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Phospho-Rb(S788) Antibody Blocking peptide
Synthetic peptide
Catalog # BP3238a

Specification

Phospho-Rb(S788) Antibody Blocking peptide - Product Information

Primary Accession P06400

Phospho-Rb(S788) Antibody Blocking peptide - Additional Information

Gene ID 5925

Other Names
Retinoblastoma-associated protein, p105-Rb, pRb, Rb, pp110, RB1

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP3238a was selected from the region of human Phospho-Rb-S788. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

Phospho-Rb(S788) Antibody Blocking peptide - Protein Information

Name RB1

Function
Tumor suppressor that is a key regulator of the G1/S transition of the cell cycle (PubMed:10499802). The hypophosphorylated form binds transcription regulators of the E2F family, preventing transcription of E2F-responsive genes (PubMed:10499802). Both physically blocks E2Fs transactivating domain and recruits chromatin- modifying enzymes that actively repress transcription (PubMed:10499802). Cyclin and CDK-dependent phosphorylation of RB1 induces its dissociation from E2Fs, thereby activating transcription of E2F responsive genes and triggering entry into S phase (PubMed:10499802). RB1 also promotes the G0-G1 transition upon phosphorylation and activation by CDK3/cyclin-C (PubMed:15084261). Directly

involved in heterochromatin formation by maintaining overall chromatin structure and, in particular, that of constitutive heterochromatin by stabilizing histone methylation. Recruits and targets histone methyltransferases SUV39H1, KMT5B and KMT5C, leading to epigenetic transcriptional repression. Controls histone H4 'Lys-20' trimethylation. Inhibits the intrinsic kinase activity of TAF1. Mediates transcriptional repression by SMARCA4/BRG1 by recruiting a histone deacetylase (HDAC) complex to the c-FOS promoter. In resting neurons, transcription of the c-FOS promoter is inhibited by BRG1- dependent recruitment of a phospho-RB1-HDAC1 repressor complex. Upon calcium influx, RB1 is dephosphorylated by calcineurin, which leads to release of the repressor complex (By similarity).

Cellular Location
Nucleus. Note=During keratinocyte differentiation, acetylation by KAT2B/PCAF is required for nuclear localization.

Tissue Location
Expressed in the retina. Expressed in foreskin keratinocytes (at protein level) (PubMed:20940255)

Phospho-Rb(S788) Antibody Blocking peptide - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Blocking Peptides

Phospho-Rb(S788) Antibody Blocking peptide - Images

Phospho-Rb(S788) Antibody Blocking peptide - Background

RB1 likely acts as a regulator of other genes. It forms a complex with adenovirus E1A and with SV40 large T antigen, acts as a tumor suppressor, and may bind and modulate functionally certain cellular proteins with which T and E1A compete for pocket binding. RB1 is a potent inhibitor of E2F-mediated trans-activation, and also recruits and targets histone methyltransferase SUV39H1 leading to epigenetic transcriptional repression. This protein inhibits the intrinsic kinase activity of TAF1. Defects in RB1 are the cause of childhood cancer retinoblastoma (RB), a congenital malignant tumor that arises from the nuclear layers of the retina. Defects in RB1 are also a cause of bladder cancer and osteogenic sarcoma.

Phospho-Rb(S788) Antibody Blocking peptide - References

Wagner, S., et al., Biochem. Pharmacol. 69(7):1059-1067 (2005).Roesch, A., et al., Mod. Pathol. 18(4):565-572 (2005).Lieman, J.H., et al., J. Biol. Chem. 280(11):10484-10490 (2005).Budde, A., et al., Oncogene 24(10):1802-1808 (2005).Zapata, E., et al., FEBS J. 272(6):1343-1353 (2005).