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Short Communication about Interactions between the RNA-Binding Protein IGF2BP1 and Tumorigenic Viruses

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Abstract

Viral infections are responsible for a significant proportion of human cancers. Virus propagation as well as infection-induced tumorigenesis rely on interactions with host factors. The review published in Viruses summarizes the current knowledge about the collaborations between the oncofetal RNA-binding protein IGF2BP1 and distinct tumorigenic viruses. The interplay between IGF2BP1 and viruses can support pro-proliferative traits of the infected cells but can also augment the propagation of the virus.

Keywords: IGF2BP1; Hepatitis B and C viruses; Human papillomaviruses

About the Study

Infections with tumorigenic viruses are estimated to be responsible for around 12% of all human cancers [1,2]. Hepatitis B and C Viruses (HBV/HCV) as well as Human Papillomaviruses (HPV) alone are attributable for more than 80% of all virus induced cancers and these viruses have been reported to be associated with the oncofetal RNA-binding protein IGF2BP1 [2-5]. This protein, that is severely upregulated in various malignancies and associated with the expression of oncogenic factors, interacts with the before-mentioned as well as other viruses *via* different mechanisms [6]. These interactions have recently been summarized in a review by Glaß and Hüttelmaier [7].

IGF2BP1 cooperatively interacts with tumorigenic viruses

Yan, et al. described the stabilization of the proto-oncogenic transcription factor c-Myc by IGF2BP1 in dependence of HBV's HBx protein. HBx interacts with the DNA methyl-transferase DNMT3A leading to hypermethylation of the promoter of the protein tyrosine phosphatase PTPN13, resulting in reduced PTPN13 transcription. Reduced PTPN13 levels, in turn, facilitate increased binding between IGF2BP1 and c-Myc mRNA, leading to stabilization of these transcripts and thus contribute to promotion of tumor progression [3].

By binding to the internal ribosomal entry site (IRES) and 3' UTR regions of the HCV RNA genome, IGF2BP1 increases translation rates of HCV. Furthermore, HCV proteins were shown to increase IGF2BP1 protein levels in hepatocellular carcinoma-derived cell lines [4]. MRNA of HPV's E7 protein, facilitating progression of cell cycle transition in infected cells, was reported by Wang, et al. to be bound and thus stabilized by IGF2BP1. This association led to increased E7 levels in HPV16 positive cell lines. However, the E7-IGF2BP1 complex was shown to be vulnerable to heat stress, thus, offering a putative new treatment option [5].

The RNA-binding protein IGF2BP1 plays a multifaceted role in post-transcriptional gene regulation. This protein is recognized for its ability to selectively interact with specific RNA molecules, thus influencing their localization, stability, translation, and ultimately impacting various cellular processes. IGF2BP1's intricate involvement in RNA metabolism extends to its participation in cellular pathways such as cell proliferation, migration, and differentiation. Moreover, its interactions with viral RNAs or proteins have been found to extend beyond specific virus species, encompassing a diverse range of families.

Conclusion

The RNA-binding protein IGF2BP1 has been shown to interact with a multitude of virus species. Besides the viruses mentioned here, additional virus species from various families have been reported to interact with IGF2BP1 and its homologs. Associations between IGF2BP1 and viral RNAs or proteins can lead to alterations in virus production, infectivity and replication. In transformed cells infected with HBV or HPV, collaborations between IGF2BP1 and viral proteins or RNAs further contribute to proliferative phenotypes and, thus, disrupting IGF2BP1-virus associations could lead to novel treatment strategies.

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