

# p27Kip1 (Mitotic Inhibitor/Suppressor Protein) Antibody - With BSA and Azide Mouse Monoclonal Antibody [Clone DCS-72.F6] **Catalog # AH11010**

### **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

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: <a

href="http://www.uniprot.org/citations/28666995" target=" blank">28666995</a>). 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 stoichometry.

#### **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**

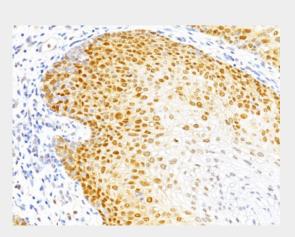
Expressed in kidney (at protein level) (PubMed:15509543). Expressed in all tissues tested (PubMed:8033212) Highest levels in skeletal muscle, lowest in liver and kidney (PubMed:8033212).

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

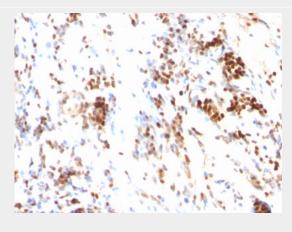
Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- <u>Immunohistochemistry</u>
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

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

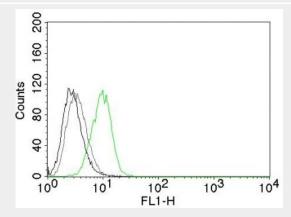


Formalin-fixed, paraffin-embedded human Cervical Cancer stained with p27 Monoclonal Antibody (DCS-72.F6)





Formalin-fixed, paraffin-embedded human Colon Carcinoma stained with p27 Monoclonal Antibody (DCS-72.F6)



Flow Cytometry of human p27 on HeLa Cells. Black: Cells alone; Grey: Isotype Control; Green: AF488-labeled p27 Monoclonal Antibody (DCS-72.F6).

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

Recognizes a 27kDa protein, identified as the p27Kip1, a cell cycle regulatory mitotic inhibitor. Its epitope spans between aa 83-204 of p27. 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 co-precipitates cdk4 in complex p27Kip1 and 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