SANTA CRUZ BIOTECHNOLOGY, INC.

p-Adducin (Thr 445): sc-16738

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Adducins are a family of cytoskeleton proteins encoded by three genes (α, β and γ). Adducin is a protein associated with the inner leaflet of the plasma membrane and is one of the proteins localized at the spectrin-actin junction of the membrane skeleton. Adducins promote association of spectrin with actin and cap the fast growing end of actin filaments. Adducins contain an N-terminal core, neck and C-terminal tail domains, are substrates for protein kinase A (PKA) and C (PKC), and bind to Ca^{2+}/calmodulin. The major phosphorylation sites common to the Adducins are Ser 726 and Ser 713 in the C-terminal MARCKS-related domains of Adducin α and Adducin β, which are phosphorylated by PKA and PKC, respectively. In addition, PKA phosphorylates Adducin α at Ser 408, 436 and 481. Calmodulin-binding is inhibited by phosphorylation of Adducin β. Calmodulin itself inhibits the rate of phosphorylation of Adducin β, but not Adducin α. Rho-kinase can phosphorylate Adducin α at Thr 445 and Thr 480 downstream of Rho *in vivo*. The phosphorylation of Adducin by Rho-kinase plays an important role in the regulation of membrane ruffling and cell motility. In addition, phosphorylation at Ser 726 of Adducin α is required for cleavage by caspase-3 .

REFERENCES

1. Matsuoka, Y., et al. 1996. Adducin regulation. Definition of the calmodulin-binding domain and sites of phosphorylation by protein kinases A and C. J. Biol. Chem. 271: 25157-25166.
2. Fukata, Y., et al. 1999. Phosphorylation of Adducin by Rho-kinase plays a crucial role in cell motility. J. Cell Biol. 145: 347-361.
3. Gilligan, D.M., et al. 1999. Targeted disruption of the β Adducin gene (Add2) causes red blood cell spherocytosis in mice. Proc. Natl. Acad. Sci. USA 96: 10717-10722.
4. Muro, A.F., et al. 2000. Mild spherocytic hereditary elliptocytosis and altered levels of α and γ Adducins in β Adducin-deficient mice. Blood 95: 3978-3985.

CHROMOSOMAL LOCATION

Genetic locus: ADD2 (human) mapping to 2p13.3; Add3 (mouse) mapping to 19 D2.

SOURCE

p-Adducin (Thr 445) is available as either goat (sc-16738) or rabbit (sc-16738-R) polyclonal affinity purified antibody raised against a short amino acid sequence containing Thr 445 phosphorylated Adducin of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-16738 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

STORAGE

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

APPLICATIONS

p-Adducin (Thr 445) is recommended for detection of Thr 445 phosphorylated Adducin of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

p-Adducin (Thr 445) is also recommended for detection of correspondingly phosphorylated Adducin in additional species, including equine, canine, bovine, porcine and avian.

Molecular Weight of p-Adducin α: 120 kDa.

Molecular Weight of p-Adducin β: 97 kDa.

Molecular Weight of p-Adducin γ: 94 kDa.

Positive Controls: NIH/3T3 whole cell lysate: sc-2210, K-562 whole cell lysate: sc-2203 or K-562 + PMA cell lysate: sc-2280.

SELECT PRODUCT CITATIONS

1. Satoh, S., et al. 2003. Chronic inhibition of Rho kinase blunts the process of left ventricular hypertrophy leading to cardiac contractile dysfunction in hypertension-induced heart failure. J. Mol. Cell. Cardiol. 35: 59-70.
2. Bao, W., et al. 2004. Inhibition of Rho-kinase protects the heart against ischemia/reperfusion injury. Cardiovasc. Res. 61: 548-558.
3. Yagita, Y., et al. 2007. Rho-kinase activation in endothelial cells contributes to expansion of infarction after focal cerebral ischemia. J. Neurosci. Res. 85: 2460-2469.
4. Yamashita, K., et al. 2007. Fasudil, a Rho kinase (ROCK) inhibitor, protects against ischemic neuronal damage *in vitro* and *in vivo* by acting directly on neurons. Brain Res. 1154: 215-224.

RESEARCH USE

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

PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.