

p27Kip1 (Mitotic Inhibitor/Suppressor Protein) Antibody - With BSA and Azide
Mouse Monoclonal Antibody [Clone KIP1/769]
Catalog # AH11007

Specification

p27Kip1 (Mitotic Inhibitor/Suppressor Protein) Antibody - With BSA and Azide - Product Information

Application	,1,2,3,4,
Primary Accession	P46527
Other Accession	1027 , 238990
Reactivity	Human, Mouse, Rat, Monkey
Host	Mouse
Clonality	Monoclonal
Isotype	Mouse / IgG1, kappa
Calculated MW	25-26kDa KDa

p27Kip1 (Mitotic Inhibitor/Suppressor Protein) Antibody - With BSA and Azide - Additional Information

Gene ID 1027

Other Names

Cyclin-dependent kinase inhibitor 1B, Cyclin-dependent kinase inhibitor p27, p27Kip1, CDKN1B, KIP1

Storage

Store at 2 to 8°C. Antibody is stable for 24 months.

Precautions

p27Kip1 (Mitotic Inhibitor/Suppressor Protein) Antibody - With BSA and Azide is for research use only and not for use in diagnostic or therapeutic procedures.

p27Kip1 (Mitotic Inhibitor/Suppressor Protein) Antibody - With BSA and Azide - Protein Information

Name CDKN1B {ECO:0000303|PubMed:20824794}

Function

Important regulator of cell cycle progression. Inhibits the kinase activity of CDK2 bound to cyclin A, but has little inhibitory activity on CDK2 bound to SPDYA (PubMed:28666995). Involved in G1 arrest. Potent inhibitor of cyclin E- and cyclin A-CDK2 complexes. Forms a complex with cyclin type D-CDK4 complexes and is involved in the assembly, stability, and modulation of CCND1-CDK4 complex activation. Acts either as an inhibitor or an activator of cyclin type D-CDK4 complexes depending on its phosphorylation state and/or stoichiometry.

Cellular Location

Nucleus. Cytoplasm. Endosome. Note=Nuclear and cytoplasmic in quiescent cells. AKT- or

RSK-mediated phosphorylation on Thr-198, binds 14-3-3, translocates to the cytoplasm and promotes cell cycle progression. Mitogen-activated UHMK1 phosphorylation on Ser-10 also results in translocation to the cytoplasm and cell cycle progression. Phosphorylation on Ser-10 facilitates nuclear export. Translocates to the nucleus on phosphorylation of Tyr-88 and Tyr-89. Colocalizes at the endosome with SNX6; this leads to lysosomal degradation (By similarity)

Tissue Location

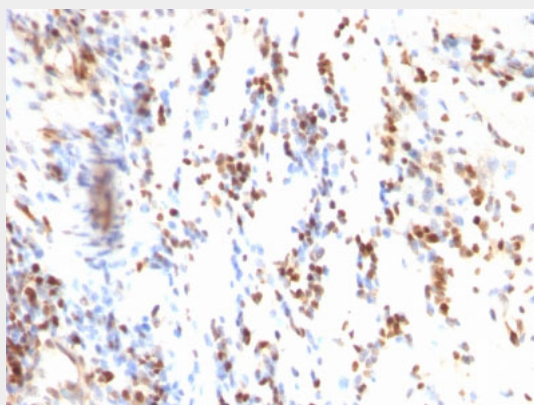
Expressed in kidney (at protein level) (PubMed:15509543). Expressed in all tissues tested (PubMed:8033212) Highest levels in skeletal muscle, lowest in liver and kidney (PubMed:8033212).

p27Kip1 (Mitotic Inhibitor/Suppressor Protein) Antibody - With BSA and Azide - Protocols

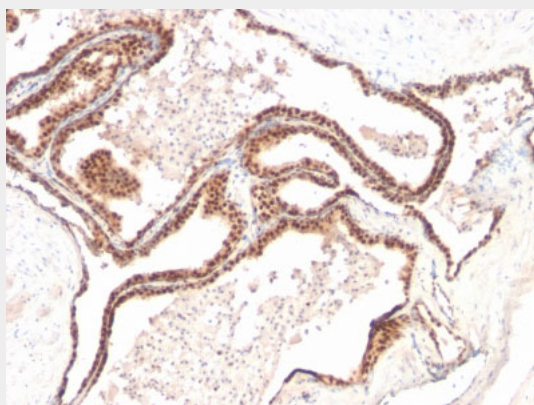
Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

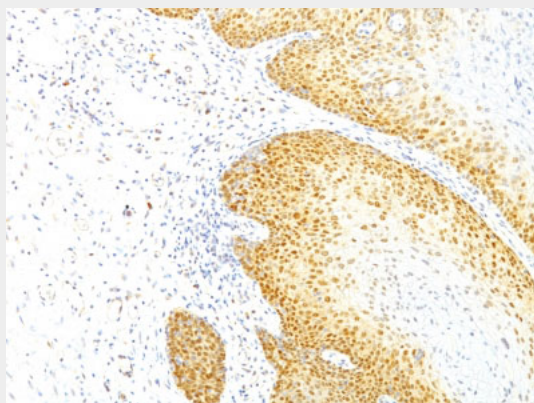
p27Kip1 (Mitotic Inhibitor/Suppressor Protein) Antibody - With BSA and Azide - Images



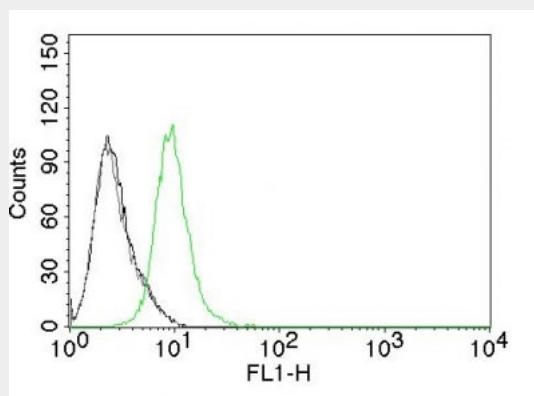
Formalin-fixed, paraffin-embedded human Colon Carcinoma stained with p27 Monoclonal Antibody (KIP1/769)



Formalin-fixed, paraffin-embedded human Prostate cancer stained with p27 Monoclonal Antibody (KIP1/769)



Formalin-fixed, paraffin-embedded human Cervical cancer stained with p27 Monoclonal Antibody (KIP1/769)



Flow Cytometry of human p27 on Jurkat Cells. Black: Cells alone; Grey: Isotype Control; Green: AF488-labeled p27 Monoclonal Antibody (KIP1/769).



Formalin-fixed, paraffin-embedded Rat Colon stained with p27 Monoclonal Antibody (KIP1/769)

p27Kip1 (Mitotic Inhibitor/Suppressor Protein) Antibody - With BSA and Azide - Background

This MAb recognizes a 27kDa protein, identified as the p27Kip1, a cell cycle regulatory mitotic inhibitor. It is highly specific and shows no cross-reaction with other related mitotic inhibitors. p27Kip1 functions as a negative regulator of G1 progression and has been proposed to function as a possible mediator of TGF- induced G1 arrest. p27Kip1 is a candidate tumor suppressor gene. This MAb is excellent for staining of formalin-fixed tissues.

**p27Kip1 (Mitotic Inhibitor/Suppressor Protein) Antibody - With BSA and Azide -
References**

Fredersdorf S et. al. Proc Natl Acad Sci 1997;94:6380-5. |