Datasheet for ABIN6136575

anti-ADH1C antibody (AA 1-375)

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Overview

Quantity:	100 µL
Target:	ADH1C
Binding Specificity:	AA 1-375
Reactivity:	Human
Host:	Rabbit
Clonality:	Polyclonal
Conjugate:	This ADH1C antibody is un-conjugated
Application:	Western Blotting (WB), Immunohistochemistry (IHC)

Product Details

Immunogen:	Recombinant fusion protein containing a sequence corresponding to amino acids 1-375 of human ADH1C (NP_000660.1).
Sequence:	MSTAGKVIKC KAAVLWELKK PFSIEEVEVA PPKAHEVRIK MVAAGICRSD EHVVSGNLVT PLPVILGHEA AGIVESVGEG VTTVKPGDKV IPLFTPQCGK CRICKNPESN YCLKNDLGNP RGTLQDGTRR FTCSGKPIHH FVGVSTFSQY TVVDENAVAK IDAASPLEKV CLIGCGFSTG YGSAVKVAKV TPGSTCAVFG LGGVGLSVVM GCKAAGAARI IAVDINKDKF AKAKELGATE CINPQDYKKP IQEVLKEMTD GGVDFSFEVI GRLDTMMASL LCCHEACGTS VIVGVPPDSQ NLSINPMLLL TGRTWKGAIF GGFKSKESVP KLVADFMAKK FSLDALITNI LPFEKINEGF DLLRSGKSIR TVLTF
Isotype:	IgG
Cross-Reactivity:	Human, Mouse, Rat

Product Details

Characteristics:	Polyclonal Antibodies
Purification:	Affinity purification

Target Details

Target:	ADH1C
Alternative Name:	ADH1C (ADH1C Products)
Background:	This gene encodes class I alcohol dehydrogenase, gamma subunit, which is a member of the alcohol dehydrogenase family. Members of this enzyme family metabolize a wide variety of substrates, including ethanol, retinol, other aliphatic alcohols, hydroxysteroids, and lipid peroxidation products. Class I alcohol dehydrogenase, consisting of several homo- and heterodimers of alpha, beta, and gamma subunits, exhibits high activity for ethanol oxidation and plays a major role in ethanol catabolism. Three genes encoding alpha, beta and gamma subunits are tandemly organized in a genomic segment as a gene cluster.,ADH1C,ADH3,Signal Transduction,Cell Biology & Developmental Biology,Endocrine & Metabolism,ADH1C
Molecular Weight:	39 kDa
Gene ID:	126
UniProt:	P00326

Application Details

Application Notes:	WB,1:500 - 1:2000,IHC,1:50 - 1:200
Comment:	HIGH QUALITY
Restrictions:	For Research Use only

Handling

Format:	Liquid
Buffer:	PBS with 0.02 % sodium azide,50 % glycerol, pH 7.3.
Preservative:	Sodium azide
Precaution of Use:	This product contains Sodium azide: a POISONOUS AND HAZARDOUS SUBSTANCE which should be handled by trained staff only.
Storage:	-20 °C

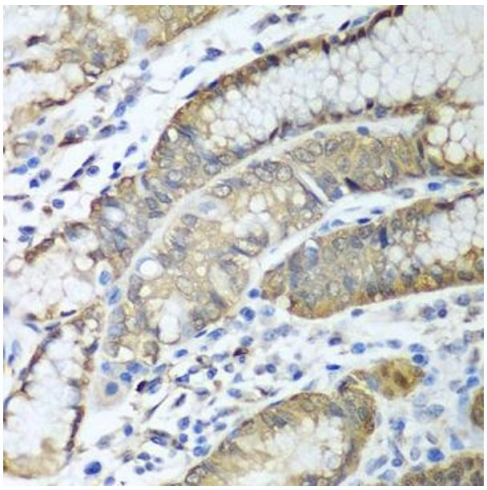
Handling

Storage Comment:	Store at -20°C. Avoid freeze / thaw cycles.

