SANTA CRUZ BIOTECHNOLOGY, INC.

DDAH II (3E3): sc-293229

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.
3. Leiper, J., et al. 2002. S-nitrosylation of dimethylarginine dimethylaminohydrolase regulates enzyme activity: further interactions betwen nitric oxide synthase and dimethylarginine dimethylaminohydrolase. Proc. Natl. Acad. Sci. USA 99: 13527-13532.
4. Lin, K.Y., et al. 2002. Impaired nitric oxide synthase pathway in diabetes mellitus: role of asymmetric dimethylarginine and dimethylaminohydrolase. Circulation 106: 987-992.
5. Achan, V., et al. 2002. All-*trans*-Retinoic acid increases nitric oxide synthesis by endothelial cells: a role for the induction of dimethylarginine dimethylaminohydrolase. Circ. Res. 90: 764-769.
6. Knipp, M., et al. 2003. Zn(II)-free dimethylargininase-1 (DDAH-1) is inhibited upon specific Cys-S-nitrosylation. J. Biol. Chem. 278: 3410-3416.
7. Swiss-Prot/TrEMBL (O94760). World Wide Web URL: http://www.expasy.ch/sprot/sprot-top.html

CHROMOSOMAL LOCATION

Genetic locus: DDAH2 (human) mapping to 6p21.33.

SOURCE

DDAH II (3E3) is a mouse monoclonal antibody raised against amino acids 1-285 representing full length DDAH II of human origin.

PRODUCT

Each vial contains 100 µg IgG_{2b} kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

STORAGE

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

APPLICATIONS

DDAH II (3E3) is recommended for detection of DDAH II of human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)] and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

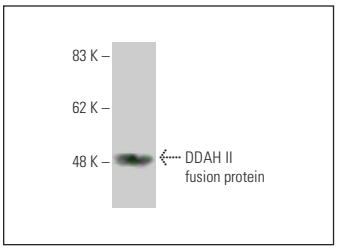
Suitable for use as control antibody for DDAH II siRNA (h): sc-40474, DDAH II shRNA Plasmid (h): sc-40474-SH and DDAH II shRNA (h) Lentiviral Particles: sc-40474-V.

Molecular Weight of DDAH II: 30 kDa.

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).

DATA



DDAH II (3E3): sc-293229. Western blot analysis of human recombinant DDAH II fusion protein.

RESEARCH USE

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

PROTOCOLS

See our web site at www.scbt.com for detailed protocols and support products.