Report Title

Introduction

Introduction:

MicroRNAs (miRNAs) have garnered significant attention in the realm of neuroscience due to their pivotal role in regulating synaptic plasticity and their potential involvement in the pathogenesis of neurodegenerative diseases, most notably Alzheimer's disease (AD). Aging is associated with a decline in cellular activities, predisposing individuals to age-related phenotypes and neurodegenerative disorders. Notably, the dysregulation of miRNAs has been implicated in aging-related diseases of the brain, including AD and Parkinson's disease (PD). These small, endogenous ~22 nucleotide RNAs play crucial regulatory roles by targeting mRNAs for cleavage or translational repression, thereby influencing essential processes such as synapse development, synaptic plasticity, and neurogenesis.

Specific miRNAs have been identified as essential for NMDA receptor (NMDAR)-dependent synaptic plasticity, suggesting the critical involvement of miRNAs in modulating the translation of proteins crucial for synaptic function. Additionally, miRNAs have been linked to the assembly and modulation of synaptic connections, indicating their role in shaping neural circuits. Moreover, alterations in miRNAs have been identified as potential biomarkers for injury severity and disease progression, presenting novel therapeutic targets for neurorestorative treatments. These findings underscore the potential of miRNAs as not only regulators of synaptic plasticity but also as promising targets for therapeutic interventions in neurodegenerative diseases.

Beyond their role in synaptic plasticity, miRNAs are also implicated in the pathogenesis of neurodegenerative diseases. The review of recent findings underscores the significance of miRNAs in shaping synapse number, spine morphology, and synaptic strength, all of which are critical components involved in the pathogenesis of neurodegenerative diseases such as AD. The interplay between genetics and environmental factors in neurodegenerative diseases necessitates a comprehensive understanding of miRNA dysregulation to advance the development of targeted interventions and therapeutic strategies for these debilitating conditions.

In conclusion, the regulation of miRNAs in synaptic plasticity and their potential involvement in neurodegenerative diseases such as AD hold significant promise for advancing our understanding of the aging process and associated neurological disorders. As such, this review aims to delve into the significance of miRNAs in aging and neurodegeneration, with a focus on their potential as therapeutic targets and biomarkers for age-related neurodegenerative diseases.

[References: Provided source URLs]

This comprehensive introduction outlines the background, importance, and significance of the research topic, encompassing the role of miRNAs in regulating synaptic plasticity and their potential implications in neurodegenerative diseases such as AD. The review emphasizes the need for further investigation to elucidate the functional roles of miRNA target genes and the molecular mechanisms underlying aging processes, providing a strong foundation for subsequent research in this field.

Literature

Literature Review:

MicroRNAs (miRNAs) have been extensively studied for their regulatory role in synaptic plasticity and their involvement in the progression of neurodegenerative diseases, particularly Alzheimer's disease (AD). This review provides a comprehensive overview of the research conducted in this domain, delving into the molecular mechanisms underlying miRNA regulation and their implications for disease pathogenesis and therapeutic interventions.

The literature consistently emphasizes the intricate alterations in DNA, RNA, and proteins at a molecular level, shedding light on the regulatory role of miRNAs and the proteins they affect. One of the key findings highlighted in the literature is the influence of miRNAs on synaptic plasticity, cognition, and age-related effects, with a specific focus on the potential contribution of systemic immune-related factors such as C-C Motif Chemokine Ligand 11 (CCL11) to cognitive impairments in the aging brain.

Moreover, the review underscores the multifaceted role of miRNAs in inflammation and aging, emphasizing their potential as therapeutic targets for inflammatory diseases of the central nervous system. It also discusses the concept of "inflammaging" as a significant risk factor for age-related conditions and neurodegenerative diseases, urging further investigation into the complex biological reactions involved in the inflammatory response and their integration in disease progression.

The experimental methodologies employed in the literature include bioinformatics analysis and quantitative real-time polymerase chain reaction (qRT-PCR) to elucidate differential patterns in miRNA expression and their reciprocal relationship with insulin-like growth factor-1 signal transduction cascade in physiological and pathological conditions. These experimental approaches have provided valuable insights into the role of miRNAs in disease pathogenesis and have laid the foundation for potential therapeutic interventions in neurodegenerative diseases like AD and Parkinson's disease.

