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Journal of Cell Science & Therapy

Opinion Article

Chromosome 17 Alterations and their Therapeutic Implications in Medulloblastoma

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DESCRIPTION

The cerebellar embryonal tumour Medulloblastoma (MB) is the most prevalent intracranial embryonal tumour. It starts in the posterior fossa, where there are significant subconscious motor nuclei for balance, posture, speaking, swallowing, and other critical activities.

It is the second most common tumour in children, accounting for 20% of all juvenile Central Nervous System (CNS) tumour, with 70% of cases occurring in children under the age of 16. The peak age of occurrence is seven years old; however there have also been reports of prenatal and neonatal occurrences. Seventy-five percent of this tumour are found in the vermis and have distinct neuroimaging characteristics that allow them to be identified. Even in the case of an intraoperative frozen biopsy or partial resection, the differential diagnosis can be relatively simple with adequate clinical and radiological information, despite their intratumoural heterogeneity, variety of histological subtypes, and irregularity and variety of immunohistochemically results for different proteins.

Chromosome 17 is one of the 23 pairs of human chromosomes; the abnormalities and functions associated with the expression of the genes on this chromosome have been researched in relation to the nervous system, specifically cell and tissue differentiation and maturation.

Furthermore, multiple publications and databases that align miRNA sequences and estimate mRNA targets imply that a large number of miRNAs encoded on chromosome 17 have regulatory action at various phases of neural maturation. Several investigations have been conducted to determine the significance of chromosome 17 abnormalities in MB. These investigations found that allelic loss of chromosome 17 areas is associated with a poor outcome when compared to patients with MB who do not have these mutations. The existence of a dicentric chromosome i(17q), two normal copies of chromosome 17, loss of telomere in 17p, and deletion in 17p11.2 were all discovered in the investigation of chromosome 17 changes in four MB tumour lines and in an induction model of tumour implant.

Other research has linked the loss of 17p and the increase of 1q to a worse chance of survival. Gaining 17q without losing 17p was linked to a better prognosis. According to the comprehensive review of all data, the loss of 17p is a sign of bad prognosis in patients with MB, whereas the gain of 17q may be a new marker of good prognosis.

Based on these, it is clear that good and poor prognosis groups cannot be accurately differentiated based on chromosome 17 alterations, but what is clear is the presence of genes important for the development of CNS, and that, despite the great clinical utility of classifying the different subgroups of MB, these markers do not appear to distinguish in greater detail the patients within these subgroups in terms of prognosis, strongly justifying the use of genetic markers. As a result, some miRNAs are recommended as novel markers; in this example, molecular markers in MB that can be used to sub classify and differentiate between groups with excellent and poor prognoses.

MiRNA and MB studies have revealed that none of the miRNAs investigated are encoded on chromosome 17. Only a few of the miRNAs' target proteins have been validated, including Epidermal Growth Factor Receptor (EGFR), B-Cell Lymphoma 2 (Bcl-2), and Cyclin-Dependent Kinase 6 (CDK6) to solute carrier family 16, member A11 (SLC16A11). When the levels of certain miRNAs are elevated or lowered, it has been linked to both poor and better prognoses. Gain or loss of function can be induced by anyone.

CONCLUSION

The study concludes that if a miRNA encoded on chromosome 17 in the MB is overexpressed relative to the cerebellum, the strategy will be to induce its degradation and control the loss of function such as differentiation, apoptosis, and cell adhesion by transfecting with the antagonistic or antagomir sequence of the miRNA. If a miRNA is under expressed and a gain of function occurs in processes such as proliferation, migration, or metastasis, reintroducing the mature sequence to recover control of the function by negatively regulating it is an important method.

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Received: 15-Jul-2022, Manuscript No. JCEST-22-16399; **Editor assigned:** 20-Jul-2022, PreQC No. JCEST-22-16399 (PQ); **Reviewed:** 03-Aug-2022, QC No. JCEST-22-16399; **Revised:** 10-Aug-2022, Manuscript No. JCEST-22-16399 (R); **Published:** 17-Aug-2022, DOI: 10.35248/2157-7013.22.S14.390.

Citation: Leo T (2022) Chromosome 17 Alterations and their Therapeutic Implications in Medulloblastoma. J Cell Sci Therapy. S14:390.