RNA-Binding Proteins and Circular RNAs: A New Frontier in Cancer Therapy

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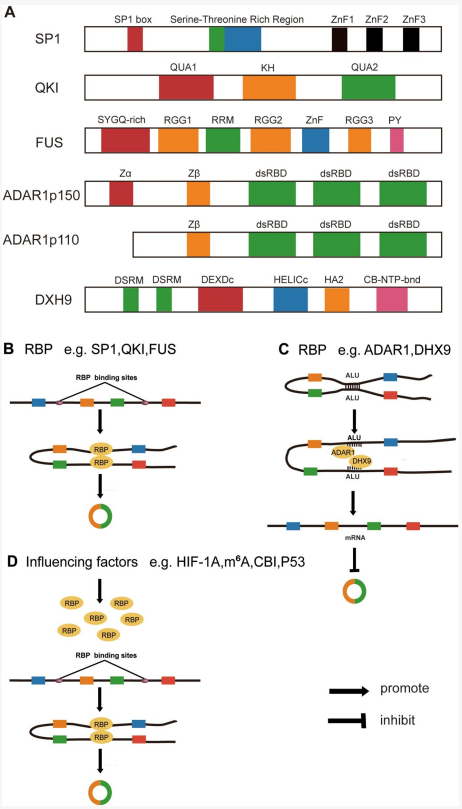
The interaction between RNA-binding proteins (RBPs) and circular RNAs (circRNAs) has emerged as a key area of interest in understanding cancer biology. As critical regulators of gene expression, RBPs control the formation and function of circRNAs, influencing various cancer-related processes such as tumor proliferation, metastasis, drug resistance, and immune evasion. This dynamic interplay has positioned the circRNA-RBP network as a promising target for developing innovative cancer therapies.



The interaction between circular RNA (circRNA) and RNA-binding protein (RBP).

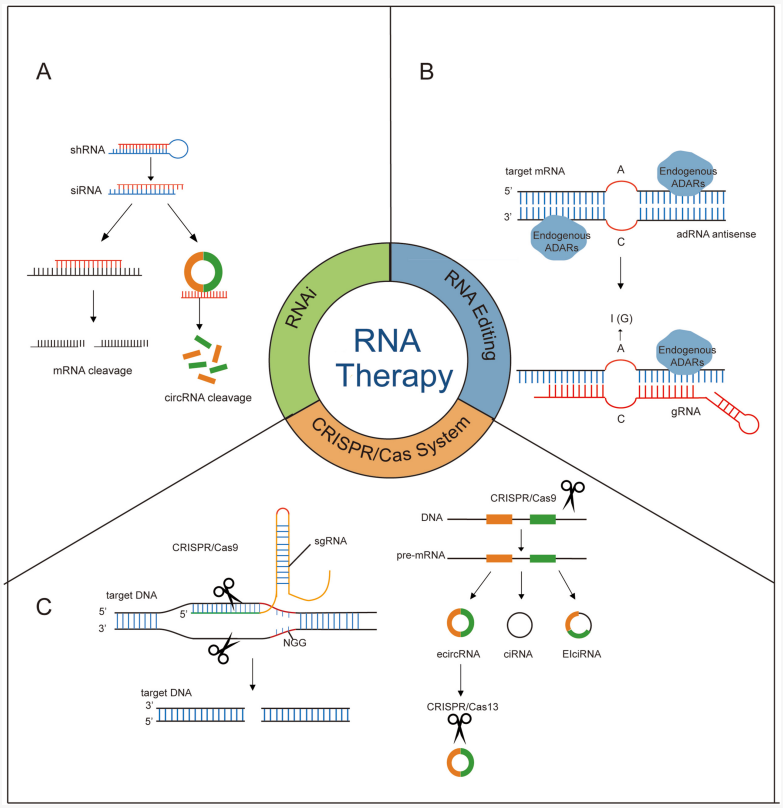
CircRNAs are unique RNA molecules formed through a back-splicing mechanism that connects the 5' and 3' ends of precursor mRNA. Previously considered splicing errors, circRNAs are now recognized for their roles as molecular sponges for microRNAs (miRNAs) and proteins, as well as their capacity to regulate RNA-protein interactions. RBPs play a crucial role in circRNA biogenesis and function, with specific proteins like QKI, SP1, FUS, ADAR1, and DHX9 shown to either promote or inhibit circRNA production, thereby shaping tumor characteristics.

RBPs such as QKI and FUS enhance circRNA formation by facilitating the reverse splicing process. For instance, QKI's binding to precursor mRNA promotes circularization, while FUS interacts with circRNAs to form positive feedback loops, sustaining the expression of oncogenic circRNAs. In contrast, ADAR1 and DHX9 suppress circRNA production by editing RNA sequences or destabilizing RNA structures. The intricate regulation by RBPs makes circRNAs pivotal in controlling tumor growth, metastasis, and response to therapy.

A notable aspect of circRNA-RBP interactions is their role in the tumor microenvironment (TME), where factors like hypoxia alter RBP expression and activity. Hypoxia-induced changes can either enhance or inhibit circRNA formation, affecting tumor behavior. Furthermore, N6-methyladenosine (m6A) modification on circRNAs has been shown to modulate their interaction

with RBPs, impacting tumorigenesis and cancer progression. This modification not only affects RBP binding but also regulates circRNA stability and translation, highlighting its potential as a therapeutic target.

The discovery of the circRNA-RBP network has spurred the development of RNA-based therapies. Techniques such as RNA interference (RNAi), RNA editing, and the CRISPR/Cas system are being explored to target specific RBPs and circRNAs. By disrupting harmful circRNA-RBP interactions or enhancing beneficial ones, these therapies aim to inhibit cancer progression while minimizing off-target effects. Innovations like ADAR-mediated RNA editing and CRISPR-Cas13 systems demonstrate the feasibility of precisely targeting the circRNA-RBP axis, paving the way for personalized cancer treatment.



The approaches to RNA therapy targeting the circular RNA (circRNA)-RBP (RNA-binding protein) interactions.

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Genes & Diseases Editorial Office
Genes & Diseases
+86 23 6571 4691
editor@genesndiseases.com
