SANTA CRUZ BIOTECHNOLOGY, INC.

p-NOS3 (pT495.33): sc-136519

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Nitric oxide (NO), produced by the endothelial NO synthase (NOS3), is a fundamental determinant of cardiovascular homeostasis that maintains system blood pressure, vascular remodeling and angiogenesis. NOS3 is stimulated, in a phosphatidylinositol 3-kinase (PI 3-kinase)-dependent fashion, by treatment of endothelial cells with Insulin-like growth factor-1 and vascular endothelial growth factor (VEGF). The serine/threonine protein kinase Akt/PKB is an important downstream target of PI 3-kinase, regulating VEGF-stimulated endothelial cell survival. NOS3 activation via phosphorylation of Serine 1177 by Akt/PKB is necessary and sufficient for VEGF-mediated endothelial cell migration. Therefore, Akt/PKB can directly phosphorylate NOS3 on Serine 1177, activating the enzyme and leading to NO production. Phosphorylation of NOS3 on Threonine 495 is mediated by PKC, and is increased due to VEGF stimulation. NOS3 activity may be regulated through complex events mediated by multiple kinases at various phosphorylation sites, including Serine 495, Serine 633 and Serine 1177.

REFERENCES

1. Rudic, R.D., et al. 1998. Direct evidence for the importance of endothelium-derived nitric oxide in vascular remodeling. J. Clin. Invest. 101: 731-736.
2. Murohara, T., et al. 1998. Nitric oxide synthase modulates angiogenesis in response to tissue ischemia. J. Clin. Invest. 101: 2567-2578.
3. Fulton, D., et al. 1999. Regulation of endothelium-derived nitric oxide production by the protein kinase Akt. Nature 399: 597-601.
4. Dimmeler, S., et al. 1999. Activation of nitric oxide synthase in endothelial cells by Akt-dependent phosphorylation. Nature 399: 601-605.
5. Chen, Z.P., et al. 1999. AMP-activated protein kinase phosphorylation of endothelial NO synthase. FEBS Lett. 443: 285-289.

CHROMOSOMAL LOCATION

Genetic locus: NOS3 (human) mapping to 7q36.1; Nos3 (mouse) mapping to 5 A3.

SOURCE

p-NOS3 (pT495.33) is a mouse monoclonal antibody raised against a short amino acid sequence containing Thr 495 phosphorylated NOS3 of human origin.

PRODUCT

Each vial contains 200 µg IgG_{2a} kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin. Also available as TransCruz reagent for Gel Supershift and ChIP applications, sc-136519 X, 200 µg/0.1 ml.

p-NOS3 (pT495.33) is available conjugated to agarose (sc-136519 AC), 500 µg/0.25 ml agarose in 1 ml, for IP; and to HRP (sc-136519 HRP), 200 µg/ml, for WB, IHC(P) and ELISA.

STORAGE

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

APPLICATIONS

p-NOS3 (pT495.33) is recommended for detection of Thr 495 phosphorylated NOS3 of human origin and correspondingly Thr 492 phosphorylated NOS3 of mouse and rat origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000) and immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)].

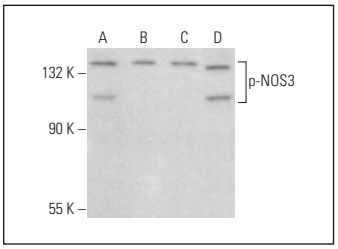
p-NOS3 (pT495.33) is also recommended for detection of Thr 495 phosphorylated NOS3 in additional species, including canine.

Suitable for use as control antibody for NOS3 siRNA (h): sc-36093, NOS3 siRNA (m): sc-36094, NOS3 siRNA (r): sc-270518, NOS3 shRNA Plasmid (h): sc-36093-SH, NOS3 shRNA Plasmid (m): sc-36094-SH, NOS3 shRNA Plasmid (r): sc-270518-SH, NOS3 shRNA (h) Lentiviral Particles: sc-36093-V, NOS3 shRNA (m) Lentiviral Particles: sc-36094-V and NOS3 shRNA (r) Lentiviral Particles: sc-270518-V.

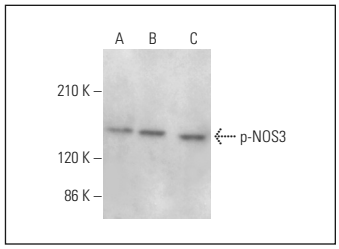
p-NOS3 (pT495.33) X TransCruz antibody is recommended for Gel Supershift and ChIP applications.

Molecular Weight of p-NOS3: 140 kDa.

DATA



p-NOS3 (pT495.33): sc-136519. Western blot analysis of NOS3 phosphorylation in CCRF-CEM (**A**), Jurkat (**B**), BJAB (**C**) and TK-1 (**D**) whole cell lysates.



p-NOS3 (pT495.33): sc-136519. Western blot analysis of NOS3 phosphorylation in JAR (**A**), HEK293 (**B**) and NIH/3T3 (**C**) whole cell lysates.

SELECT PRODUCT CITATIONS

1. Ansari, M.A., et al. 2014. A time course of NADPH-oxidase up-regulation and endothelial nitric oxide synthase activation in the hippocampus following neurotrauma. Free Radic. Biol. Med. 77: 21-29.
2. Enkhjargal, B., et al. 2017. Intranasal administration of vitamin D attenuates blood-brain barrier disruption through endogenous upregulation of osteopontin and activation of CD44/P-gp glycosylation signaling after subarachnoid hemorrhage in rats. J. Cereb. Blood Flow Metab. 37: 2555-2566.
3. Reyes-Goya, C., et al. 2020. Mechanism of vascular toxicity in rats subjected to treatment with a tyrosine kinase inhibitor. Toxics 8: 49.
4. Wang, Y., et al. 2022. mTOR contributes to endothelium-dependent vasorelaxation by promoting eNOS expression and preventing eNOS uncoupling. Commun. Biol. 5: 726.

RESEARCH USE

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