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The Crosstalk between microRNAs and Long Non-coding RNAs in Hepatocellular Carcinoma

Xueping Jiang

Department of Oncology, The Central Hospital of Enshi Tujia and Miao Autonomous Prefecture, Enshi, Hubei, 445000, China

Abstract: Hepatocellular carcinoma (HCC) is one of the most prevalent malignant tumors worldwide, with China exhibiting the highest incidence and mortality rates in Asia. Over the past decades, significant progress has been made in the early detection and treatment of HCC. However, the molecular mechanisms underlying hepatocarcinogenesis remain incompletely understood. Increasing evidence indicates that non-coding RNAs (ncRNAs) are crucial regulators of diverse biological and pathological processes, including X chromosome inactivation, transcriptional activation or interference, genomic imprinting, and intranuclear transport. Moreover, accumulating studies have revealed intricate network interactions among different ncRNAs. In particular, the crosstalk between microRNAs (miRNAs) and long non-coding RNAs (lncRNAs) has attracted considerable attention. In this review, we summarize current knowledge regarding miRNA–lncRNA interactions and their functional roles in HCC, aiming to provide novel insights into the development of molecularly targeted therapies for this disease.

Keywords: Hepatocellular Carcinoma; Non-coding RNAs; MicroRNAs; Long non-coding RNAs; Crosstalk; Molecular Targeted Therapy.

1. Introduction

Hepatocellular carcinoma (HCC) is one of the most common malignant tumors worldwide, characterized by a high incidence and poor prognosis. In 2018, approximately 841,080 new cases and 781,631 deaths were reported globally [1]. Although remarkable progress has been made in the diagnosis and treatment of HCC over the past decades, the aggressive biological behavior of the disease continues to pose significant challenges for improving patient outcomes. Therefore, a more comprehensive understanding of the molecular mechanisms underlying HCC may contribute to the development of novel strategies for molecularly targeted therapies in the future [2].

Previously, it was found that most of the genome can be transcribed into RNA; however, only 1%–2% of the human genome encodes proteins. Based on transcript length, non-coding RNAs (ncRNAs) are generally divided into two major categories [3]. NcRNAs shorter than 200 nucleotides (nt) are classified as small or short ncRNAs, whereas those longer than 200 nt are referred to as long non-coding RNAs (lncRNAs). In this review, we mainly focus on microRNAs (miRNAs) and lncRNAs [4].

MiRNAs, a class of highly conserved small RNAs of approximately 18–25 nt, regulate gene expression by binding to the 3′ untranslated region (3′UTR) of target mRNAs, thereby promoting mRNA degradation or inhibiting translation [5]. Accumulating evidence has demonstrated that miRNAs participate in diverse physiological processes, including cell proliferation, apoptosis, and death, and they are also implicated in the development of multiple tumor types. For instance, overexpression of the miR-23b/27b/24-1 cluster markedly inhibits proliferation, migration, and invasion of prostate cancer cells [6].

LncRNAs, typically longer than 200 nt, are usually transcribed by RNA polymerase II and possess a 5′ cap structure and a 3′ poly(A) tail (4). Increasing attention has been paid to their roles in the progression of HCC. For example, lncRNAs such as highly upregulated in liver cancer (HULC), HOX transcript antisense intergenic RNA (HOTAIR), and HOXA distal transcript antisense RNA (HOTTIP) function as oncogenes, whereas maternally expressed gene 3 (MEG3) acts as a tumor suppressor in HCC [7]. In this review, we further analyze the interplay between miRNAs and lncRNAs, aiming to provide novel insights into molecularly targeted therapies for HCC [7].

2. Mechanisms of the Interplay among non-coding RNAs

MiRNAs and lncRNAs are closely interrelated, exerting important biological functions through mutual regulation and interaction. Increasing research attention has been devoted to their remarkable characteristics, including specificity, diversity, and regulatory efficiency.

(1) MiRNAs regulate lncRNAs

Both miRNAs and lncRNAs are classified as non-coding RNAs, and a large number of lncRNAs are regulated by miRNAs. Recent evidence has demonstrated that miRNAs can modulate the expression of more than one-third of protein-coding genes by binding to their 3′ untranslated regions (3′UTRs) [8]. Among them, miR-21 has been identified as an oncogenic miRNA in various malignancies, including gastric, esophageal, and breast cancers [8]. In breast cancer, miR-21 regulates the expression of the lncRNA growth arrest-specific transcript 5 (GAS5) through the RNA-induced silencing complex (RISC) signaling pathway [9]. Argonaute 2 (AGO2), the core component of RISC, can be pulled down by a Bio-GAS5 RNA probe, and miR-21 has been identified as a critical component of the GAS5–RISC complex. These findings suggest that miR-21 regulates GAS5 expression via a RISC-dependent pathway and thereby contributes to the regulation of biological functions [9]. Because many lncRNAs and mRNAs share similar structural regions, miRNAs are capable of targeting mRNAs through binding to their 3′UTRs while simultaneously regulating lncRNAs via the RISC-associated pathway. For example, miR-9 has been shown to regulate the lncRNA metastasis-

associated lung adenocarcinoma transcript 1 (MALAT1) through RISC-mediated interactions [10].

