p-Rb (ser608) (51B7): sc-56174



The Power to Overtio

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. Bremner, R., et al. 1995. Direct transcriptional repression by pRB and its reversal by specific cyclins. Mol. Cell. Biol. 15: 3256-3265.
- Weinberg, R.A. 1995. The retinoblastoma protein and cell cycle control. Cell 81: 323-330.
- 3. Sherr, C.J. 1996. Cancer cell cycles. Science 274: 1672-1677.
- 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.
- Luo, R.X., et al. 1998. Rb interacts with histone deacetylase to repress transcription. Cell 92: 463-473.
- 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.
- 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; Rb1 (mouse) mapping to 14 D3.

SOURCE

p-Rb (51B7) is a mouse monoclonal antibody raised against a short amino acid sequence containing phosphorylated Immunogen not available of Rb of origin.

PRODUCT

Each vial contains 50 $\mu g \; lg G_1$ in 0.5 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

STORAGE

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

APPLICATIONS

p-Rb (ser608) (51B7) is recommended for detection of Ser 608 phosphorylated Rb of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 μ g per 100-500 μ g of total protein (1 ml of cell lysate)] and immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500); non cross-reactive with non-phosphorylated Rb or Rb phosphorylated at other sites.

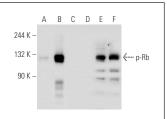
Suitable for use as control antibody for Rb siRNA (h): sc-29468, Rb siRNA (m): sc-29469, Rb shRNA Plasmid (h): sc-29468-SH, Rb shRNA Plasmid (m): sc-29469-SH, Rb shRNA (h) Lentiviral Particles: sc-29468-V and Rb shRNA (m) Lentiviral Particles: sc-29469-V.

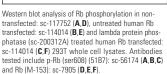
Molecular Weight (predicted) of p-Rb: 106 kDa.

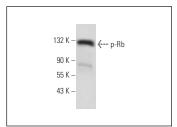
Molecular Weight (observed) of p-Rb: 107-140 kDa.

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

DATA







p-Rb (51B7): sc-56174. Western blot analysis of phosphorylated Rb expression in K-562 whole cell

SELECT PRODUCT CITATIONS

- Liu, Y., et al. 2010. Rosiglitazone inhibits cell proliferation by inducing G₁ cell cycle arrest and apoptosis in ADPKD cyst-lining epithelia cells. Basic Clin. Pharmacol. Toxicol. 106: 523-530.
- Rizzolio, F., et al. 2012. Retinoblastoma tumor-suppressor protein phosphorylation and inactivation depend on direct interaction with Pin1. Cell Death Differ. 19: 1152-1161.
- Joo, M.K., et al. 2015. CpG island promoter hypermethylation of Ras association domain family 1A gene contributes to gastric carcinogenesis. Mol. Med. Rep. 11: 3039-3046.
- Wang, Y., et al. 2016. Dysfunctional telomeres induce p53-dependent and independent apoptosis to compromise cellular proliferation and inhibit tumor formation. Aging Cell 15: 646-660.

RESEARCH USE

For research use only, not for use in diagnostic procedures