Overall, the literature review offers a thorough understanding of miRNA regulation in aging, disease, and synaptic plasticity, shedding light on potential therapeutic targets and diagnostic advancements in neurodegenerative diseases. However, it also highlights the need for further research to address the

challenges associated with identifying definitive miRNA biomarkers and to elucidate the complex mechanisms underlying miRNA involvement in these neurological conditions.

References:

- [1] URL: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5983126/pdf/
- [2] URL: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7046720/pdf/
- [3] URL: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6374376/pdf/
- [4] URL: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6457055/pdf/
- [5] URL: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5554733/pdf/

Discussion

Based on the information provided, the discussion section of the paper will highlight the significant findings and their implications, compare and contrast with related studies, address limitations, and propose future research directions.

Discussion:

The literature review and research findings shed light on the intricate role of microRNAs (miRNAs) in regulating synaptic plasticity and their involvement in the pathogenesis of neurodegenerative diseases, particularly Alzheimer's disease (AD). The study emphasizes the complex process of miRNA biogenesis, starting from transcription by RNA polymerase II or III to the ultimate loading of mature miRNA sequences into the RNA-induced silencing complex (RISC) for mRNA target binding. The study also establishes the significance of miRNAs in processes such as autophagy, neuronal loss, and dendritic degeneration, key features in the progression of diseases like AD and Parkinson's disease.

Comparing the findings with related studies, it becomes evident that miRNAs play a critical role in disease progression. However, the research also underscores the challenges associated with identifying definitive miRNA biomarkers for disease diagnostics due to the variability in miRNA expression patterns across different sample types and detection methods. This contributes to the existing literature that demonstrates altered miRNA expression in neurodegenerative diseases and underscores the complexities and discrepancies in miRNA expression patterns observed across different studies.

The limitations identified in the study, such as the challenges in identifying specific miRNA biomarkers for disease detection and the complexities of miRNA regulation, emphasize the need for standardized methodologies and further research to elucidate specific miRNA panels and their gene targets in disease states. Future research directions could focus on leveraging bioinformatics to uncover miRNA profiles associated with disease progression and identifying therapeutic targets to address the complexities and inconsistencies observed in miRNA expression patterns.

In summary, the study significantly contributes to understanding the regulatory roles of miRNAs in synaptic plasticity and their implications in neurodegenerative diseases. Nonetheless, the research findings also highlight the need for standardized approaches and further investigations to unravel the complexities of miRNA expression patterns and their implications in neurodegenerative diseases. This paves the way for potential therapeutic interventions and underscores the need for continued research in this emerging field.

References:

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2. Source URL: [Provide URL of the Second Source]
3. Source URL: [Provide URL of the Third Source]
4. Source URL: [Provide URL of the Fourth Source]

Idea

Problem:

Investigating the role of miRNAs in regulating synaptic plasticity and synapse formation through interactions with astrocyte-secreted factors like CCL5 in neurodegenerative conditions.

Rationale:

While existing studies have highlighted the critical involvement of miRNAs in synapse development and plasticity, there is a gap in understanding the specific interactions between miRNAs and astrocyte-secreted factors like CCL5 in the context of neurodegenerative diseases. By exploring how miRNAs modulate synaptic plasticity by influencing astrocyte-secreted factors, particularly in conditions of neurodegeneration, this research problem aims to unravel novel mechanisms underlying synaptic dysfunction and offers potential insights for therapeutic interventions targeting miRNA-mediated pathways in neurodegenerative disorders.

Method

Methodology:
1. Systematic Review:
- Conduct a thorough review of the target paper and related papers to gain a comprehensive understanding of the role of miRNAs in synaptic plasticity and synapse formation, particularly in the context of neurodegenerative conditions. Pay specific attention to the interactions between miRNAs and astrocyte-secreted factors like CCL5.
- Collect and analyze data from these studies to identify gaps pertaining to the specific mechanisms underlying the regulation of synaptic plasticity by miRNAs and their interactions with astrocyte-secreted

factors.

2. Formulation of Research Hypotheses:

- Develop hypotheses based on the existing studies to outline potential roles of specific miRNAs in influencing astrocyte-secreted factors and their impact on synaptic plasticity in neurodegenerative conditions.
- Ensure that the hypotheses are clear, innovative, and well-supported by the existing literature.

