

MicroRNA Regulation: A Key Player in Neurodegenerative Processes and Brain Aging

Punam Chowdhury*

Department of Neurology & Neurosurgery, All India Institute of Medical Sciences, India

Abstract

MicroRNAs (miRNAs) have emerged as pivotal regulators of gene expression, orchestrating intricate networks that influence a wide array of cellular processes. In recent years, research has illuminated their significant role in the aging brain and the pathogenesis of neurodegenerative diseases. This article delves into the remarkable impact of miRNAs on neurodegenerative processes and brain aging, shedding light on their potential as therapeutic targets and diagnostic tools.

Introduction

Neurodegenerative diseases, including Alzheimer's, Parkinson's, and Huntington's disease, represent a growing public health concern as populations age worldwide. A fundamental hallmark of these diseases is the progressive loss of neurons and neuronal function, leading to cognitive and motor deficits. Recent research has unveiled the multifaceted involvement of miRNAs in these processes, presenting a new layer of complexity in our understanding of neurodegeneration and brain aging [1].

Multiple cellular and functional transformations take place in the brain during aging. Neural cells may respond to these changes by reprogramming metabolic circuits in order to adapt and maintain its functionality, or they may give in to neurodegenerative cascades that result in disorders such as Alzheimer's, cerebellar ataxias, and Parkinson's diseases. A number of mechanisms are employed to maintain the integrity of nerve cell networks and to facilitate responses to external and internal environmental stimuli and maintain neuron integrity and functional capability after damage [2, 3].

Although rodent models to study brain aging and neurodegenerative disorders have been developed, these models do not satisfactorily parallel the brain changes and behavioural features observed in humans. The close physiological, neurological, and genetic similarities between humans and higher primates offer the opportunity to study the aging process and associated abnormalities in monkeys. Aged nonhuman primates undergo age-associated structural and functional brain changes similar to those that occur in aged humans and, to some degree, in individuals with Alzheimer's disease. As in humans, declines in performance on cognitive and memory tasks begin at the monkey equivalent of late-middle life. The brains of old monkeys show degenerative changes in neurons, abnormal axons and neurites, and accumulations of amyloid plaques and lipofuscin around blood vessels and in the residential macrophages. Moreover, old nonhuman primates exhibit decline of specific neurotransmitter networks, most notably the forebrain cholinergic system that has been suggested to contribute to the memory deficit characteristic for older individuals.

miRNAs play an important role in the regulation of several cell processes, including cell proliferation, development, cancer formation, stress responses, and apoptosis. The rapid progression of miRNA research in these areas has revealed its prominent role in modulating gene expression. However, the role of miRNAs in senescence remains poorly understood. miRNA can affect pathways involved in ageing, and miRNA profiling has shown significant alterations in their expression level [4].

MicroRNAs: Molecular regulators of gene expression

MiRNAs are small, non-coding RNA molecules that play a critical role in post-transcriptional gene regulation. Through their interaction with target messenger RNAs (mRNAs), miRNAs can suppress gene expression by promoting mRNA degradation or inhibiting translation. This intricate control mechanism allows miRNAs to modulate the expression of numerous genes simultaneously, affecting diverse biological pathways [5].

MicroRNAs in neurodegenerative diseases

Mounting evidence suggests that dysregulation of miRNA expression is closely linked to the onset and progression of neurodegenerative diseases. Altered miRNA profiles have been observed in the brains of patients with Alzheimer's, Parkinson's, and other neurodegenerative disorders. These miRNAs often target genes involved in crucial cellular processes, including protein aggregation, synaptic plasticity, inflammation, and oxidative stress, all of which are implicated in disease pathogenesis [6].

MicroRNAs and brain aging

Aging is the primary risk factor for many neurodegenerative diseases. As the brain ages, miRNA expression patterns shift, contributing to the decline in neuronal function and plasticity. Dysregulated miRNAs can disrupt the delicate balance between neuroprotective and neurodegenerative mechanisms, accelerating the aging process and making the brain more susceptible to disease-associated insults [7,8].

