CDKN2A homozygous deletion

- MTA-Cooperative PRMT5 Inhibitors are Efficacious in MTAP-Deleted Malignant Peripheral Nerve Sheath Tumor Models
- Real-life implementation of molecular criteria for diagnosing gliomas according to 2021 WHO Classification: a national survey from the Italian Association of Neuro-Oncology and Society of Neurosurgery
- Recurrent parietal lobe supratentorial ependymoma, ZFTA fusion-positive, CNS WHO grade 3, with new dural and calvarial reactive changes in a child: illustrative case
- Extra-Gonadal Urinary Tract Neoplasms with Yolk Sac Differentiation: Clinical, Histologic, and Molecular Evidence of Derivation from Underlying Urothelial Carcinoma
- Pleomorphic xanthoastrocytoma with multiple recurrences and continuous malignant progression to bone metastasis: a case report
- Diagnostic Utility of Intratumoral Susceptibility Signals in Adult Diffuse Gliomas: Tumor Grade Prediction and Correlation with Molecular Markers Within the WHO CNS5 (2021) Classification
- Assessment of CDKN2A homozygous and heterozygous deletions in gliomas across multiple detection platforms
- Unlocking new horizons: advances in treating IDH-mutant, 1p/19q-codeleted oligodendrogliomas

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CDKN2A homozygous deletion characterizes diffuse malignant IDH-mutant gliomas with worst outcome.

Microvascular proliferation stratifies IDH-mutant gliomas lacking CDKN2A homozygous deletion.

CDKN2A, also known as cyclin-dependent kinase inhibitor 2A, is a gene which in humans is located at chromosome 9, band p21.3.

It is ubiquitously expressed in many tissues and cell types.

The gene codes for two proteins, including the INK4 family member p16 (or p16INK4a) and p14arf.

Both act as tumor suppressors by regulating the cell cycle. p16 inhibits cyclin dependent kinases 4 and 6 (CDK4 and CDK6) and thereby activates the retinoblastoma (Rb) family of proteins, which block traversal from G1 to S-phase. p14ARF (known as p19ARF in the mouse) activates the p53 tumor suppressor. Somatic mutations of CDKN2A are common in the majority of human cancers, with estimates that CDKN2A is the second most commonly inactivated gene in cancer after p53. Germline mutations of CDKN2A are associated with familial melanoma, glioblastoma and pancreatic cancer.

The CDKN2A gene also contains one of 27 SNPs associated with increased risk of coronary artery disease.

MGMT and CDKN2A status subdivided a cohort into three race-specific groups with different

prognoses. This findings indicate that bevacizumab (BEV) approval in Japan led to overall survival (OS) improvement exclusively for patients with concurrent unmethylated MGMT promoter status and CDKN2A homozygous deletion [1)]

Meningiomas are heterogeneous tumors as depicted by the current 15 distinct variants defined by histology in the 2021 WHO classification, which also incorporated the first molecular criteria for meningioma grading: homozygous loss of CDKN2A/B and TERT promoter mutation as criteria for a WHO grade 3 meningioma.

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Funakoshi Y, Hata N, Takigawa K, Arita H, Kuga D, Hatae R, Sangatsuda Y, Fujioka Y, Sako A, Umehara T, Yoshitake T, Togao O, Hiwatashi A, Yoshimoto K, Iwaki T, Mizoguchi M. Clinical significance of CDKN2A homozygous deletion in combination with methylated MGMT status for IDH-wildtype glioblastoma. Cancer Med. 2021 Apr 10. doi: 10.1002/cam4.3860. Epub ahead of print. PMID: 33838014.