PRODUCT INFORMATION

eNOS (bovine, recombinant; *E. coli* expressed)

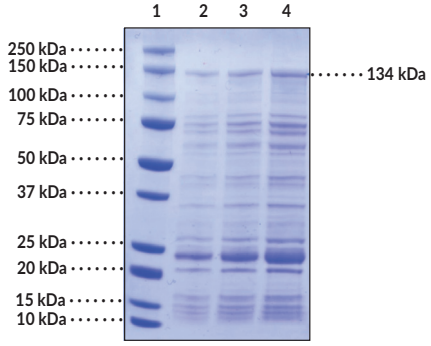
Item No. 11868

Overview and Properties

Synonyms:	Constitutive NOS, cNOS, Endothelial NOS, Endothelial Nitric Oxide Synthase, Nitric Oxide Synthase 3
Source:	Active recombinant bovine N-terminal His-tagged eNOS expressed in *E. coli*
Amino Acids:	2-1,205 (full length)
Uniprot No.:	P29473
Molecular Weight:	134 kDa
Storage:	-80°C (as supplied)
Stability:	≥1 year
Purity:	Partially pure
Supplied in:	50 mM HEPES, pH 7.8, with 0.5 mM DTT, 0.5 mM EDTA, 300 mM sodium chloride, 2 mM calcium chloride, 10 µM tetrahydro-L-biopterin (hydrochloride), and 20% glycerol
Protein Concentration:	***batch specific*** mg/ml
Activity:	***batch specific*** U/ml
Specific Activity:	***batch specific*** U/mg
Unit Definition:	One unit of enzyme produces 1 nmol of nitric oxide per minute at 37°C in 50 mM HEPES, pH 7.8, with 5 µM oxyhemoglobin, 1 mM calcium chloride, 20 µg/ml calmodulin, 0.1 mM NADPH, 50 µM arginine, 12 µM tetrahydrobiopterin, and 170 µM DTT.

Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

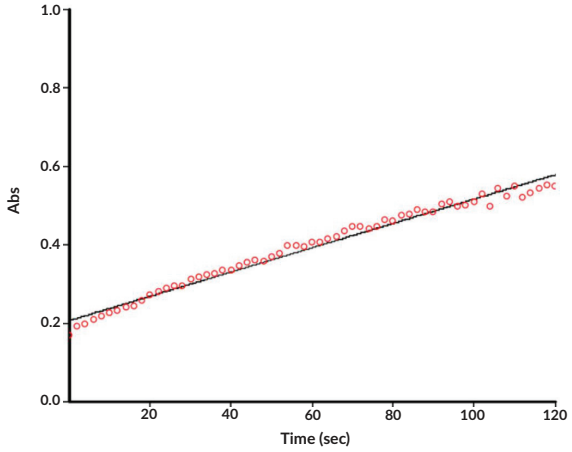
Images



Lane 1: MW Markers
Lane 2: eNOS (2 µg)
Lane 3: eNOS (4 µg)
Lane 4: eNOS (8 µg)

SDS-PAGE Analysis of eNOS.

Representative gel image shown; actual purity may vary between each batch.



eNOS (bovine, recombinant; *E. coli* expressed) Activity

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PRODUCT INFORMATION

Description

Endothelial nitric oxide synthase (eNOS), also known as NOS3, converts arginine to citrulline and nitric oxide (NO) to regulate endothelial homeostasis and vascular tone.[1-3] It is composed of an N-terminal oxygenase domain that contains binding sites for heme, its substrate L-arginine (Item No. 23703), and the cofactor tetrahydrobiopterin (BH_4) and a reductase domain with binding sites for NADPH, flavin mononucleotide, flavin-adenine dinucleotide, and calmodulin.[3] eNOS is expressed in endothelial cells, as well as blood cells and circulating microparticles, with expression levels changing in response to growth factors, cytokines, and mechanical stimulation.[1,2] Localization of eNOS is mediated by protein fatty acid acylation, with myristoylated and palmitoylated eNOS targeted to plasma membrane caveolae and the Golgi complex. eNOS functions as a homodimer and is activated by the binding of calcium-bound calmodulin. eNOS uncoupling and dysfunction, primarily induced by the loss of BH_4 due to oxidation or decreased expression of dihydrofolate reductase (DHFR), induces the production of superoxide in place of NO and is a hallmark of most cardiovascular diseases, including atherosclerosis.[4,5] Cayman's eNOS (bovine, recombinant; *E. coli* expressed) can be used for enzyme assay applications.

References

1. Qian, J. and Fulton, D. Post-translational regulation of endothelial nitric oxide synthase in vascular endothelium. *Front. Physiol.* **4**, 347 (2013).
2. Heiss, C., Rodriguez-Mateos, A., and Kelm, M. Central role of eNOS in the maintenance of endothelial homeostasis. *Antioxid. Redox Signal.* **22(14)**, 1230-1242 (2015).
3. Siragusa, M. and Fleming, I. The eNOS signalosome and its link to endothelial dysfunction. *Pflugers Arch.* **468(7)**, 1125-1137 (2016).
4. Daiber, A., Xia, N., Steven, S., *et al.* New therapeutic implications of endothelial nitric oxide synthase (eNOS) function/dysfunction in cardiovascular disease. *Int. J. Mol. Sci.* **20(1)**, 187 (2019).
5. Roe, N.D. and Ren, J. Nitric oxide synthase uncoupling: A therapeutic target in cardiovascular diseases. *Vascul. Pharmacol.* **57(5-6)**, 168-172 (2012).

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