Epigenetic alterations of brain tumor therapy.

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Introduction

Epigenetic alterations have been increasingly recognized as key players in the development and progression of brain tumors. In recent years, there has been a growing interest in developing therapies that target these epigenetic changes to improve patient outcomes. In this article, we will explore the role of epigenetic alterations in brain tumor therapy and the current state of epigenetic-based therapies. Epigenetic alterations are heritable changes in gene expression that do not involve changes in the DNA sequence. These changes can occur through DNA methylation, histone modifications, and non-coding RNA molecules such as microRNAs. Epigenetic alterations can lead to the silencing of tumor suppressor genes and the activation of oncogenes, contributing to tumorigenesis [1].

Brain tumors, particularly gliomas, have been shown to have extensive epigenetic alterations that contribute to tumor growth and resistance to therapy. DNA methylation is a common epigenetic alteration in brain tumors, with hypermethylation of promoter regions leading to the silencing of tumor suppressor genes. In addition, global hypomethylation has been associated with the activation of oncogenes and chromosomal instability. Histone modifications, including acetylation and methylation, also play a role in brain tumor development and progression. These modifications can alter the chromatin structure, leading to changes in gene expression. For example, histone deacetylase (HDAC) inhibitors have been shown to have anti-tumor effects in preclinical models of glioma by promoting the acetylation of histones and the activation of tumor suppressor genes [2].

MicroRNAs are small non-coding RNA molecules that regulate gene expression by binding to mRNA transcripts and inhibiting their translation. Dysregulation of microRNA expression has been observed in brain tumors and has been associated with tumor progression and treatment resistance. Given the importance of epigenetic alterations in brain tumor development and progression, there has been growing interest in developing therapies that target these changes. One approach is to use drugs that inhibit the enzymes responsible for DNA methylation or histone modifications. For example, DNA methyltransferase (DNMT) inhibitors such as decitabine and azacitidine have been shown to have anti-tumor effects in preclinical models of glioma by reactivating tumor suppressor genes that have been silenced by hypermethylation.

Histone deacetylase (HDAC) inhibitors, such as vorinostat and panobinostat, have also been investigated as potential therapies for brain tumors. These drugs promote the acetylation of histones, leading to the activation of tumor suppressor genes and the inhibition of oncogenes. Another approach is to use drugs that target specific microRNAs that are dysregulated in brain tumors. For example, miR-21 is upregulated in gliomas and has been shown to promote tumor growth and resistance to chemotherapy. Inhibitors of miR-21, such as antagomirs, have been shown to have anti-tumor effects in preclinical models of glioma. Despite the promise of epigenetic-based therapies, there are several challenges that need to be overcome. One challenge is the development of biomarkers that can accurately predict which patients are likely to respond to these therapies. Currently, there are no reliable biomarkers that can predict response to DNMT inhibitors, HDAC inhibitors, or microRNA inhibitors [3].

Another challenge is the potential for off-target effects and toxicity. Many epigenetic-based therapies target enzymes that are involved in normal cellular processes, raising concerns about potential toxicity. For example, DNMT inhibitors can cause myelosuppression, and HDAC inhibitors can cause gastrointestinal and cardiac toxicities. DNMT inhibitors have demonstrated the ability to reactivate tumor suppressor genes silenced by hypermethylation, while HDAC inhibitors promote the acetylation of histones, leading to the activation of tumor suppressor genes and the inhibition of oncogenes. MicroRNA inhibitors target dysregulated microRNAs, such as miR-21, to suppress tumor growth and enhance treatment sensitivity [4].

However, there are still significant challenges to overcome before epigenetic-based therapies can be widely implemented in clinical practice. One major hurdle is the identification of reliable biomarkers that can accurately predict patient response to these therapies. Personalized medicine approaches that consider specific epigenetic profiles of individual tumors may help identify patients who are most likely to benefit from these treatments. Furthermore, the potential for off-target effects and toxicity is a concern. Epigenetic modifications are involved in various cellular processes beyond tumorigenesis, and inhibiting or modifying these processes can lead to adverse effects. It is crucial to carefully monitor and manage potential toxicities associated with epigenetic therapies to ensure patient safety [5].

Conclusion

Epigenetic alterations play a crucial role in brain tumor development and progression. Targeting these alterations through epigenetic-based therapies has shown promise in preclinical studies and early-phase clinical trials. However,

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further research is needed to overcome challenges related to biomarker identification, potential toxicities, and optimizing treatment combinations. With continued advancements in our understanding of epigenetic mechanisms and the development of innovative therapeutic approaches, epigenetic alterations may become an integral part of brain tumor therapy, leading to improved patient outcomes in the future.

References

1. Bettegowda C, Agrawal N, Jiao Y, et al. Exomic sequencing of four rare central nervous system tumor types. Oncotarget. 2013;4(4):572.
2. Parsons DW, Jones S, Zhang X, et al. An integrated genomic analysis of human glioblastoma multiforme. Science. 2008;321(5897):1807-12.
3. Northcott PA, Nakahara Y, Wu X, et al. Multiple recurrent genetic events converge on control of histone lysine methylation in medulloblastoma. Nat Genet. 2009;41(4):465-72.
4. Jones DT, Jäger N, Kool M, et al. Dissecting the genomic complexity underlying medulloblastoma. Nature. 2012;488(7409):100-5.
5. Torchia J, Picard D, Lafay-Cousin L, et al. Molecular subgroups of atypical teratoid rhabdoid tumours in children: An integrated genomic and clinicopathological analysis. Lancet Oncol. 2015;16(5):569-82.

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