SANTA CRUZ BIOTECHNOLOGY, INC.

p120 (S-19): sc-1101

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

The catenins, α, β and γ, are proteins which bind to the highly conserved, intracellular cytoplasmic tail of E-cadherin. Together, the catenin/cadherin complexes play an important role mediating cellular adhesion. α-catenin was initially described as an E-cadherin-associated protein and has been shown to associate with other members of the cadherin family, N-cadherin and P-cadherin. β-catenin associates with the cytoplasmic portion of E-cadherin which is necessary for the function of E-cadherin as an adhesion molecule. β-catenin has also been found in complexes with the tumor suppressor protein APC. γ-catenin, also known as plakoglobin, binds with α-catenin and N-cadherin. A related protein, p120, exhibits sequence homology with the catenins at four discreet domains. p120 not only serves as a substrate for Src, but is also found in E-cadherin complexes containing catenins.

CHROMOSOMAL LOCATION

Genetic locus: CTNND1 (human) mapping to 11q12.1; Ctnnd1 (mouse) mapping to 2 D.

SOURCE

p120 (S-19) is an affinity purified rabbit polyclonal antibody raised against a peptide mapping at the C-terminus of p120 of mouse 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-1101 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

p120 (S-19) is recommended for detection of p120 of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], 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).

p120 (S-19) is also recommended for detection of p120 in additional species, including equine, canine and porcine.

Suitable for use as control antibody for p120 siRNA (h): sc-36139, p120 siRNA (m): sc-36140, p120 siRNA (r): sc-106992, p120 shRNA Plasmid (h): sc-36139-SH, p120 shRNA Plasmid (m): sc-36140-SH, p120 shRNA Plasmid (r): sc-106992-SH, p120 shRNA (h) Lentiviral Particles: sc-36139-V, p120 shRNA (m) Lentiviral Particles: sc-36140-V and p120 shRNA (r) Lentiviral Particles: sc-106992-V.

Molecular Weight of p120: 100-120 kDa.

Positive Controls: NIH/3T3 whole cell lysate: sc-2210, RAW 264.7 whole cell lysate: sc-2211 or HeLa whole cell lysate: sc-2200.

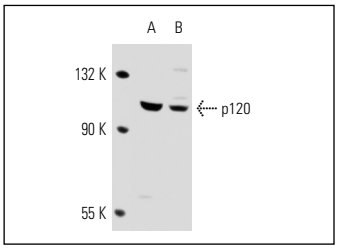
RESEARCH USE

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

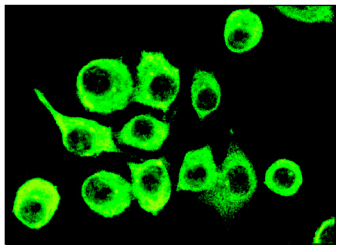
STORAGE

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

DATA



p120 (S-19): sc-1101. Western blot analysis of p120 expression in HeLa (**A**) and NIH/3T3 (**B**) whole cell lysates.



p120 (S-19): sc-1101. Immunofluorescence staining of methanol-fixed NIH/3T3 cells showing cytoplasmic localization.

SELECT PRODUCT CITATIONS

1. Joseloff, E., et al. 2002. Src family kinases phosphorylate protein kinase C on tyrosine residues and modify the neoplastic phenotype of skin keratinocytes. J. Biol. Chem. 277: 12318-12323.
2. Wong, C.H., et al. 2004. Regulation of blood-testis barrier dynamics: an *in vivo* study. J. Cell Sci. 117: 783-798.
3. Potter, M., et al. 2005. Tyrosine phosphorylation of VE-cadherin prevents binding of p120- and β-catenin and maintains the cellular mesenchymal state. J. Biol. Chem. 280: 31906-31912.
4. Sarkar, O., et al. 2006. Adjudin-mediated junction restructuring in the seminiferous epithelium leads to displacement of soluble guanylate cyclase from adherens junctions. J. Cell. Physiol. 208: 175-187.
5. Sarrió, D., et al. 2009. Functional characterization of E- and P-cadherin in invasive breast cancer cells. BMC Cancer 9: 74.
6. Asgarian, N., et al. 2010. Learning to predict relapse in invasive ductal carcinomas based on the subcellular localization of junctional proteins. Breast Cancer Res. Treat. 121: 527-538.
7. Adam, A.P., et al. 2010. Src-induced tyrosine phosphorylation of VE-cadherin is not sufficient to decrease barrier function of endothelial monolayers. J. Biol. Chem. 285: 7045-7055.
8. Humtsoe, J.O., et al. 2010. Lipid phosphate phosphatase 3 stabilization of β-catenin induces endothelial cell migration and formation of branching point structures. Mol. Cell. Biol. 30: 1593-1606.
9. Oas, R.G., et al. 2013. p120-catenin and β-catenin differentially regulate cadherin adhesive function. Mol. Biol. Cell 24: 704-714.