Product Data Sheet: Phospho-NOS3 (S1177)

Cat. No:	ABP-0421
Conjugate:	Unconjugated
Size:	100 ug
Clone:	Poly
Concentration:	1mg/ml
Host:	Rb
Isotype:	IgG
Reactivity:	Hu, Ms, Rt
Applications:	WB 1:1000, IF 1:200-1:1000
Molecular Weight:	140 kDa
Purification:	Polyclonal antibodies are produced by immunizing rabbits with a synthetic phospho-peptide (KLH-coupled) corresponding to residues surrounding Ser1177 of human eNOS. Antibodies are purified by protein A and peptide affinity chromatography.
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 hydrogen peroxide activate eNOS activity by promoting Thr495 dephosphorylation (6,7).Phospho-eNOS (Ser1177) Antibody detects endogenous levels of eNOS only when phosphorylated at Ser1177.
Form:	liquid
Storage:	Store at -20°C, and avoid repeat freezethaw cycles.

References

Selected Application References: Brouet, A. et al. (2001) Hsp90 ensures the transition from the early Ca2+-dependent to the late phosphorylation-dependent activation of the endothelial nitric-oxide synthase in vascular endothelial growth factor-exposed endothelial cells. J. Biol. Chem. 276, 32663–32669. Application: W. Thomas, S.R. et al. (2002) Hydrogen peroxide activates endothelial nitric-oxide synthase through coordinated phosphorylation and dephosphorylation via a phosphoinositide 3-kinase-dependent signaling pathway. J. Biol. Chem. 277, 6017–6024. Application: W. Du, X.L. et al. (2001) Hyperglycemia inhibits endothelial nitric oxide synthase activity by posttranslational modification at the Akt site. J. Clin. Invest. 108, 1341–1348. Application: W. Boo, Y.C. et al. (2002) Shear stress stimulates phosphorylation of endothelial nitric-oxide synthase at Ser1179 by Akt-independent mechanisms: role of protein kinase A. J. Biol. Chem. 277, 3388–3396. Application: W.Background References: (1) Fulton, D. et al. (2001) J. Pharmacol. Exp. Ther. 299, 818–824. (2) Shaul, P.W. (2002) Annu. Rev. Physiol. 64, 749–774. (3) Chen, Z.P. et al. (1999) FEBS Lett. 443, 285–289. (4) Dimmeler, S. et al. (1999) Nature 399, 601–605. (5) Fulton, D. et al. (1999) Nature 399, 597–601. (6) Harris, M.B. et al. (2001) J. Biol. Chem. 276, 16587–16591. (7) Thomas, S.R. et al. (2002) J. Biol. Chem. 277, 6017–6024.

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