

LOXL2 Antibody (Internal)
Rabbit Polyclonal Antibody
Catalog # ALS11094**Specification**

LOXL2 Antibody (Internal) - Product Information

Application	IHC
Primary Accession	O9Y4K0
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Calculated MW	87kDa KDa

LOXL2 Antibody (Internal) - Additional Information**Gene ID** 4017**Other Names**

Lysyl oxidase homolog 2, 1.4.3.13, Lysyl oxidase-like protein 2, Lysyl oxidase-related protein 2, Lysyl oxidase-related protein WS9-14, LOXL2

Target/Specificity

Human LOXL2. BLAST analysis of the peptide immunogen showed no homology with other human proteins.

Reconstitution & Storage

Long term: -70°C; Short term: +4°C

Precautions

LOXL2 Antibody (Internal) is for research use only and not for use in diagnostic or therapeutic procedures.

LOXL2 Antibody (Internal) - Protein Information**Name** LOXL2**Function**

Mediates the post-translational oxidative deamination of lysine residues on target proteins leading to the formation of deaminated lysine (allysine) (PubMed:[27735137](http://www.uniprot.org/citations/27735137)). Acts as a transcription corepressor and specifically mediates deamination of trimethylated 'Lys-4' of histone H3 (H3K4me3), a specific tag for epigenetic transcriptional activation (PubMed:[27735137](http://www.uniprot.org/citations/27735137)). Shows no activity against histone H3 when it is trimethylated on 'Lys-9' (H3K9me3) or 'Lys-27' (H3K27me3) or when 'Lys-4' is monomethylated (H3K4me1) or dimethylated (H3K4me2) (PubMed:[27735137](http://www.uniprot.org/citations/27735137)). Also mediates deamination of methylated TAF10, a member of the transcription factor IID (TFIID) complex, which induces release of TAF10 from promoters, leading to inhibition of TFIID-dependent

transcription (PubMed:25959397). LOXL2-mediated deamination of TAF10 results in transcriptional repression of genes required for embryonic stem cell pluripotency including POU5F1/OCT4, NANOG, KLF4 and SOX2 (By similarity). Involved in epithelial to mesenchymal transition (EMT) via interaction with SNAI1 and participates in repression of E-cadherin CDH1, probably by mediating deamination of histone H3 (PubMed:16096638, PubMed:27735137, PubMed:24414204). During EMT, involved with SNAI1 in negatively regulating pericentromeric heterochromatin transcription (PubMed:24239292). SNAI1 recruits LOXL2 to pericentromeric regions to oxidize histone H3 and repress transcription which leads to release of heterochromatin component CBX5/HP1A, enabling chromatin reorganization and acquisition of mesenchymal traits (PubMed:24239292). Interacts with the endoplasmic reticulum protein HSPA5 which activates the IRE1-XBP1 pathway of the unfolded protein response, leading to expression of several transcription factors involved in EMT and subsequent EMT induction (PubMed:28332555). Involved in E-cadherin repression following hypoxia, a hallmark of EMT believed to amplify tumor aggressiveness, suggesting that it may play a role in tumor progression (PubMed:20026874). When secreted into the extracellular matrix, promotes cross-linking of extracellular matrix proteins by mediating oxidative deamination of peptidyl lysine residues in precursors to fibrous collagen and elastin (PubMed:20306300). Acts as a regulator of sprouting angiogenesis, probably via collagen IV scaffolding (PubMed:21835952). Acts as a regulator of chondrocyte differentiation, probably by regulating expression of factors that control chondrocyte differentiation (By similarity).

Cellular Location

Secreted, extracellular space, extracellular matrix, basement membrane. Nucleus. Chromosome. Endoplasmic reticulum. Note=Associated with chromatin (PubMed:27735137). It is unclear how LOXL2 is nuclear as it contains a signal sequence and has been shown to be secreted (PubMed:23319596) However, a number of reports confirm its intracellular location and its key role in transcription regulation (PubMed:22204712, PubMed:22483618).

Tissue Location

Expressed in many tissues (PubMed:10212285). Highest expression in reproductive tissues, placenta, uterus and prostate (PubMed:10212285). In esophageal epithelium, expressed in the basal, prickle and granular cell layers (PubMed:22204712). Up-regulated in a number of cancers cells and tissues.

Volume

100 µl

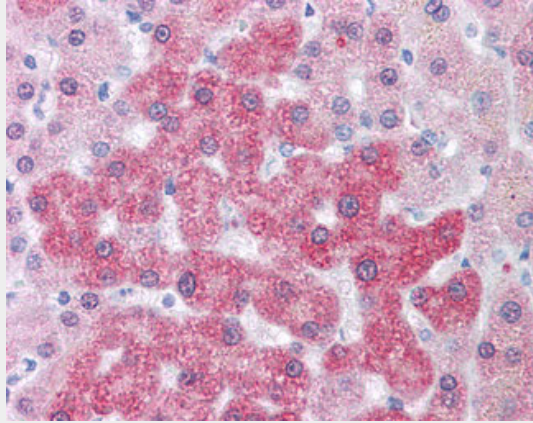
LOXL2 Antibody (Internal) - Protocols

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

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)

- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

LOXL2 Antibody (Internal) - Images



Anti-LOXL2 antibody ALS11094 IHC of human liver.

LOXL2 Antibody (Internal) - Background

Mediates the post-translational oxidative deamination of lysine residues on target proteins leading to the formation of deaminated lysine (allysine). When secreted in extracellular matrix, promotes cross-linking of extracellular matrix proteins by mediating oxidative deamination of peptidyl lysine residues in precursors to fibrous collagen and elastin. Acts as a regulator of sprouting angiogenesis, probably via collagen IV scaffolding. When nuclear, acts as a transcription corepressor and specifically mediates deamination of trimethylated 'Lys-4' of histone H3 (H3K4me3), a specific tag for epigenetic transcriptional activation. Involved in epithelial to mesenchymal transition (EMT) via interaction with SNAIL and participates in repression of E-cadherin, probably by mediating deamination of histone H3. Also involved in E-cadherin repression following hypoxia, a hallmark of epithelial to mesenchymal transition believed to amplify tumor aggressiveness, suggesting that it may play a role in tumor progression. Acts as a regulator of chondrocyte differentiation, probably by regulating expression of factors that control chondrocyte differentiation.

LOXL2 Antibody (Internal) - References

- Saito H., et al. J. Biol. Chem. 272:8157-8160(1997).
Ota T., et al. Nat. Genet. 36:40-45(2004).
Suzuki Y., et al. Submitted (APR-2005) to the EMBL/GenBank/DDBJ databases.
Nusbaum C., et al. Nature 439:331-335(2006).
Jourdan-Le Saux C., et al. J. Biol. Chem. 274:12939-12944(1999).