SANTA CRUZ BIOTECHNOLOGY, INC.

DDAH I (C-4): sc-271337

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

DDAH, a dimethylarginine dimethylaminohydrolase, hydrolyzes dimethyl arginine (ADMA) and monomethyl arginine (MMA), both inhibitors of nitric oxide synthases, and may be involved in *in vivo* modulation of nitric oxide production. Impairment of DDAH causes ADMA accumulation and a reduction in cGMP generation. DDAH II, the predominant DDAH isoform in endothelial cells, facilitates the induction of nitric oxide synthesis by all-*trans*-Retinoic acid (atRA). DDAH proteins are highly expressed in colon, kidney, stomach and liver tissues.

REFERENCES

1. Nakagomi, S., et al. 1999. Dimethylarginine dimethylaminohydrolase (DDAH) as a nerve-injury-associated molecule: mRNA localization in the rat brain and its coincident up-regulation with neuronal NO synthase (nNOS) in axotomized motoneurons. Eur. J. Neurosci. 11: 2160-2166.
2. Knipp, M., et al. 2001. Structural and functional characterization of the Zn(II) site in dimethylargininase-1 (DDAH-1) from bovine brain. Zn(II) release activates DDAH-1. J. Biol. Chem. 276: 40449-40456.

CHROMOSOMAL LOCATION

Genetic locus: DDAH1 (human) mapping to 1p22.3; Ddah1 (mouse) mapping to 3 H2.

SOURCE

DDAH I (C-4) is a mouse monoclonal antibody specific for an epitope mapping between amino acids 198-237 within an internal region of DDAH I of human origin.

PRODUCT

Each vial contains 200 µg IgG_1 kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-271337 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% stabilizer protein).

APPLICATIONS

DDAH I (C-4) is recommended for detection of DDAH I of mouse, rat and human origin by Western Blotting (starting dilution 1:100, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for DDAH I siRNA (h): sc-105276, DDAH I siRNA (m): sc-142914, DDAH I shRNA Plasmid (h): sc-105276-SH, DDAH I shRNA Plasmid (m): sc-142914-SH, DDAH I shRNA (h) Lentiviral Particles: sc-105276-V and DDAH I shRNA (m) Lentiviral Particles: sc-142914-V.

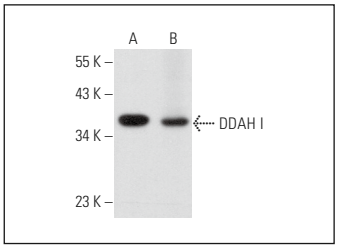
Molecular Weight of DDAH I: 31 kDa.

Positive Controls: KNRK whole cell lysate: sc-2214, human liver extract: sc-363766 or human cerebral cortex extract: sc-516707.

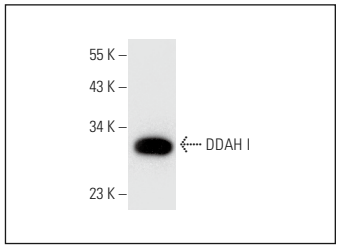
RECOMMENDED SUPPORT REAGENTS

To ensure optimal results, the following support reagents are recommended: 1) Western Blotting: use m-IgGκ BP-HRP: sc-516102 or m-IgGκ BP-HRP (Cruz Marker): sc-516102-CM (dilution range: 1:1000-1:10000), Cruz Marker™ Molecular Weight Standards: sc-2035, UltraCruz® Blocking Reagent: sc-516214 and Western Blotting Luminol Reagent: sc-2048. 2) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml). 3) Immunofluorescence: use m-IgGκ BP-FITC: sc-516140 or m-IgGκ BP-PE: sc-516141 (dilution range: 1:50-1:200) with UltraCruz® Mounting Medium: sc-24941 or UltraCruz® Hard-set Mounting Medium: sc-359850.

DATA



DDAH I (C-4): sc-271337. Western blot analysis of DDAH I expression in human cerebral cortex (**A**) and human liver (**B**) tissue extracts.



DDAH I (C-4): sc-271337. Western blot analysis of DDAH I expression in KNRK whole cell lysate.

SELECT PRODUCT CITATIONS

1. Osorio-Yáñez, C., et al. 2017. The ADMA/DDAH/NO pathway in human vein endothelial cells exposed to arsenite. Toxicol. In Vitro 42: 281-286.
2. Xie, Z., et al. 2022. Mechanical force promotes dimethylarginine dimethylaminohydrolase 1-mediated hydrolysis of the metabolite asymmetric dimethylarginine to enhance bone formation. Nat. Commun. 13: 50.
3. Zhong, Y., et al. 2022. Inhibition of miR-21 improves pulmonary vascular responses in bronchopulmonary dysplasia by targeting the DDAH1/ADMA/NO pathway. Open Med. 17: 1949-1964.
4. Tain, Y.L., et al. 2023. Anti-hypertensive property of an NO nanoparticle in an adenine-induced chronic kidney disease young rat model. Antioxidants 12: 513.
5. Wu, M., et al. 2023. LncRNA DANCR deficiency promotes high glucose-induced endothelial to mesenchymal transition in cardiac microvascular cells via the FoxO1/DDAH1/ADMA signaling pathway. Eur. J. Pharmacol. 950: 175732.

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

RESEARCH USE

For research use only, not for use in diagnostic procedures.