Recombinant human DDAH1 protein with GST tag (rhDDAH1-GST), active.

Catalog Number: 4001-20UG, 4001-50UG, 4001-100UG

Produced and purified from E. coli

Product Description

Name: **N(G),N(G)-dimethylarginine dimethylaminohydrolase 1**

Synonyms: Dimethylargininase-1

Species: Human

Amino acids: 2 to 285

Predicted Molecular Weight: 58.1 kDa with GST tag

Protein ID: O94760 (DDAH1_HUMAN)

AA Sequence:

MAGLGHPAAFGRATHAVVRALPESLGQHALRSAKGEEVDVARAERQHQLYVGVLGSKLGLQVVELPADESLPDCVFVED
VAVVCEETALITRPGAPSRRKEVDMMKEALEKLQLNIVEMKDENATLDGGDVLFTGREFFVGLSKRTNQRGAEILADTF
KDYAVSTVPVADGLHLKSFCSMAGPNLIAIGSSESAQKALKIMQQMSDHRYDKLTVPDDIAANCIYLNIPNKGHVLLHR
TPEEYPESAKVYEKLKDHMLIPVSMSELEKVDGLLTCCSVLINKKVDS*

**Recombinant proteins are expressed from synthetic genes. DAPCEL Inc. synthetic gene design technology provides highest protein quality in terms of protein folding and bioactivity.*

Product specifications

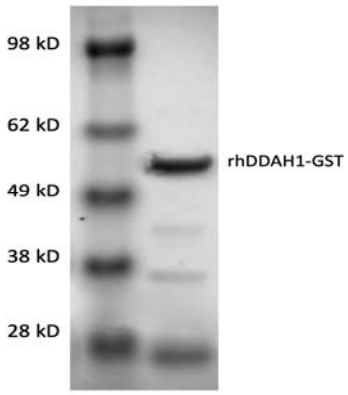
Estimated Molecular Weight, SDS-PAGE: 58 kDa (shown below).

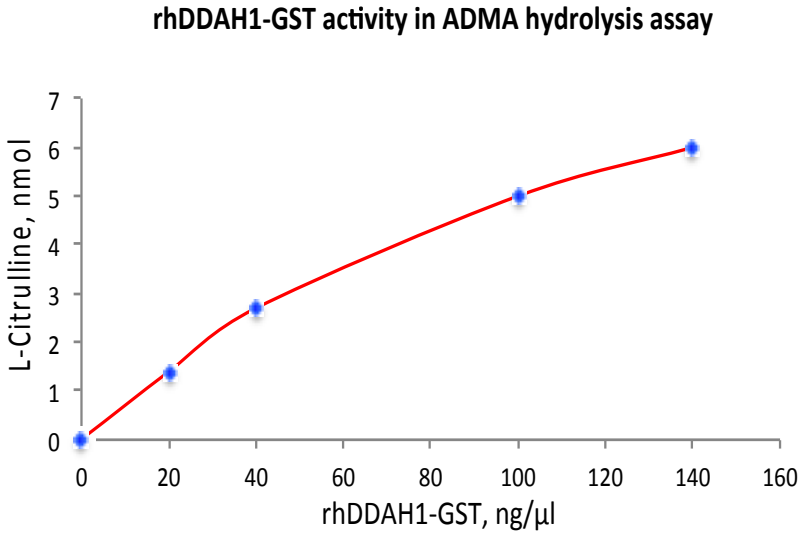
Grade & Purity: >85%, (according to SDS-PAGE stained with SimplyBlue SafeStain, (Invitrogen)).

Endotoxins: Less than 0.1 ng/μg (1 IEU/μg), as measured by LAL method.

Bioactivity: >50 pmol/min/μg, tested in ADMA hydrolysis assay (see below).

Formulation: 0.5mg/mL in 50mM Tris-HCl pH 8.0, 20mM reduced glutathione, 1mM DTT; frozen.





H2O2 + Nω,Nω-dimethyl-L-arginine = dimethylamine + L-citrulline

Shipping

Product is shipped on dry ice. Upon receipt, store at -80°C.

Storage

To avoid loss of the protein, store the protein in aliquots at -80°C. Avoid repeated freeze-thaw cycles.

Stability

12 months from the date of receipt, stored at -20 to -80 °C as supplied.

Application Note: *For research purposes only. Not for use in humans.*

N(G),N(G)-dimethylarginine dimethylaminohydrolase 1, background information:

DDAH1 is the major enzyme that degrades the endogenous nitric oxide (NO) synthase inhibitor asymmetric dimethylarginine (ADMA). It catalyzes the chemical reaction ADMA + H2O2 + Nω,Nω-dimethyl-L-arginine = dimethylamine + L-citrulline. In DDAH1 transgenic mice, ADMA is decreased while NO production and angiogenesis are increased (1). Conversely, in the DDAH1 KO mice, ADMA was increased with a consequent decrease in NO production and angiogenesis (2). An impairment of DDAH activity results in the elevation of plasma ADMA, and impairment of vascular relaxation observed in humans with cardiovascular diseases. The importance of the DDAH/ADMA pathway on angiogenesis has been demonstrated in a number of systems. DDAH1 transgenic mice show increased blood vessel formation in a mouse model of hind limb ischemia as well as in the fibro-vascular disc system (1, 3). The DDAH/ADMA pathway has been considered as a potential therapeutic target to treat cardiovascular disease, lung disease, renal disease and cancer (4).

1. Jacobi et al. (2005) *Circulation* 111: 1431–1438.
2. Hu et al. (2011) *Arterioscler Thromb Vasc Biol* 31(7): 1540–6.
3. Achan et al. (2005) *Vasc Med* 10: 7–14.
4. Leiper, Nandi (2011) *Nat Rev Drug Discov* 10(4): 277–91