

ALDH1A1

Purified Mouse Monoclonal Antibody Catalog # AO2559a

Specification

ALDH1A1 - Product Information

Application WB, IHC, ICC, E

Primary Accession
Reactivity
Host
Clonality
Isotype
Calculated MW
P00352
Human
Mouse
Mouse
Monoclonal
Mouse IgG1
54.9kDa KDa

Immunogen

Purified recombinant fragment of human ALDH1A1 (AA: 1-110) expressed in E. Coli.

Formulation

Purified antibody in PBS with 0.05% sodium azide

ALDH1A1 - Additional Information

Gene ID 216

Other Names

ALDC; ALDH1; HEL-9; HEL12; PUMB1; ALDH11; RALDH1; ALDH-E1; HEL-S-53e

Dilution

WB~~ 1/500 - 1/2000 IHC~~ 1/200 - 1/1000 ICC~~N/A E~~ 1/10000

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

ALDH1A1 is for research use only and not for use in diagnostic or therapeutic procedures.

ALDH1A1 - Protein Information

Name ALDH1A1 (HGNC:402)

Function

Cytosolic dehydrogenase that catalyzes the irreversible oxidation of a wide range of aldehydes to their corresponding carboxylic acid (PubMed:12941160, PubMed:<a href="http://www.uniprot.org/citations/15623782"



target=" blank">15623782, PubMed:17175089, PubMed:19296407, PubMed:25450233, PubMed:26373694). Functions downstream of retinol dehydrogenases and catalyzes the oxidation of retinaldehyde into retinoic acid, the second step in the oxidation of retinol/vitamin A into retinoic acid (By similarity). This pathway is crucial to control the levels of retinol and retinoic acid, two important molecules which excess can be teratogenic and cytotoxic (By similarity). Also oxidizes aldehydes resulting from lipid peroxidation like (E)-4-hydroxynon-2-enal/HNE, malonaldehyde and hexanal that form protein adducts and are highly cytotoxic. By participating for instance to the clearance of (E)-4-hydroxynon-2-enal/HNE in the lens epithelium prevents the formation of HNE-protein adducts and lens opacification (PubMed:12941160, PubMed:15623782. PubMed:19296407). Also functions downstream of fructosamine-3-kinase in the fructosamine degradation pathway by catalyzing the oxidation of 3-deoxyglucosone, the carbohydrate product of fructosamine 3-phosphate decomposition, which is itself a potent glycating agent that may react with lysine and arginine side-chains of proteins (PubMed:17175089). Also has an aminobutyraldehyde dehydrogenase activity and is probably part of an alternative pathway for the biosynthesis of GABA/4-aminobutanoate in midbrain, thereby playing a role in GABAergic synaptic transmission (By similarity).

Cellular Location

Cytoplasm, cytosol. Cell projection, axon {ECO:0000250|UniProtKB:P24549}

Tissue Location

Expressed by erythrocytes (at protein level).

ALDH1A1 - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- <u>Immunohistochemistry</u>
- Immunofluorescence
- <u>Immunoprecipitation</u>
- Flow Cytomety
- Cell Culture

ALDH1A1 - Images



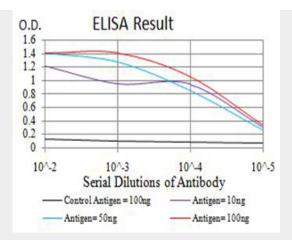


Figure 1:Black line: Control Antigen (100 ng); Purple line: Antigen (10ng); Blue line: Antigen (50 ng); Red line: Antigen (100 ng)

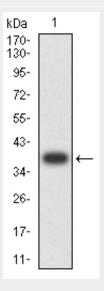


Figure 2:Western blot analysis using ALDH1A1 mAb against human ALDH1A1 (AA: 1-110) recombinant protein. (Expected MW is 38.4 kDa)

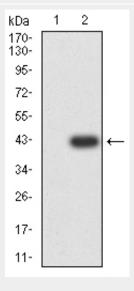


Figure 3:Western blot analysis using ALDH1A1 mAb against HEK293 (1) and ALDH1A1 (AA: 1-110)-hlgGFc transfected HEK293 (2) cell lysate.



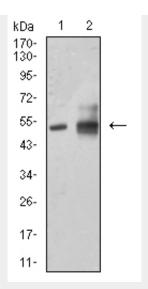


Figure 4:Western blot analysis using ALDH1A1 mouse mAb against HepG2 (1) and A549 (2) cell lysate.

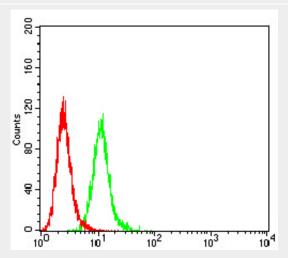


Figure 5:Flow cytometric analysis of HeLa cells using ALDH1A1 mouse mAb (green) and negative control (red).

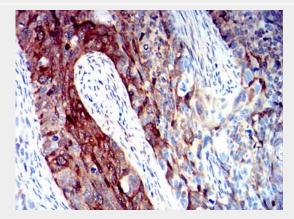


Figure 6:Immunohistochemical analysis of paraffin-embedded cervical cancer tissues using ALDH1A1 mouse mAb with DAB staining.



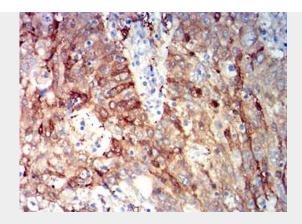


Figure 7:Immunohistochemical analysis of paraffin-embedded stomach cancer tissues using ALDH1A1 mouse mAb with DAB staining.

ALDH1A1 - References

1.Oncotarget. 2015 Dec 1;6(38):41360-9.2.Biomark Med. 2015;9(8):777-90.