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SOX9 Antibody (aa1-150)
Rabbit Polyclonal Antibody
Catalog # ALS12789

Specification

SOX9 Antibody (aa1-150) - Product Information

Application	**IHC**
Primary Accession	P48436
Reactivity	**Human**
Host	**Rabbit**
Clonality	**Polyclonal**
Calculated MW	**56kDa KDa**

SOX9 Antibody (aa1-150) - Additional Information

Gene ID 6662

Other Names
Transcription factor SOX-9, SOX9

Reconstitution & Storage
2°C to 8°C

Precautions
SOX9 Antibody (aa1-150) is for research use only and not for use in diagnostic or therapeutic procedures.

SOX9 Antibody (aa1-150) - 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}

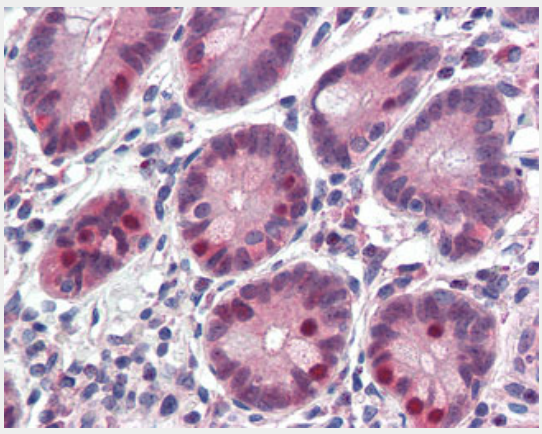
Volume
250 µl

SOX9 Antibody (aa1-150) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

SOX9 Antibody (aa1-150) - Images



Anti-SOX9 antibody IHC of human small intestine.

SOX9 Antibody (aa1-150) - Background

Plays an important role in the normal skeletal development. May regulate the expression of other genes involved in chondrogenesis by acting as a transcription factor for these genes.

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Foster J.W.,et al.Nature 372:525-530(1994).
Wagner T.,et al.Cell 79:1111-1120(1994).
Kalnine N.,et al.Submitted (MAY-2003) to the EMBL/GenBank/DDBJ databases.
Mural R.J.,et al.Submitted (JUL-2005) to the EMBL/GenBank/DDBJ databases.
Cox J.J.,et al.N. Engl. J. Med. 364:91-93(2011).