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Epigenetic events in medulloblastoma development.

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Abstract

Over the last decade, the analysis of genetic defects in primary tumors has been central to the identification of molecular events and biological pathways involved in the pathogenesis of medulloblastoma, the most common malignant brain tumor of childhood. Despite this, understanding of the molecular basis of the majority of cases remains poor. In recent years, the emerging field of epigenetics, which describes heritable alterations in gene expression that occur in the absence of DNA sequence changes, has forced a revision of the understanding of the mechanisms of gene disruption in cancer. Accumulating evidence indicates a significant involvement for epigenetic events in medulloblastoma development. Recent studies have identified a series of candidate tumor suppressor genes (for example, RASSF1A, CASP8, and HIC1) that are each specifically epigenetically inactivated in a large proportion (> 30%) of medulloblastomas by promoter hypermethylation, leading to the silencing of their gene expression. These findings shed new light on medulloblastoma and offer great potential for an improved understanding of its molecular pathology. The authors review the current understanding of epigenetic events in cancer and their contribution to medulloblastoma development. Their nature, origins, and functional role(s) in tumorigenesis are considered, and the authors assess the potential utility of these events as a basis for novel diagnostic and therapeutic approaches.

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