SunLong Biotech Co.,LTD
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[Primary Antibody]

Rabbit Anti-CDKN2A/p14ARF antibody

Catalog Number: SL22495R

FOR RESEARCH USE ONLY

[DATASHEET]

Host:	Rabbit
Isotype:	IgG
Clonality:	Polyclonal
Target:	CDKN2A/p14ARF
Immunogen:	KLH conjugated synthetic peptide derived from human CDKN2A/P14arf: 1-100/132
Purification:	affinity purified by Protein A
Concentration:	1mg/ml
Storage:	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol. Shipped at 4℃. Store at -20 °C for one year. Avoid repeated freeze/thaw cycles.
Applications:	IHC-P=1:100-500 IHC-F=1:100-500 IF=1:100-500 （Paraffin sections need antigen repair） not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Reactivity:	Human
Predicted MW.:	14kDa
Subcellular Location:	The nucleus
Background:	The CDKN2A locus gives rise to 2 distinct transcripts from different promoters. The transcripts have been designated p16(INK4A) and p14(ARF). This chromosomal region undergoes a number of inversions, translocations, heterozygous deletions, and homozygous deletions in a variety of malignant cell lines including those from glioma, non-small cell lung cancer, leukemia, and melanoma. Deletion of the region containing CDKN2A is found in more than half of all melanoma cell lines. In spite of the structural and some functional differences, all the proteins encoded by the CDKN2A gene are involved in cell cycle G1 control. **Function:** Capable of inducing cell cycle arrest in G1 and G2 phases. Acts as a tumor suppressor. Binds to MDM2 and blocks its nucleocytoplasmic shuttling by sequestering it in the nucleolus.

Important Note: This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

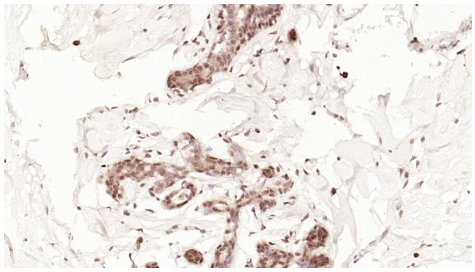
	This inhibits the oncogenic action of MDM2 by blocking MDM2-induced degradation of p53 and enhancing p53-dependent transactivation and apoptosis. Also induces G2 arrest and apoptosis in a p53-independent manner by preventing the activation of cyclin B1/CDC2 complexes. Binds to BCL6 and down-regulates BCL6-induced transcriptional repression. Binds to E2F1 and MYC and blocks their transcriptional activator activity but has no effect on MYC transcriptional repression. Binds to TOP1/TOPOI and stimulates its activity. This complex binds to rRNA gene promoters and may play a role in rRNA transcription and/or maturation. Interacts with NPM1/B23 and promotes its polyubiquitination and degradation, thus inhibiting rRNA processing. Interacts with COMMD1 and promotes its 'Lys63'-linked polyubiquitination. Interacts with UBE2I/UBC9 and enhances sumoylation of a number of its binding partners including MDM2 and E2F1. Binds to HUWE1 and represses its ubiquitin ligase activity. May play a role in controlling cell proliferation and apoptosis during mammary gland development. **Subunit:** Does not interact with cyclins, CDK1, CDK2, CDK4, CDK5 or CDK6. Binds to BCL6, E2F1, HUWE1, MDM2, MYC, NPM1/B23, TOP1/TOPOI and UBE2I/UBC9. Interacts with TBRG1 and COMMD1. Interacts with CDKN2AIP and E4F1. **Subcellular Location:** Nucleus, nucleolus. Nucleus, nucleoplasm. **Post-translational modifications:** Ubiquitinated in normal cells by TRIP12 via the ubiquitin fusion degradation (UFD) pathway, a process that mediates ubiquitination at the N-terminus, regardeless of the absence of lysine residues. Ubiquitination leads to its degradation. In cancer cells, however, TRIP12 is located in a different cell compartment, preventing ubiquitination and degradation. **Database links:** **Entrez Gene:** 1029 Human Omim: 600160 Human **SwissProt:** P42771 Human **SwissProt:** Q8N726 Human **Unigene:** 512599 Human Loss of cell cycle control is an important cause of carcinogenesis. p16 is the first tumor suppressor gene discovered in recent years that directly participates in cell cycle regulation, and its expression product is p16 protein. The p16 gene is an important tumor suppressor gene that exerts a negative feedback effect in normal cells. When the p16 gene is mutated or lost, cell proliferation loses control, causing cells to proliferate indefinitely.

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	The main function of p16 is to prevent cells from progressing from G1 phase to S phase by inhibiting CDK4, thereby restricting cell proliferation. It is used in research on various malignant tumours such as lung cancer, malignant melanoma, and breast cancer. Current research shows that the cyclin-dependent kinase inhibitor p16INK4a protein serves as a new marker in cervical intraepithelial neoplasia (CIN). Overexpression of p16INK4a is closely related to the activity of the HPV E7 region (viral early protein, i.e., the viral oncogene coding region). The p16/CDKN2 gene is a recently discovered tumor suppressor gene, and studies have shown that this gene exhibits deletion, mutation, or rearrangement in many tumors.

[VALIDATION IMAGES]



Paraformaldehyde-fixed, paraffin embedded (human breast); Antigen retrieval by boiling in sodium citrate buffer (pH6.0) for 15min; Block endogenous peroxidase by 3% hydrogen peroxide for 20 minutes; Blocking buffer (normal goat serum) at 37°C for 30min; Antibody incubation with (CDKN2 AP14arf) Polyclonal Antibody, Unconjugated (SL22495R) at 1:400 overnight at 4°C, followed by operating according to SP Kit(Rabbit) (sp-0023) instructionsand DAB staining.

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