Therapeutic potential and diagnostic applications

Harnessing the regulatory power of miRNAs holds great promise for the development of innovative therapeutic strategies

***Corresponding author:** Punam Chowdhury, Department of Neurology & Neurosurgery, All India Institute of Medical Sciences, India, E-mail: Chowdhurypunam165@gmail.com

Received: 03-July-2023, Manuscript No: nctj-23-109978, **Editor assigned:** 05-July-2023, PreQC No: nctj-23-109978 (PQ), **Reviewed:** 19-July-2023, QC No: nctj-23-109978, **Revised:** 25-July-2023, Manuscript No: nctj-23-109978 (R), **Published:** 31-July-2023, DOI: 10.4172/nctj.1000155

Citation: Chowdhury P (2023) MicroRNA Regulation: A Key Player in Neurodegenerative Processes and Brain Aging. Neurol Clin Therapeut J 7: 155.

Copyright: © 2023 Chowdhury P. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

for neurodegenerative diseases. Researchers are exploring various approaches, such as miRNA replacement therapy to restore normal gene expression patterns and miRNA-based gene editing to modify disease-associated targets. Additionally, the distinct miRNA profiles associated with specific neurodegenerative diseases offer potential diagnostic biomarkers, enabling early disease detection and personalized treatment plans [9].

Challenges and future directions

Despite the excitement surrounding miRNA research in neurodegeneration, challenges remain. Identifying the precise targets of specific miRNAs, deciphering the intricate miRNA-mRNA networks, and establishing efficient delivery methods for miRNA-based therapies are areas that require further investigation. Moreover, the potential off-target effects of manipulating miRNA pathways must be carefully considered to ensure the safety and efficacy of therapeutic interventions [10].

Conclusion

MicroRNAs have emerged as pivotal players in the complex landscape of neurodegenerative processes and brain aging. Their ability to modulate gene expression in a precise and coordinated manner highlights their potential as therapeutic targets and diagnostic tools for neurodegenerative diseases. As we uncover more about the roles of specific miRNAs in disease pathways, we move closer to unveiling innovative interventions that could transform the landscape of neurodegenerative disease treatment and prevention.

The discovery of miRNAs has revealed a new layer of regulation of gene expression, and studies in recent years have shown that miRNAs not only have a unique expression profile in the brain and peripheral nervous system but also play crucial roles in the regulation of both neuronal cell development and function. miRNA play an important role in the molecular control of brain development and subsequently in the aging process and associated neuron pathologies.

The field of miRNA and ncRNA research has developed quickly, and with the identification of brain-specific miRNAs in recent years, a new level of understanding of brain abnormalities associated with the aging has been acquired. However, more work remains to be done to fully

understand the miRNA mechanism of action in normal brain aging and neurodegenerative conditions, so that expression of the miRNAs can potentially be exploited as a new point of entry for therapy. With the growing number of miRNAs and ncRNAs, each carrying a long list of putative targets, the challenge is now to annotate their biological functions.

Acknowledgement

None

Conflict of Interest

None

References

1. Finkel RS, Chiriboga CA, Vajsar J, Day JW, Montes J, et al. (2016) Treatment of infantile-onset spinal muscular atrophy with nusinersen: a phase 2, open-label, dose-escalation study. *Lancet* 388: 3017-3026.
2. Gitler AD, Bevis BJ, Shorter J, Strathearn KE, Hamamichi S, et al. (2008) The Parkinson's disease protein alpha-synuclein disrupts cellular Rab homeostasis. *Proc Natl Acad Sci USA* 105: 145-150.
3. Cortez MA, McKerlie C, Snead OC (2001) A model of atypical absence seizures: EEG, pharmacology, and developmental characterization. *Neurology* 56: 341-349.
4. Pillai J, Sperling MR (2006) Interictal EEG and the diagnosis of epilepsy. *Epilepsia* 47: 14-22.
5. Khurana V, Chung C Y, Tardiff DF (2017) From yeast to patients: the audacity and vision of Susan Lindquist. *Cell Syst* 4: 147-148.
6. Hubel DH, Wiesel TN (1968) Receptive fields and functional architecture of monkey striate cortex.
7. Fukushima K (1980) Neocognitron: a self organizing neural network model for a mechanism of pattern recognition unaffected by shift in position. *Biol Cybern* 36: 193-202.
8. Marton RM, Paşca SP (2016) Neural differentiation in the third dimension: generating a human midbrain. *Cell Stem Cell* 19: 145-146.
9. Görg B, Schliess F, Häussinger D (2013) Osmotic and oxidative/nitrosative stress in ammonia toxicity and hepatic encephalopathy. *Arch Biochem Biophys* 536: 158-163.
10. Rahmlow MR, Kantarci O (2013) Fulminant demyelinating diseases. *Neurohospitalist* 3: 81-91.