

**p27Kip1 (Mitotic Inhibitor/Suppressor Protein) Antibody - With BSA and Azide**  
**Mouse Monoclonal Antibody [Clone SPM348 ]**  
**Catalog # AH10378**

**Specification**

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

Application	WB, IHC-P, IF, FC
Primary Accession	<a href="#">P46527</a>
Other Accession	<a href="#">1027</a> , <a href="#">238990</a>
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

**Application Note**

WB~~1:1000  
IHC-P~~N/A  
IF~~1:50~200  
FC~~1:10~50

**Format**

200ug/ml of Ab purified from Bioreactor Concentrate by Protein A/G. Prepared in 10mM PBS with 0.05% BSA & 0.05% azide. Also available WITHOUT BSA & azide at 1.0mg/ml.

**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:<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 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**

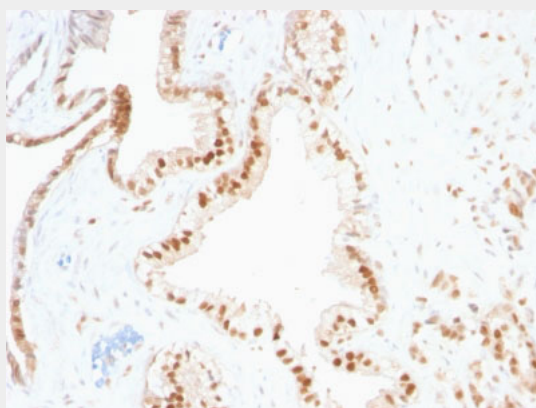
Expressed in kidney (at protein level) (PubMed:15509543). Expressed in all tissues tested (PubMed:8033212) Highest levels in skeletal muscle, lowest in liver and kidney (PubMed:8033212).

### **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 Prostate Carcinoma stained with p27 Monoclonal Antibody (SPM348)

### **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