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Introduction

Hepatocellular carcinoma and the role of RNA-binding proteins

Hepatocellular Carcinoma (HCC), constituting a primary liver cancer, stands as a formidable contributor to cancer-related mortality globally. The molecular intricacies governing the malignant progression of HCC have seized the attention of the scientific community, unveiling a complex landscape in which RNA-binding proteins (RBPs) emerge as pivotal orchestrators of gene expression. This article delves into the evolving understanding of the role played by RBPs in hepatocellular carcinoma, placing a specific focus on the ribosomal protein S5 (RPS5).

The landscape of RNA-binding proteins in cancer: RNA-binding proteins, a diverse class of cellular regulators, play crucial roles in the post-transcriptional regulation of gene expression. These proteins intricately engage in mRNA processing, stability maintenance, transport facilitation, and translation control, thereby exerting substantial influence over fundamental cellular functions. The delicate balance maintained by RBPs in gene expression becomes disrupted in various cancers, including HCC. Such dysregulation contributes significantly to the initiation and progression of tumors by influencing critical cellular processes.

The crucial role of RPS5 in hepatocellular carcinoma: Within the expansive realm of RNA-binding proteins, the ribosomal protein S5 (RPS5) has emerged as a focal point of investigation concerning hepatocellular carcinoma. Traditionally acknowledged for its integral role in protein synthesis as part of the small ribosomal subunit, RPS5 has captivated researchers with the revelation of its involvement beyond the ribosome. Emerging evidence suggests that RPS5 extends its influence to regulatory processes that impact essential cellular functions. Beyond its canonical role, RPS5 has been implicated in pivotal cellular processes associated with cancer biology. Notably, RPS5 has been linked to cell proliferation, a hallmark of cancer [1,2]. Its involvement in regulating the cell cycle and promoting cell division underscores its potential significance in driving the uncontrolled growth observed in hepatocellular carcinoma. Additionally, RPS5 exhibits connections to apoptosis, the programmed cell death process, raising intriguing questions about its role in evading this natural safeguard against abnormal cell proliferation. The potential involvement of RPS5 in carcinogenesis, the process of tumor initiation, further adds layers to its significance in the malignant progression of HCC. As research endeavors delve deeper into the intricate mechanisms by which RPS5 influences hepatocellular carcinoma, the multifaceted nature of its role becomes increasingly apparent. The traditional view of RPS5 as a structural component of the ribosome expands to encompass its participation in critical regulatory circuits governing cell fate decisions [3,4]. In conclusion, the exploration of RNA-binding proteins, particularly the ribosomal protein S5, in the context of hepatocellular carcinoma represents a promising frontier in cancer research. Unraveling the precise mechanisms through which RPS5 contributes to the malignant progression of HCC not only deepens our understanding of this complex disease but also opens avenues for targeted therapeutic interventions. As our comprehension evolves, the hope is that the knowledge gained will translate into effective strategies for diagnosis, prognosis, and the development of precision therapies tailored to the unique molecular intricacies of hepatocellular carcinoma.

Interaction of RPS5 with oncogenic pathways in HCC: Research has provided compelling evidence suggesting that the ribosomal protein S5 (RPS5) interacts with key oncogenic pathways implicated in Hepatocellular Carcinoma (HCC). Its association with signaling cascades involving Wnt/β-catenin, PI3K/Akt, and MAPK pathways points towards a multifaceted role in the malignant transformation of hepatocytes [5]. These pathways, pivotal in cellular regulation, are often dysregulated in cancer, contributing to uncontrolled cell proliferation, evasion of apoptosis, and enhanced tumor cell survival.

Citation: