

LSD1 Antibody (Center)
Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP1218b**Specification**

LSD1 Antibody (Center) - Product Information

Application	WB, IHC-P,E
Primary Accession	O60341
Other Accession	O6ZQ88
Reactivity	Human
Predicted	Mouse
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	92903
Antigen Region	457-490

LSD1 Antibody (Center) - Additional Information**Gene ID** 23028**Other Names**

Lysine-specific histone demethylase 1A, 1---, BRAF35-HDAC complex protein BHC110, Flavin-containing amine oxidase domain-containing protein 2, KDM1A, AOF2, KDM1, KIAA0601, LSD1

Target/Specificity

This LSD1 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 457-490 amino acids from the Central region of human LSD1.

Dilution

WB~~1:1000
IHC-P~~1:50~100

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. 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

LSD1 Antibody (Center) is for research use only and not for use in diagnostic or therapeutic procedures.

LSD1 Antibody (Center) - Protein Information

Name KDM1A ([HGNC:29079](#))

Function Histone demethylase that can demethylate both 'Lys-4' (H3K4me) and 'Lys-9' (H3K9me) of histone H3, thereby acting as a coactivator or a corepressor, depending on the context (PubMed:[15620353](#), PubMed:[15811342](#), PubMed:[16079794](#), PubMed:[16079795](#), PubMed:[16140033](#), PubMed:[16223729](#), PubMed:[27292636](#)). Acts by oxidizing the substrate by FAD to generate the corresponding imine that is subsequently hydrolyzed (PubMed:[15620353](#), PubMed:[15811342](#), PubMed:[16079794](#), PubMed:[21300290](#)). Acts as a corepressor by mediating demethylation of H3K4me, a specific tag for epigenetic transcriptional activation. Demethylates both mono- (H3K4me1) and di-methylated (H3K4me2) H3K4me (PubMed:[15620353](#), PubMed:[20389281](#), PubMed:[21300290](#), PubMed:[23721412](#)). May play a role in the repression of neuronal genes. Alone, it is unable to demethylate H3K4me on nucleosomes and requires the presence of RCOR1/CoREST to achieve such activity (PubMed:[16079794](#), PubMed:[16140033](#), PubMed:[16885027](#), PubMed:[21300290](#), PubMed:[23721412](#)). Also acts as a coactivator of androgen receptor (AR)-dependent transcription, by being recruited to AR target genes and mediating demethylation of H3K9me, a specific tag for epigenetic transcriptional repression. The presence of PRKCB in AR-containing complexes, which mediates phosphorylation of 'Thr-6' of histone H3 (H3T6ph), a specific tag that prevents demethylation H3K4me, prevents H3K4me demethylase activity of KDM1A (PubMed:[16079795](#)). Demethylates di-methylated 'Lys- 370' of p53/TP53 which prevents interaction of p53/TP53 with TP53BP1 and represses p53/TP53-mediated transcriptional activation. Demethylates and stabilizes the DNA methylase DNMT1 (PubMed:[29691401](#)). Demethylates methylated 'Lys-42' and methylated 'Lys-117' of SOX2 (PubMed:[29358331](#)). Required for gastrulation during embryogenesis. Component of a RCOR/GFI/KDM1A/HDAC complex that suppresses, via histone deacetylase (HDAC) recruitment, a number of genes implicated in multilineage blood cell development (PubMed:[16079794](#), PubMed:[16140033](#)). Facilitates epithelial-to-mesenchymal transition by acting as an effector of SNAI1-mediated transcription repression of epithelial markers E-cadherin/CDH1, CDN7 and KRT8 (PubMed:[20562920](#), PubMed:[27292636](#)). Required for the maintenance of the silenced state of the SNAI1 target genes E-cadherin/CDH1 and CDN7 (PubMed:[20389281](#)).

Cellular Location

Nucleus. Chromosome. Note=Associates with chromatin

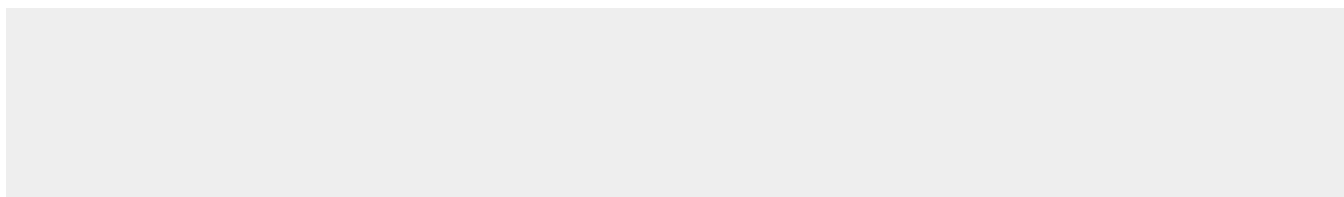
Tissue Location

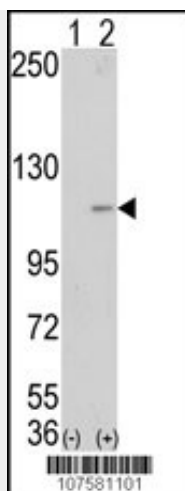
Ubiquitously expressed.

LSD1 Antibody (Center) - Protocols

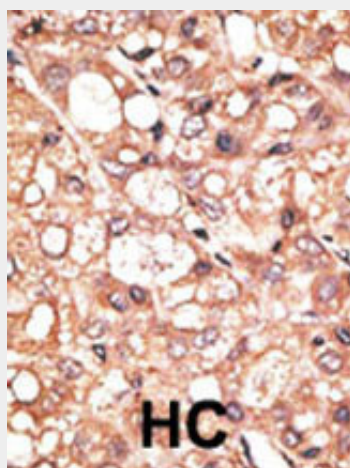
Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

LSD1 Antibody (Center) - Images



Western blot analysis of AOF2 (arrow) using LSD1 Antibody (Center) (Cat.#AP1218b). 293 cell lysates (2 ug/lane) either nontransfected (Lane 1) or transiently transfected with the AOF2 gene (Lane 2) (Origene Technologies).



Formalin-fixed and paraffin-embedded human cancer tissue reacted with the primary antibody, which was peroxidase-conjugated to the secondary antibody, followed by DAB staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated. BC = breast carcinoma; HC = hepatocarcinoma.

LSD1 Antibody (Center) - Background

LSD1 is a histone demethylase that specifically demethylates 'Lys-4' of histone H3, a specific tag for epigenetic transcriptional activation, thereby acting as a corepressor. LSD1 contains a SWIRM domain, a FAD-binding motif, and an amine oxidase domain. This protein is a component of several histone deacetylase complexes, though it silences genes by functioning as a histone demethylase. It acts by oxidizing the substrate by FAD to generate the corresponding imine that is subsequently hydrolyzed. LSD1 demethylates both mono- and tri-methylated 'Lys-4' of histone H3. This protein may play a role in the repression of neuronal genes. Alone, it is unable to demethylate H3 'Lys-4' on nucleosomes and requires the presence of RCOR1/CoREST to achieve such activity. It may also demethylate 'Lys-9' of histone H3, a specific tag for epigenetic transcriptional repression, thereby leading to derepression of androgen receptor target genes.

LSD1 Antibody (Center) - References

Forneris, F., et al. FEBS Lett. 579 (10), 2203-2207 (2005)
Shi, Y., et al. Cell 119 (7), 941-953 (2004)

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Humphrey,G.W., et al. J. Biol. Chem. 276 (9), 6817-6824 (2001)
Ota, T., et al., Nat. Genet. 36(1):40-45 (2004).