mAb recognizes endogenous levels of total POLR1A protein.

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Pro36 of human POLR1A protein.

Background

RNA polymerase I (RNAPI) is a large multi-protein complex that functions as a DNA dependent, RNA polymerase, which is primarily responsible for the transcription of ribosomal RNA (rRNA) genes. The largest subunit, Rpa194, or POLR1A, along with selectivity factor 1 (SL1), and the transactivator protein upstream binding factor (UBF) make up the core transcriptional machinery of the RNAPI complex (1-3). The RNAPI complex is recruited specifically to rDNA promoters by SL1, which contains TBP and TAF proteins, to transcribe precursors to rRNA (2). These precursors are processed into 18S, 5.8S, and 28S mature rRNAs, which make up most of the ribosomal structure (1). Similar to the RNA polymerase II complex, there are other core components that are required for transcription of target genes such as the TFIIF complex and SPT6 (4, 5). Overexpression of nascent rRNA has been shown to be associated with poor prognosis in certain cancer types, and enlarged nucleoli are a hallmark of cellular proliferation and aggressive tumors (6-8). Oncogenes such as Myc, Ras, and PI3K can drive RNAPI-mediated rRNA transcription, making POLR1A a key therapeutic target (9). Indeed, specific targeting of RNAPI activity with a small molecule inhibitor can induce autophagy selectively in tumor cells while having minimal effects in normal cells (10). Additionally, mutations in POLR1A are associated with acrofacial dysostosis, Cincinnati type, a cranioskeletal malformation syndrome. Loss of function mutations result in disrupted ribosome biogenesis and p53-mediated cell death affecting skeletal precursor cells or the neural-crest (11).

Background References

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3. Comai, L. et al. (1992) *Cell* 68, 965-76.
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6. Williamson, D. et al. (2006) *Genes Chromosomes Cancer* 45, 839-45.
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9. Woods, S.J. et al. (2015) *Biochim Biophys Acta* 1849, 821-9.
10. Drygin, D. et al. (2011) *Cancer Res* 71, 1418-30.
11. Weaver, K.N. et al. (2015) *Am J Hum Genet* 96, 765-74.

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 **ChIP:** Chromatin IP

Cross-Reactivity Key

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

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