Opinion

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Role of microRNAs in the Pathogenesis of Pituitary Tumors

Mitchell Parkin*

Department of Neurosurgery, Poznan University of Medical Science, Przybyszewskiego, Poznań, Poland

Corresponding author: Mitchell Parkin, Department of Neurosurgery, Poznan University of Medical Science, Przybyszewskiego, Poznań, Poland, E-mail: park.mithchell@poznan.edu

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Introduction

Pituitary tumors are among the most common intracranial neoplasms, typically benign but with significant clinical implications due to hormonal imbalances and mass effects. The molecular mechanisms driving their initiation and progression are complex and multifactorial, involving genetic, epigenetic, and environmental factors. In recent years, microRNAs (miRNAs), a class of small non-coding RNAs that regulate gene expression post-transcriptionally, have emerged as key players in tumor biology. Their dysregulated expression in pituitary tumors has provided new insights into tumorigenesis, suggesting roles in cell proliferation, apoptosis, invasion, and endocrine dysfunction [1].

Description

MiRNAs regulate gene expression by binding to the 3' untranslated regions of target mRNAs, leading to degradation or translational repression. In pituitary tumors, aberrant expression of certain miRNAs has been consistently observed. For example, downregulation of tumor-suppressive miRNAs, such as miR-15a and miR-16, has been associated with unchecked cell proliferation, while overexpression of oncogenic miRNAs like miR-26b promotes tumor cell survival. This dysregulation alters the expression of key signaling molecules and transcription factors, driving tumor initiation and progression [2].

Several studies have highlighted the role of miRNAs in modulating pituitary tumor cell cycle regulators and apoptotic pathways. For instance, altered expression of miR-21 and miR-34a impacts the p53 pathway, leading to reduced apoptosis and enhanced tumor growth. Additionally, miRNAs such as miR-143 and miR-145, which normally suppress oncogenic signaling pathways, are often downregulated in pituitary adenomas, contributing to their uncontrolled expansion. These findings underscore the functional relevance of miRNAs in maintaining the balance between growth and programmed cell death within pituitary tissue [3].

Endocrine activity in pituitary tumors is also influenced by miRNAs. Hormone-secreting adenomas, such as prolactinomas and corticotropinomas, often exhibit unique miRNA signatures. Dysregulated miRNAs affect transcription factors like Pit-1 and Tpit, which govern hormone production. For example, overexpression of miR-107 and miR-375 has been linked to altered prolactin secretion, while changes in miR-124 and miR-132 levels influence Adrenocorticotropic Hormone (ACTH) release. These regulatory effects reveal how miRNAs contribute not only to tumor growth but also to the endocrine dysfunctions characteristic of pituitary adenomas [4].

Moreover, miRNAs play a role in tumor invasiveness and response to therapy. In invasive pituitary adenomas, overexpression of miR-93 and downregulation of miR-200 family members have been associated with epithelial-mesenchymal transition (EMT)-like processes, enhancing tumor aggressiveness. Importantly, miRNA expression profiles are being explored as potential biomarkers for predicting tumor subtype, invasiveness, and treatment response. For instance, resistance to dopamine agonists in prolactinomas has been linked to altered expression of specific miRNAs, raising the possibility of using miRNA-based diagnostics to personalize therapy [5].

Conclusion

MicroRNAs are central regulators in the pathogenesis of pituitary tumors, influencing tumor growth, hormone secretion, invasiveness, and treatment response. Their dual roles as oncogenes and tumor suppressors highlight the complexity of miRNA-mediated regulation in pituitary tumor biology. With advancing research, miRNA profiling holds promise as a diagnostic and prognostic tool, and miRNA-targeted therapies may emerge as novel treatment strategies. Ultimately, a deeper understanding of miRNA networks could pave the way for precision medicine approaches in the management of pituitary tumors.

Acknowledgement

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Conflict of Interest

None.

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