

## NOS3

**Reactivity:**Human Bovine

**Tested applications:**WB IP

**Recommended Dilution:**WB 1:500 - 1:1000 IP 1:20 - 1:50

**Calculated MW:**133kDa

**Observed MW:**Refer to Figures

**Immunogen:**

A synthetic peptide of human NOS3

**Storage Buffer:**

Store at -20. Avoid freeze / thaw cycles. Buffer: PBS with 0.02% sodium azide, 50% glycerol, pH7.3.

**Concentration:**

a

**Synonym:**

eNOS; ECNOS;

**Catalog #:**A2459

**Antibody Type:**

Polyclonal Antibody

**Species:**Rabbit

**Gene ID:**4846

**Isotype:**IgG

**Swiss Prot:**P29474

**Purity:**Affinity purification

For research use only.

**Background:**

Endothelial nitric-oxide synthase (eNOS) is an important enzyme in the cardiovascular system. It catalyzes the production of nitric oxide (NO), a key regulator of blood pressure, vascular remodeling, and angiogenesis (1,2). The activity of eNOS is regulated by phosphorylation at multiple sites. The two most thoroughly studied sites are the activation site Ser1177 and the inhibitory site Thr495 (3). Several protein kinases including Akt/PKB, PKA, and AMPK activate eNOS by phosphorylating Ser1177 in response to various stimuli (4,5). In contrast, bradykinin and H<sub>2</sub>O<sub>2</sub> activate eNOS activity by promoting both Ser1177 phosphorylation and Thr495 dephosphorylation (6,7). eNOS is activated by VEGF, and this activation is associated with dephosphorylation of eNOS at serine 113. Cyclosporin A blocks this dephosphorylation of eNOS upon VEGF stimulation (8).

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