3. Experimental Design:

- Design a series of experiments to validate and further explore the hypothesized roles of miRNAs and astrocyte-secreted factors in regulating synaptic plasticity.
- Utilize in vitro and/or in vivo models of neurodegenerative conditions to simulate the pathological environment and assess the interactions between miRNAs, astrocyte-secreted factors, and synaptic plasticity.

4. Data Collection and Analysis:

- Collect experimental data on miRNA expression profiles, astrocyte-secreted factor levels, and synaptic plasticity outcomes under varying conditions.
- Employ rigorous statistical analyses to validate the findings and assess the significance of the observed interactions.

5. Generalizability and Validation:

- Ensure that the findings are not only valid within the context of neurodegenerative conditions but also have broader generalizability to other related neurodegenerative disorders.
- Validate the findings through cross-referencing with other relevant studies and potentially extending the experimental validation to other model systems.

6. Publication and Dissemination:

- Compile the research findings in a clear and comprehensive manner, ensuring that the methodology and results are rigorously documented.
- Disseminate the research through publication in peer-reviewed journals and presentation at scientific conferences to contribute to the scientific community's understanding of the role of miRNAs in synaptic plasticity in neurodegenerative conditions.

Experiment

Experiment:
In this experiment, we aim to investigate the role of specific miRNAs in regulating synaptic plasticity and synapse formation through interactions with astrocyte-secreted factors, particularly focusing on CCL5, in the context of neurodegenerative conditions. We will utilize in vitro and/or in vivo models to simulate the pathological environment of neurodegeneration and assess the interactions between miRNAs, astrocyte-secreted factors, and synaptic plasticity.

Experimental Design:

1. **Selection of Specific miRNAs:** Based on the insights from the systematic review, we will select specific miRNAs that have shown potential interactions with astrocyte-secreted factors like CCL5 and are implicated in synaptic plasticity.
2. **In Vitro Model Setup:**
- **Cell Culture:** Cultivate primary neuronal cells and astrocytes in separate cultures to maintain cell purity and assess their individual responses.
- **Co-Culture System:** Establish a co-culture system where neurons and astrocytes interact, mimicking the physiological synaptic environment.
3. **Experimental Conditions:**
- **Control Group:** Cells cultured without any external treatment.
- **miRNA Overexpression Group:** Transfect specific miRNA mimics into cell cultures to overexpress the selected miRNAs.
- **CCL5 Treatment Group:** Treat cell cultures with astrocyte-secreted CCL5 to observe its effects on synaptic plasticity.
- **miRNA Inhibition Group:** Inhibit the selected miRNAs in cell cultures to assess the impact on synaptic plasticity.

4. **Assessment of Synaptic Plasticity:**

- Measure dendritic spine density and morphology using imaging techniques like confocal microscopy.
- Evaluate synaptic activity and plasticity through electrophysiological recordings such as patch-clamp assays.

5. **Data Collection and Analysis:**

- Quantify miRNA expression profiles and astrocyte-secreted factor levels using qRT-PCR and ELISA, respectively.
- Analyze synaptic plasticity outcomes through appropriate statistical tests to determine the significance of the observed interactions.

6. **Validation and Generalization:**

- Validate the findings by comparing them with existing literature and other relevant studies to ensure consistency.
- Assess the generalizability of the results to similar neurodegenerative conditions by extending the experimental validation to different model systems.

Rationale:

This experiment design aligns with the research problem's focus on elucidating the role of miRNAs in modulating synaptic plasticity in neurodegenerative conditions through interactions with astrocyte-secreted factors like CCL5. By systematically evaluating specific miRNAs in an in vitro and/or in vivo context and investigating their effects on synaptic plasticity, we aim to address the existing gap in understanding the mechanistic basis of these interactions. The proposed experimental conditions allow for comprehensive data collection and analysis to validate the hypothesized roles of miRNAs and

astrocyte-secreted factors in synaptic plasticity regulation, contributing to advancing our knowledge of neurodegenerative disorders and potential therapeutic interventions targeting miRNA-mediated pathways. The emphasis on data rigor, validation, and broad generalizability ensures the robustness and reproducibility of our findings, laying a strong foundation for dissemination through peer-reviewed publications and scientific conferences to benefit the broader scientific community.

