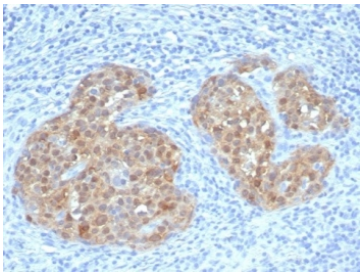
CDKN2A Antibody Mouse Monoclonal / p16INK4a [clone rCDKN2A/7659] (V5276)

Catalog No.	Formulation	Size
V5276-100UG	0.2 mg/ml in 1X PBS with 0.1 mg/ml BSA (US sourced), 0.05% sodium azide	100 ug
V5276-20UG	0.2 mg/ml in 1X PBS with 0.1 mg/ml BSA (US sourced), 0.05% sodium azide	20 ug
V5276SAF-100UG	1 mg/ml in 1X PBS; BSA free, sodium azide free	100 ug

Recombinant | MOUSE MONOCLONAL

Bulk quote request

Availability	1-3 business days
Species Reactivity	Human
Format	Purified
Host	Mouse
Clonality	Recombinant Mouse Monoclonal
Isotype	Mouse IgG1, kappa
Clone Name	rCDKN2A/7659
Purity	Protein A/G affinity
UniProt	P42771
Localization	Nucleus, Cytoplasm
Applications	Immunohistochemistry (FFPE): 1-2ug/ml for 30 min at RT
Limitations	This CDKN2A antibody is available for research use only.



Immunohistochemistry of CDKN2A antibody in human cervical carcinoma tissue. The mouse monoclonal clone rCDKN2A/7659 demonstrates strong nuclear and cytoplasmic HRP-DAB brown staining in malignant epithelial cell clusters, consistent with overexpression of p16INK4a in RB pathway dysregulation. Tumor cell nests are clearly highlighted against surrounding stromal and inflammatory cells, which show minimal background staining. Heat-induced epitope retrieval was performed by boiling tissue sections in pH 9 10mM Tris with 1mM EDTA for 20 minutes followed by cooling prior to antibody incubation.

Description

CDKN2A antibody recognizes Cyclin-dependent kinase inhibitor 2A, the tumor suppressor protein widely known as

p16INK4a, encoded by the CDKN2A gene on chromosome 9p21.3. CDKN2A Antibody Mouse Monoclonal is developed to detect this key regulator of cell cycle progression in research applications involving normal and neoplastic tissues. The p16INK4a protein localizes predominantly to the nucleus, with additional cytoplasmic distribution, where it binds CDK4 and CDK6 to inhibit phosphorylation of the retinoblastoma protein and block the transition from G1 to S phase.

p16INK4a antibody, also referred to as CDKN2A antibody and INK4a antibody in the literature, targets a central component of the RB pathway. The CDKN2A locus encodes multiple distinct tumor suppressor proteins through alternative reading frames, including p14ARF, but p16INK4a specifically inhibits cyclin D-CDK4 and cyclin D-CDK6 complexes. Loss of CDKN2A function through deletion, mutation, or promoter methylation is one of the most frequent molecular events in melanoma, pancreatic carcinoma, glioma, bladder carcinoma, and other malignancies.

In normal tissues, p16INK4a expression is typically low in proliferating cells but increases during cellular senescence or in response to oncogenic stress. Marked overexpression is commonly observed in high-risk human papillomavirus-associated lesions, including cervical intraepithelial neoplasia and carcinoma, where viral inactivation of RB results in compensatory upregulation of p16INK4a. As a result, CDKN2A antibody is widely used in studies investigating viral oncogenesis, cell cycle dysregulation, and tumor suppressor pathway alterations.

Structurally, p16INK4a contains ankyrin repeat domains that mediate its interaction with CDK4 and CDK6. By maintaining RB in a hypophosphorylated state, p16INK4a enforces cell cycle arrest and prevents inappropriate S phase entry. Disruption of this regulatory pathway contributes directly to uncontrolled cellular proliferation and tumor development.

The mouse monoclonal clone rCDKN2A/7659 provides specific detection of p16INK4a in formalin-fixed tissues and cell-based systems. Visualization of nuclear and cytoplasmic staining patterns supports evaluation of CDKN2A expression status and investigation of RB pathway integrity in research applications from NSJ Bioreagents.

Application Notes

Optimal dilution of the CDKN2A antibody mouse monoclonal should be determined by the researcher.

Immunogen

Purified recombinant prokaryotic full-length human protein was used as the immunogen for the CDKN2A antibody mouse monoclonal.

Storage

Aliquot the CDKN2A antibody and store frozen at -20oC or colder. Avoid repeated freeze-thaw cycles.

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