10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

LOXL2 Antibody (C-term)

Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP16131b

Specification

LOXL2 Antibody (C-term) - Product Information

Application	**WB,E**
Primary Accession	Q9Y4K0
Other Accession	B5DF27, P58022, E1C3U7, A6H737, NP_002309.1
Reactivity	**Human**
Predicted	**Bovine, Chicken, Mouse, Rat**
Host	**Rabbit**
Clonality	**Polyclonal**
Isotype	**Rabbit IgG**
Calculated MW	**86725**
Antigen Region	**589-617**

LOXL2 Antibody (C-term) - Additional Information

Gene ID 4017

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

Target/Specificity
This LOXL2 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 589-617 amino acids from the C-terminal region of human LOXL2.

Dilution
WB~~1:1000

Format
Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

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

LOXL2 Antibody (C-term) - 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). 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). 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). 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:24414204, PubMed:27735137). 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.

LOXL2 Antibody (C-term) - 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

LOXL2 Antibody (C-term) - Images

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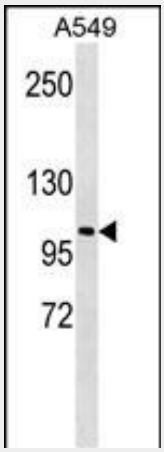

LOXL2 Antibody (C-term) (Cat. #AP16131b) western blot analysis in A549 cell line lysates (35ug/lane).This demonstrates the LOXL2 antibody detected the LOXL2 protein (arrow).

LOXL2 Antibody (C-term) - Background

This gene encodes a member of the lysyl oxidase gene family. The prototypic member of the family is essential to the biogenesis of connective tissue, encoding an extracellular copper-dependent amine oxidase that catalyses the first step in the formation of crosslinks in collagens and elastin. A highly conserved amino acid sequence at the C-terminus end appears to be sufficient for amine oxidase activity, suggesting that each family member may retain this function. The N-terminus is poorly conserved and may impart additional roles in developmental regulation, senescence, tumor suppression, cell growth control, and chemotaxis to each member of the family.

LOXL2 Antibody (C-term) - References

Rodriguez, H.M., et al. J. Biol. Chem. 285(27):20964-20974(2010)
Ruckert, F., et al. Int J Colorectal Dis 25(3):303-311(2010)
Schietke, R., et al. J. Biol. Chem. 285(9):6658-6669(2010)
Sano, M., et al. Int. J. Oncol. 36(2):321-330(2010)
Kim, Y., et al. Oncol. Rep. 22(4):799-804(2009)