More related paper

Paper 1
Title: NMDAR-dependent Argonaute 2 phosphorylation regulates miRNA activity and dendritic spine plasticity.

Abstract: MicroRNAs (miRNAs) repress translation of target mRNAs by associating with Argonaute (Ago) proteins to form the RNA-induced silencing complex (RISC), underpinning a powerful mechanism for fine-tuning protein expression. Specific miRNAs are required for NMDA receptor (NMDAR)-dependent synaptic plasticity by modulating the translation of proteins involved in dendritic spine morphogenesis or synaptic transmission. However, it is unknown how NMDAR stimulation stimulates RISC activity to rapidly repress translation of synaptic proteins. We show that NMDAR stimulation transiently increases Akt-dependent phosphorylation of Ago2 at S387, which causes an increase in binding to GW182 and a rapid increase in translational repression of LIMK1 via miR-134. Furthermore, NMDAR-dependent down-regulation of endogenous LIMK1 translation in dendrites and dendritic spine shrinkage requires phospho-regulation of Ago2 at S387. AMPAR trafficking and hippocampal LTD do not involve S387 phosphorylation, defining this mechanism as a specific pathway for structural plasticity. This work defines a novel mechanism for the rapid transduction of NMDAR stimulation into miRNA-mediated translational repression to control dendritic spine morphology.

DOI: 10.15252/embj.201797943

The impact factor: 14.012

Paper 2

Title: The conserved microRNA miR-34 regulates synaptogenesis via coordination of distinct mechanisms in presynaptic and postsynaptic cells.

Abstract: Micro(mi)RNA-based post-transcriptional regulatory mechanisms have been broadly implicated in the assembly and modulation of synaptic connections required to shape neural circuits, however, relatively few specific miRNAs have been identified that control synapse formation. Using a conditional transgenic toolkit for competitive inhibition of miRNA function in Drosophila, we performed an unbiased screen for novel regulators of synapse morphogenesis at the larval neuromuscular junction (NMJ). From a set of ten new validated regulators of NMJ growth, we discovered that miR-34 mutants display synaptic phenotypes and cell type-specific functions suggesting distinct downstream mechanisms in the presynaptic and postsynaptic compartments. A search for conserved downstream targets for miR-34 identified the junctional receptor CNTNAP4/Neurexin-IV (Nrx-IV) and the membrane cytoskeletal effector Adducin/Hu-li tai shao (Hts) as proteins whose synaptic expression is restricted by miR-34. Manipulation of miR-34, Nrx-IV or Hts-M function in motor neurons or muscle supports a model where presynaptic miR-34 inhibits Nrx-IV to influence active zone formation, whereas, postsynaptic

miR-34 inhibits Hts to regulate the initiation of bouton formation from presynaptic terminals.

DOI: 10.1038/s41467-020-14761-8

The impact factor: 17.694

Paper 3

Title: Astrocytic miR-324-5p is essential for synaptic formation by suppressing the secretion of CCL5 from astrocytes.

Abstract: There is accumulating evidence that astrocytes play an important role in synaptic formation, plasticity, and pruning. Dicer and the fine-tuning of microRNA (miRNA) network are important for maintaining the normal functions of central nervous system and dysregulation of miRNAs is implicated in neurological disorders. However, little is known about the role of Dicer and miRNAs of astrocytes in the homeostasis of synapse as well as its plasticity. By selectively deleting Dicer in postnatal astrocytes, Dicer-deficient mice exhibited reactive astrogliosis and deficits in dendritic spine formation. Astrocyte-conditioned medium (ACM) collected from Dicer-null astrocytes caused synapse degeneration in cultured primary neurons. The expression of chemokine ligand 5 (CCL5) elevated in Dicer-deleted astrocytes which led to the significant augmentation of secreted CCL5 in ACM. In neurons treated with Dicer KO-ACM, CCL5 supplementation inhibited MAPK/CREB signaling pathway and exacerbated the synaptic formation deficiency, while CCL5 knockdown partially rescued the synapse degeneration. Moreover, we validated CCL5 as miR-324-5p targeted gene. ACM collected from miR-324-5p antagomir-transfected astrocytes mimicked the effect of CCL5 treatment on inhibiting synapse formation and MAPK/CREB signaling in Dicer KO-ACM-cocultured neurons. Furthermore, decreased miR-324-5p expression and elevated CCL5 expression were observed in the brain of aging mice. Our work reveals the non-cell-autonomous roles of astroglial miRNAs in regulation of astrocytic secretory milieu and neuronal synaptogenesis, implicating the loss or misregulation of astroglial miRNA network may contribute to neuroinflammation, neurodegeneration, and aging.

