

## CDKN2A

**Reactivity:** Human

**Tested applications:** WB IHC IF

**Recommended Dilution:** WB 1:500 - 1:2000 IHC 1:50 - 1:200 IF 1:50 - 1:200

**Calculated MW:** 11kDa

**Observed MW:** Refer to Figures

**Immunogen:**

A synthetic peptide of human CDKN2A

**Storage Buffer:**

Store at -20. Avoid freeze / thaw cycles. Buffer: PBS with 0.02% sodium azide, 50% glycerol, pH7.3.

**Concentration:**

b

**Synonym:**

CDKN2A;ARF;CDK4I;CDKN2;CMM2;INK4;INK4a;MLM;MTS1;TP16;p14;p14ARF;p16;p16INK4;p16INK4a;p19;ARF;CDKN2A

**Catalog #:** A0262

**Antibody Type:**

Polyclonal Antibody

**Species:** Rabbit

**Gene ID:** 1029

**Isotype:** IgG

**Swiss Prot:** P42771

**Purity:** Affinity purification

For research use only.

**Background:**

The division cycle of eukaryotic cells is regulated by a family of protein kinases known as the cyclin-dependent kinases (CDKs). The sequential activation of individual members of this family and their consequent phosphorylation of critical substrates promotes orderly progression through the cell cycle. It has been reported that CDKN2A binds to CDK4 and inhibits the catalytic activity of the CDK4/cyclin D enzymes. CDKN2A seems to act in a regulatory feedback circuit with CDK4, D-type cyclins and retinoblastoma protein (1). The INK4 (inhibitor of cyclin-dependent kinase 4) family consists of four tumor-suppressor proteins: p15(INK4B), CDKN2A(INK4A), p18(INK4C), and p19(INK4D). While their sequences and structures are highly homologous, they show appreciable differences in conformational flexibility, stability, and aggregation tendency (2). Cell cycle arrest at the G1 checkpoint allows completion of critical macromolecular events prior to S phase.

Regulators of the G1 checkpoint include an inhibitor of cyclin-dependent kinase, CDKN2A(INK4); two tumor-suppressor proteins, p53 and RB and cyclin D1. CDKN2A(INK4) is a tumor-suppressor protein and that genetic and epigenetic abnormalities in genes controlling the G1 checkpoint can lead to both escape from senescence and cancer formation (3).

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