Publications

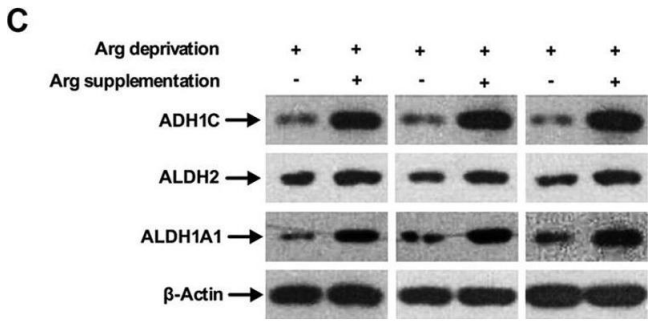
Product cited in:	Huang, Han, Sun, Zhao, Liu, Yuan, Mao, Peng, Liu, Yin, He: "Kv1.3 channel blocker (ImKTx88) maintains blood-brain barrier in experimental autoimmune encephalomyelitis." in: **Cell & bioscience**, Vol. 7, pp. 31, (2017) (PubMed).

Images



Immunohistochemistry

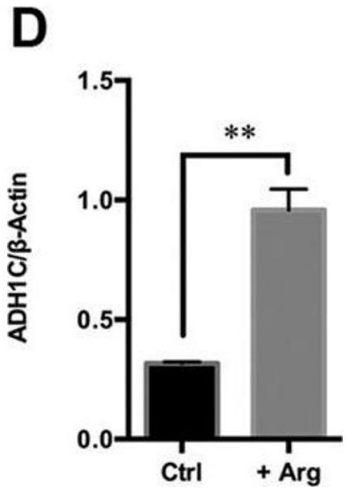
Image 1. Immunohistochemistry of paraffin-embedded human stomach using C antibody (ABIN6131394, ABIN6136575, ABIN6136576 and ABIN6224107) at dilution of 1:100 (40x lens).Perform microwave antigen retrieval with 10 mM PBS buffer pH 7.2 before commencing with IHC staining protocol.



Western Blotting

Image 2. Arg supplementation activates ethanol degradation pathways in HepG2 cells.(A) The 20 top-ranked canonical pathways based on P-values by the IPA tools. (B) Detailed information of the three top-ranked canonical pathways. (C) Western blot of ADH1C, ALDH1A1, ALDH2, and β-Actin in the HepG2 cells treated by Arg deprivation and Arg supplementation (10 mM) as indicated. (D-F) Quantification of ADH1C/β-Actin, ALDH2/β-Actin, and ALDH1A1/β-Actin as described in (C), respectively. Data are means±SD (n=3). *P<0.05, **P<0.01 (Student's t-test). (G,H) The ADH and ALDH activity in Arg-deprived (Ctrl) and Arg-supplemented (+Arg) HepG2 cells (compared to that of Ctrl) were determined by measuring the rate of NADH production at 340nm. Data are means±SD (n=4). **P<0.01 (Student's t-

test). (I) The morphological changes of the ethanol (100 mM) and/or Arg (10 mM)-treated (24h) HepG2 cells were observed by light microscopy. (J) The cytotoxicity of ethanol and/or Arg-treated HepG2 cells (as described in I) was measured by MTT assay. Data are means±SD (n=10). NS, non-significant, ****P<0.0001 (Student's t-test). - figure provided by CiteAb.



Western Blotting

Image 3. Arg supplementation activates ethanol degradation pathways in HepG2 cells.(A) The 20 top-ranked canonical pathways based on P-values by the IPA tools. (B) Detailed information of the three top-ranked canonical pathways. (C) Western blot of ADH1C, ALDH1A1, ALDH2, and β-Actin in the HepG2 cells treated by Arg deprivation and Arg supplementation (10 mM) as indicated. (D-F) Quantification of ADH1C/β-Actin, ALDH2/β-Actin, and ALDH1A1/β-Actin as described in (C), respectively. Data are means±SD (n=3). *P<0.05, **P<0.01 (Student's t-test). (G,H) The ADH and ALDH activity in Arg-deprived (Ctrl) and Arg-supplemented (+Arg) HepG2 cells (compared to that of Ctrl) were determined by measuring the rate of NADH production at 340nm. Data are means±SD (n=4). **P<0.01 (Student's t-test). (I) The morphological changes of the ethanol (100 mM) and/or Arg (10 mM)-treated (24h) HepG2 cells were observed by light microscopy. (J) The cytotoxicity of ethanol and/or Arg-treated HepG2 cells (as described in I) was measured by MTT assay. Data are means±SD (n=10). NS, non-significant, ****P<0.0001 (Student's t-test). - figure provided by CiteAb.

Please check the product details page for more images. Overall 5 images are available for ABIN6136575.