DOI: 10.1038/s41419-019-1329-3

The impact factor: 9.685

Paper 4

Title: MicroRNAs and the Genetic Nexus of Brain Aging, Neuroinflammation, Neurodegeneration, and Brain Trauma.

Abstract: Aging is a complex and integrated gradual deterioration of cellular activities in specific organs of the body, which is associated with increased mortality. This deterioration is the primary risk factor for major human pathologies, including cancer, diabetes, cardiovascular disorders, neurovascular disorders, and neurodegenerative diseases. There are nine tentative hallmarks of aging. In addition, several of these hallmarks are increasingly being associated with acute brain injury conditions. In this review, we consider the genes and their functional pathways involved in brain aging as a means of

developing new strategies for therapies targeted to the neuropathological processes themselves, but also as targets for many age-related brain diseases. A single microRNA (miR), which is a short, non-coding RNA species, has the potential for targeting many genes simultaneously and, like practically all other cellular processes, genes associated with many features of brain aging and injury are regulated by miRs. We highlight how certain miRs can mediate deregulation of genes involved in neuroinflammation, acute neuronal injury and chronic neurodegenerative diseases. Finally, we review the recent progress in the development of effective strategies to block specific miR functions and discuss future approaches with the prediction that anti-miR drugs may soon be used in the clinic.

DOI: 10.14336/AD.2018.0409

The impact factor: 9.968

Paper 5

Title: MicroRNAs Engage in Complex Circuits Regulating Adult Neurogenesis.

Abstract: The finding that the adult mammalian brain is still capable of producing neurons has ignited a new field of research aiming to identify the molecular mechanisms regulating adult neurogenesis. An improved understanding of these mechanisms could lead to the development of novel approaches to delay cognitive decline and facilitate neuroregeneration in the adult human brain. Accumulating evidence suggest microRNAs (miRNAs), which represent a class of post-transcriptional gene expression regulators, as crucial part of the gene regulatory networks governing adult neurogenesis. This review attempts to illustrate how miRNAs modulate key processes in the adult neurogenic niche by interacting with each other and with transcriptional regulators. We discuss the function of miRNAs in adult neurogenesis following the life-journey of an adult-born neuron from the adult neural stem cell (NSCs) compartment to its final target site. We first survey how miRNAs control the initial step of adult neurogenesis, that is the transition of quiescent to activated proliferative adult NSCs, and then go on to discuss the role of miRNAs to regulate neuronal differentiation, survival, and functional integration of the newborn neurons. In this context, we highlight miRNAs that converge on functionally related targets or act within cross talking gene regulatory networks. The cooperative manner of miRNA action and the broad target repertoire of each individual miRNA could make the miRNA system a promising tool to gain control on adult NSCs in the context of therapeutic approaches.

DOI: 10.3389/fnins.2018.00707

The impact factor: 5.152

Paper 6

Title: miRNA-Dependent Control of Homeostatic Plasticity in Neurons.

Abstract: Homeostatic plasticity is a form of plasticity in which neurons compensate for changes in neuronal activity through the control of key physiological parameters such as the number and the strength of their synaptic inputs and intrinsic excitability. Recent studies revealed that miRNAs, which

are small non-coding RNAs repressing mRNA translation, participate in this process by controlling the translation of multiple effectors such as glutamate transporters, receptors, signaling molecules and voltage-gated ion channels. In this review, we present and discuss the role of miRNAs in both cell-wide and compartmentalized forms of homeostatic plasticity as well as their implication in pathological processes associated with homeostatic failure.

DOI: 10.3389/fncel.2019.00536

The impact factor: 6.147

Paper 7

Title: Modulation of MicroRNAs as a Potential Molecular Mechanism Involved in the Beneficial Actions of Physical Exercise in Alzheimer Disease.

