SANTA CRUZ BIOTECHNOLOGY, INC.

p-Rb (Thr 356)-R: sc-16837-R

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Pediatric cancer retinoblastoma and the formation of other human tumors can be attributed to mutations in the retinoblastoma tumor suppressor gene (Rb). The Rb protein regulates differentiation, apoptosis and cell cycle control by coordinating the cell cycle at G_1-S with transcriptional machinery. During G_1, cyclin D-dependent kinase-mediated phosphorylation of Rb at Ser 795 marks the conversion of Rb from a transcriptionally repressive, hypophosphorylated state to an inactive, phosphorylated state, which may be sustained through mitosis by differential phosphorylation of up to 16 putative serine or threonine residues, including Ser 249/Thr 252, Thr 373, Thr 356, Ser 780, Ser 807/Ser 811, and Thr 821/Thr 826. Hypophosphorylated Rb represses the transcription of genes controlling the cell cycle through direct protein-protein interactions and through the recruitment of histone deacetylase.

REFERENCES

1. Weinberg, R.A. 1995. The retinoblastoma protein and cell cycle control. Cell 81: 323-330.
2. Bremner, R., et al. 1995. Direct transcriptional repression by pRb and its reversal by specific cyclins. Mol. Cell. Biol. 15: 3256-3265.
3. Sherr, C.J. 1996. Cancer cell cycles. Science 274: 1672-1677.
4. Connell-Crowley, L., et al. 1997. Cyclin D1/Cdk4 regulates retinoblastoma protein-mediated cell cycle arrest by site-specific phosphorylation. Mol. Biol. Cell 8: 287-301.
5. Luo, R.X., et al. 1998. Rb interacts with histone deacetylase to repress transcription. Cell 92: 463-473.
6. Driscoll, B., et al. 1999. Discovery of a regulatory motif that controls the exposure of specific upstream cyclin-dependent kinase sites that determine both conformation and growth suppressing activity of pRb. J. Biol. Chem. 274: 9463-9471.
7. Barrientes, S., et al. 2000. Glutamic acid mutagenesis of retinoblastoma protein phosphorylation sites has diverse effects on function. Oncogene 19: 562-570.

CHROMOSOMAL LOCATION

Genetic locus: RB1 (human) mapping to 13q14.2.

SOURCE

p-Rb (Thr 356)-R is a rabbit polyclonal antibody raised against a short amino acid sequence containing Thr 356 phosphorylated Rb 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-16837 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

RESEARCH USE

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

APPLICATIONS

p-Rb (Thr 356)-R is recommended for detection of Thr 356 phosphorylated Rb of 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), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

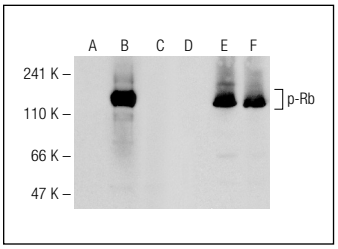
Suitable for use as control antibody for Rb siRNA (h): sc-29468, Rb shRNA Plasmid (h): sc-29468-SH and Rb shRNA (h) Lentiviral Particles: sc-29468-V.

Molecular Weight (predicted) of p-Rb: 106 kDa.

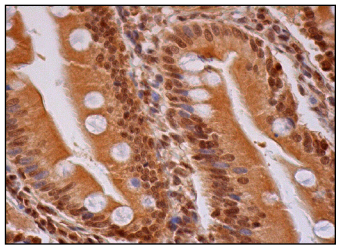
Molecular Weight (observed) of p-Rb: 107-140 kDa.

Positive Controls: Rb (h): 293T Lysate: sc-114014, SK-LMS-1 cell lysate: sc-3813 or K-562 whole cell lysate: sc-2203.

DATA



Western blot analysis of Rb phosphorylation in non-transfected: sc-117752 (**A**,**D**), untreated human Rb transfected: sc-114014 (**B**,**E**) and lambda protein phosphatase (sc-200312A) treated human Rb transfected: sc-114014 (**C**,**F**) 293T whole cell lysates. Antibodies tested include p-Rb (Thr 356)-R: sc-16837-R (**A**,**B**,**C**) and Rb (M-153): sc-7905 (**D**,**E**,**F**).



p-Rb (Thr 356)-R: sc-16837-R. Immunoperoxidase staining of formalin fixed, paraffin-embedded human duodenum tissue showing nuclear and cytoplasmic staining of glandular cells.

SELECT PRODUCT CITATIONS

1. Hara, K.T., et al. 2005. Cyclin A2-CDK2 regulates embryonic gene activation in 1-cell mouse embryos. Dev. Biol. 286: 102-113.
2. Patlolla, J.M., et al. 2006. β-escin inhibits colonic aberrant crypt foci formation in rats and regulates the cell cycle growth by inducing $p21^{waf1/cip1}$ in colon cancer cells. Mol. Cancer Ther. 5: 1459-1466.

STORAGE

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