ADGRB1 gene, which encodes Brain-specific angiogenesis inhibitor 1 (BAI1), is epigenetically silenced in medulloblastomas (MBs) through a methyl-CpG binding protein MBD2-dependent mechanism. Knockout of Adgrb1 in mice augments proliferation of cerebellar granule neuron precursors, and leads to accelerated tumor growth in the Ptch1+/- transgenic MB mouse model. BAI1 prevents Mdm2-mediated p53 polyubiquitination, and its loss substantially reduces p53 levels. Reactivation of BAI1/p53 signaling axis by a brain-permeable MBD2 pathway inhibitor suppresses MB growth in vivo. Altogether, our data define BAI1's physiological role in tumorigenesis and directly couple an ADGR to cancer formation [1].

1)
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