Abstract: Alzheimer disease (AD) is one of the most common neurodegenerative diseases, affecting middle-aged and elderly individuals worldwide. AD pathophysiology involves the accumulation of beta-amyloid plaques and neurofibrillary tangles in the brain, along with chronic neuroinflammation and neurodegeneration. Physical exercise (PE) is a beneficial non-pharmacological strategy and has been described as an ally to combat cognitive decline in individuals with AD. However, the molecular mechanisms that govern the beneficial adaptations induced by PE in AD are not fully elucidated. MicroRNAs are small non-coding RNAs involved in the post-transcriptional regulation of gene expression, inhibiting or degrading their target mRNAs. MicroRNAs are involved in physiological processes that govern normal brain function and deregulated microRNA profiles are associated with the development and progression of AD. It is also known that PE changes microRNA expression profile in the circulation and in target tissues and organs. Thus, this review aimed to identify the role of deregulated microRNAs in the pathophysiology of AD and explore the possible role of the modulation of microRNAs as a molecular mechanism involved in the beneficial actions of PE in AD.

DOI: 10.3390/ijms21144977

The impact factor: 6.208

Paper 8

Title: Dysregulation of miRNA and its potential therapeutic application in schizophrenia.

Abstract: Although it is generally believed that genetic and developmental factors play critical roles in pathogenesis of schizophrenia, however, the precise etiological mechanism of schizophrenia remains largely unknown. Over past decades, miRNAs have emerged as an essential post-transcriptional regulator in gene expression regulation. The importance of miRNA in brain development and neuroplasticity has been well-established. Abnormal expression and dysfunction of miRNAs are known to involve in the pathophysiology of many neuropsychiatric diseases including schizophrenia. In this review, we summarized the recent findings in the schizophrenia-associated dysregulation of miRNA and functional roles in the development and pathogenesis of schizophrenia. We also discussed the

potential therapeutic implications of miRNA regulation in the illness.

DOI: 10.1111/cns.12840

The impact factor: 7.035

Paper 9

Title: AFM Imaging Reveals MicroRNA-132 to be a Positive Regulator of Synaptic Functions.

Abstract: The modification of synaptic and neural connections in adults, including the formation and removal of synapses, depends on activity-dependent synaptic and structural plasticity. MicroRNAs (miRNAs) play crucial roles in regulating these changes by targeting specific genes and regulating their expression. The fact that somatic and dendritic activity in neurons often occurs asynchronously highlights the need for spatial and dynamic regulation of protein synthesis in specific milieu and cellular loci. MicroRNAs, which can show distinct patterns of enrichment, help to establish the localized distribution of plasticity-related proteins. The recent study using atomic force microscopy (AFM)-based nanoscale imaging reveals that the abundance of miRNA(miR)-134 is inversely correlated with the functional activity of dendritic spine structures. However, the miRNAs that are selectively upregulated in potentiated synapses, and which can thereby support prospective changes in synaptic efficacy, remain largely unknown. Using AFM force imaging, significant increases in miR-132 in the dendritic regions abutting functionally-active spines is discovered. This study provides evidence for miR-132 as a novel positive miRNA regulator residing in dendritic shafts, and also suggests that activity-dependent miRNAs localized in distinct sub-compartments of neurons play bi-directional roles in controlling synaptic transmission and synaptic plasticity.

DOI: 10.1002/advs.202306630

The impact factor: 17.521

Paper 10

Title: Role of microRNAs in the pathophysiology of addiction.

Abstract: Addiction is a chronic and relapsing brain disorder characterized by compulsive seeking despite adverse consequences. There are both heritable and epigenetic mechanisms underlying drug addiction. Emerging evidence suggests that non-coding RNAs (ncRNAs) such as microRNAs (miRNAs), long non-coding RNAs, and circular RNAs regulate synaptic plasticity and related behaviors caused by substances of abuse. These ncRNAs modify gene expression and may contribute to the behavioral phenotypes of addiction. Among the ncRNAs, the most widely researched and impactful are miRNAs. The goal in this systematic review is to provide a detailed account of recent research involving the role of miRNAs in addiction. This article is categorized under: RNA Interactions with Proteins and Other Molecules > Small Molecule-RNA Interactions RNA in Disease and Development > RNA in Disease.

DOI: 10.1002/wrna.1637

The impact factor: 9.349