

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 Primary Accession Other Accession Reactivity Host Clonality Isotype Calculated MW WB, IHC, IF, FC <u>P46527</u> <u>1027</u>, <u>238990</u> Human, Mouse, Rat, Monkey Mouse Monoclonal Mouse / IgG1, kappa 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<br \>IHC~~1:100~500<br \>IF~~1:50~200<br \>FC~~1:10~50

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 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

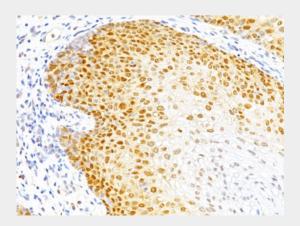
Expressed in kidney (at protein level) (PubMed:15509543). Expressed in all tissues tested (PubMed:8033212) Highest levels in skeletal muscle, lowest in liver and kidney (PubMed:8033212).

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

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

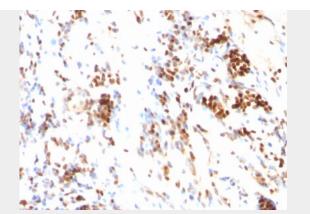
- <u>Western Blot</u>
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- <u>Cell Culture</u>

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

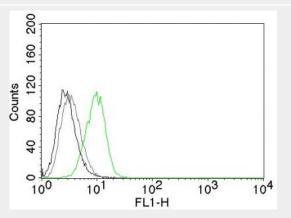


Formalin-fixed, paraffin-embedded human Cervical Cancer stained with p27 Monoclonal Antibody (DCS-72.F6)





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