

SOX9 Antibody (N-term)

Affinity Purified Rabbit Polyclonal Antibody (Pab)

Catalog # AP1409A

Product Information

Application	WB, IHC-P, IF, E
Primary Accession	P48436
Other Accession	Q18896 , Q04887
Reactivity	Mouse, Human
Predicted	Mouse, Pig, Rat, Bovine, Canine, Rabbit, Chicken
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Clone Names	RB13373
Calculated MW	56137
Antigen Region	41-70

Additional Information

Gene ID	6662
Other Names	Transcription factor SOX-9, SOX9
Target/Specificity	This SOX9 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 41-70 amino acids from the N-terminal region of human SOX9.
Dilution	WB~~1:1000 IHC-P~~1:100~500 IF~~1:10~50 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.05% (V/V) Proclin 300. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.
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	SOX9 Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	SOX9 {ECO:0000303 PubMed:7990924, ECO:0000312 HGNC:HGNC:11204}
Function	Transcription factor that plays a key role in chondrocytes differentiation and skeletal development (PubMed: 24038782). Specifically binds the 5'-ACAAAG-3'

DNA motif present in enhancers and super-enhancers and promotes expression of genes important for chondrogenesis, including cartilage matrix protein-coding genes COL2A1, COL4A2, COL9A1, COL11A2 and ACAN, SOX5 and SOX6 (PubMed:[8640233](#)). Also binds to some promoter regions (By similarity). Plays a central role in successive steps of chondrocyte differentiation (By similarity). Absolutely required for precartilaginous condensation, the first step in chondrogenesis during which skeletal progenitors differentiate into prechondrocytes (By similarity). Together with SOX5 and SOX6, required for overt chondrogenesis when condensed prechondrocytes differentiate into early stage chondrocytes, the second step in chondrogenesis (By similarity). Later, required to direct hypertrophic maturation and block osteoblast differentiation of growth plate chondrocytes: maintains chondrocyte columnar proliferation, delays prehypertrophy and then prevents osteoblastic differentiation of chondrocytes by lowering beta-catenin (CTNNB1) signaling and RUNX2 expression (By similarity). Also required for chondrocyte hypertrophy, both indirectly, by keeping the lineage fate of chondrocytes, and directly, by remaining present in upper hypertrophic cells and transactivating COL10A1 along with MEF2C (By similarity). Low lipid levels are the main nutritional determinant for chondrogenic commitment of skeletal progenitor cells: when lipids levels are low, FOXO (FOXO1 and FOXO3) transcription factors promote expression of SOX9, which induces chondrogenic commitment and suppresses fatty acid oxidation (By similarity). Mechanistically, helps, but is not required, to remove epigenetic signatures of transcriptional repression and deposit active promoter and enhancer marks at chondrocyte-specific genes (By similarity). Acts in cooperation with the Hedgehog pathway-dependent GLI (GLI1 and GLI3) transcription factors (By similarity). In addition to cartilage development, also acts as a regulator of proliferation and differentiation in epithelial stem/progenitor cells: involved in the lung epithelium during branching morphogenesis, by balancing proliferation and differentiation and regulating the extracellular matrix (By similarity). Controls epithelial branching during kidney development (By similarity).

Cellular Location

Nucleus {ECO:0000255 | PROSITE-ProRule:PRU00267, ECO:0000269 | PubMed:8640233}

Background

SOX9 recognizes the sequence CCTTGAG along with other members of the HMG-box class DNA-binding proteins. It acts during chondrocyte differentiation and, with steroidogenic factor 1, regulates transcription of the anti-Muellerian hormone (AMH) gene. Deficiencies lead to the skeletal malformation syndrome campomelic dysplasia, frequently with sex reversal.

References

- Malki,S., Cancer Lett. 255 (2), 182-193 (2007)
 Passeron,T., Proc. Natl. Acad. Sci. U.S.A. 104 (35), 13984-13989 (2007)

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