Unraveling the link: miRNA dysregulation in traumatic brain injury and epilepsy.

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Introduction

Traumatic brain injury (TBI) and epilepsy are two distinct neurological conditions with separate etiologies, but recent research has uncovered a fascinating connection between the two. Both TBI and epilepsy involve complex molecular and cellular changes in the brain, and emerging evidence suggests that microRNA (miRNA) dysregulation plays a crucial role in the development and progression of these conditions. In this article, we explore the intriguing link between miRNA dysregulation, traumatic brain injury, and epilepsy, shedding light on the potential mechanisms underlying their pathophysiology and highlighting the potential for miRNAs as therapeutic targets. MicroRNAs are small non-coding RNA molecules that play a critical role in gene expression regulation. They act as post-transcriptional regulators, binding to target messenger RNA (mRNA) molecules and either inhibiting their translation or promoting their degradation. MiRNAs have been implicated in various biological processes, including neuronal development, synaptic plasticity, and neuroinflammation [1].

Neuroinflammation is a prominent feature in both TBI and epilepsy. It involves the activation of immune cells, release of pro-inflammatory molecules, and subsequent damage to neuronal tissue. MiRNAs have been implicated in modulating the neuroinflammatory response in both conditions. For example, miR-146a, upregulated after TBI, regulates immune response and inflammation by targeting key genes involved in these processes. Similarly, miR-155 has been found to be elevated in epilepsy and contributes to the activation of microglia and the release of pro-inflammatory cytokines. Both TBI and epilepsy involve alterations in synaptic plasticity, which is essential for normal brain function. MiRNAs play a critical role in regulating synaptic plasticity by modulating the expression of genes involved in synaptic function and neuronal excitability. For instance, miR-124, downregulated in both TBI and epilepsy, is known to promote synaptic maturation and regulate neuronal excitability. Conversely, miR-134, upregulated in epilepsy, inhibits the expression of genes associated with dendritic spine formation and synaptic plasticity [2].

miRNA dysregulation in traumatic brain injury

Following a TBI, the brain undergoes a cascade of events, including primary injury and secondary injury processes. Secondary injury, which encompasses a range of pathological mechanisms, such as neuroinflammation, oxidative stress, and excitotoxicity, can significantly contribute to long-term neurological deficits. Recent studies have shown that miRNA dysregulation is a key player in these secondary injury processes. Specific miRNAs, such as miR-21, miR-146a, and miR-223, are upregulated in response to TBI and contribute to the neuroinflammatory response, glial activation, and blood-brain barrier disruption. Conversely, other miRNAs, such as miR-29 and miR-124, show downregulation, leading to impaired neuronal survival and synaptic plasticity [3].

miRNA Dysregulation in Epilepsy

Epilepsy is a chronic neurological disorder characterized by recurrent seizures. It is widely acknowledged that miRNA dysregulation contributes to epileptogenesis, the process by which a normal brain develops a propensity for seizures. Several miRNAs have been identified as crucial players in epilepsy pathogenesis. For instance, miR-134 and miR-146a are upregulated in epileptic brains, leading to dendritic spine abnormalities and increased neuroinflammation, respectively. On the other hand, miR-132, miR-29, and miR-124 are downregulated, impacting synaptic plasticity, neuronal excitability, and neurogenesis [4].

Remarkably, several miRNAs appear to be dysregulated in both TBI and epilepsy, suggesting shared molecular pathways between these two conditions. For instance, miR-21, miR-146a, and miR-124, which are implicated in both TBI and epilepsy, play critical roles in neuroinflammation and synaptic plasticity. Dysregulation of these miRNAs may contribute to the common occurrence of epilepsy following TBI, known as post-traumatic epilepsy (PTE). Furthermore, TBI-induced alterations in miRNA expression patterns might contribute to the increased susceptibility to seizures and epileptogenesis observed in individuals with a history of TBI. Given the central role of miRNA dysregulation in the pathophysiology of TBI and epilepsy, targeting specific miRNAs has emerged as a promising therapeutic approach. Developing strategies to restore normal miRNA levels or manipulate their activity holds great potential for mitigating the detrimental effects of TBI and epilepsy. Researchers are actively exploring various approaches, including the use of antagomirs (antisense oligonucleotides) to inhibit specific miRNAs or the delivery of exogenous miRNAs to restore their levels [5].

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Conclusion

The discovery of miRNA dysregulation as a common underlying mechanism in both traumatic brain injury and epilepsy opens up new avenues for understanding and potentially treating these neurological conditions. Further research is needed to elucidate the intricate miRNA networks involved and their precise contributions to the development and progression of TBI and epilepsy. By harnessing the therapeutic potential of miRNAs, we may pave the way for innovative treatments that can improve outcomes and quality of life for individuals affected by these debilitating conditions.

References

1. You WD, Tang QL, Wang L, et al. Alteration of microRNA expression in cerebrospinal fluid of unconscious patients after traumatic brain injury and a bioinformatic analysis of related single nucleotide polymorphisms. Chin J Traumatol. 2016;19(1):11-5.
2. Stein DM, Kufera JA, Lindell A, et al. Association of CSF biomarkers and secondary insults following severe traumatic brain injury. Neurocrit care. 2011;14:200-7.
3. Li Y, Zhang L, Kallakuri S, et al. Correlation of mechanical impact responses and biomarker levels: a new model for biomarker evaluation in TBI. J Neurol Sci. 2015;359(1-2):280-6.
4. Pinchi E, Frati P, Aromatario M, et al. miR-1, miR-499 and miR-208 are sensitive markers to diagnose sudden death due to early acute myocardial infarction. J Cell Mol Med. 2019;23(9):6005-16.
5. Dai W, He J, Zheng L, et al. miR-148b-3p, miR-190b, and miR-429 regulate cell progression and act as potential biomarkers for breast cancer. J Breast cancer. 2019;22(2):219-36.

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