(2) LncRNAs regulate miRNAs

MiRNAs and lncRNAs interact through diverse and intricate mechanisms, thereby influencing tumor progression. On the one hand, miRNAs can regulate lncRNAs by promoting their degradation or modulating their expression via the RNA-induced silencing complex (RISC). For example, miR-21 regulates the lncRNA GAS5 through a RISC-dependent pathway, while miR-9 targets MALAT1, highlighting the ability of miRNAs to directly control lncRNA expression. On the other hand, lncRNAs can in turn regulate miRNAs through multiple mechanisms: (i) acting as endogenous sponges to sequester miRNAs, such as lncRNA HULC functioning as a sponge for miR-372 in HCC; (ii) competing with mRNA 3′UTRs for miRNA binding, exemplified by PTENP1, which alleviates the post-transcriptional repression of PTEN [11]; (iii) serving as precursors of miRNAs, as lncRNA H19 generates miR-576 after Drosha/Dicer processing and is implicated in cancer metastasis[12]; and (iv) functioning as competitive endogenous RNAs (ceRNAs), thereby modulating miRNA availability for their target transcripts [13,14]. Collectively, these mechanisms underscore the complex regulatory networks between miRNAs and lncRNAs and their crucial roles in hepatocellular carcinoma and other malignancies.

3. The Crosstalk between miRNAs and lncRNAs in HCC

Both miRNAs and lncRNAs are critical regulatory molecules involved in gene expression and play essential roles in the development of human cancers. While individual miRNAs or lncRNAs can regulate specific cellular phenotypes, increasing evidence highlights the importance of their coordinated interactions. In this review, we focus on the crosstalk between miRNAs and lncRNAs in the development of hepatocellular carcinoma (HCC) (**Figure 1**).

(1) HULC

HULC (highly upregulated in liver cancer) is the most abundantly expressed lncRNA in HCC, located on chromosome 6p24.3 and spanning 485 nucleotides. The HULC gene contains one intron and two exons, with lengths of approximately 1153 nt, 182 nt, and 303 nt, respectively. Its upstream region harbors one canonical AATAAA and two noncanonical AATTAA polyadenylation signals, positioned 21 nt, 32 nt, and 47 nt away from the poly(A) tail [15].

A cAMP response element-binding protein (CREB) binding site has been identified in the HULC promoter region, which plays a critical role in transcriptional initiation. Activation of the protein kinase A (PKA) signaling pathway directly stimulates CREB, and phosphorylated CREB (p-CREB) enhances HULC expression [14]. In turn, HULC functions as an endogenous antagonist by binding to miR-372, thereby inhibiting its activity. Since miR-372 targets the 3′UTR of PRKACβ, which induces CREB phosphorylation, this regulatory loop ultimately leads to enhanced HULC expression. Cui et al. further demonstrated that HULC induces methylation of CpG islands in the miR-9 promoter by upregulating DNA (cytosine-5-)-methyltransferase 1 (DNMT1). This process leads to increased expression of peroxisome proliferator-activated receptor alpha (PPARA) mRNA and transcriptional activation of acyl-CoA synthetase long-chain family member 1 (ACSL1). ACSL1, in turn, promotes lipid droplet formation, accumulation of triglycerides and cholesterol, and subsequent tumor growth. Moreover, cholesterol produced via ACSL1-mediated lipid metabolism activates RXRA, further enhancing HULC expression [16].

In addition, overexpression of miR-203 has been shown to inhibit HULC expression, thereby promoting cell proliferation and inhibiting apoptosis in HCC cells [17]. HULC has also been implicated in regulating high mobility group A2 (HMGA2), a protein associated with metastatic potential. Through the HULC–miR-186–HMGA2 axis, HULC may broadly modulate oncogenes by sponging miR-186, ultimately promoting HCC cell growth and proliferation [18].

(2) HOTAIR

HOTAIR acts as a molecular scaffold that recruits the polycomb repressive complex 2 (PRC2), thereby facilitating chromatin remodeling. Knockdown of enhancer of zeste homolog 2 (EZH2), a key catalytic subunit of PRC2, has been shown to increase miR-218 expression and its promoter activity. miR-218 functions as a tumor suppressor by inhibiting cell survival and reducing tumorigenesis in HCC. Mechanistically, miR-218 directly targets Bmi-1, a protein essential for embryonic development and cell cycle regulation. Bmi-1 acts as an oncogene that maintains cancer stem cell phenotypes. Its downstream effectors include P16^INK4a and P14^ARF: the latter binds to and inhibits Mdm2, an antagonist of p53, leading to p53 accumulation; while activation of the P16^INK4a–Rb signaling pathway induces cellular senescence, although P16^INK4a is frequently inactivated in tumors. Collectively, the HOTAIR–EZH2–miR-218 axis functions as a protumorigenic pathway in HCC [19].

In addition, the FOXC1–HOTAIR–miR-1 axis has also been implicated in hepatocarcinogenesis. The transcription factor FOXC1 binds to the E-box element in the HOTAIR promoter, enhancing its transcription, whereas miR-1 directly targets HOTAIR to suppress its expression. Acting as a tumor suppressor, miR-1 inhibits tumor cell proliferation and metastasis while promoting apoptosis [20]. Furthermore, HOTAIR has been found to be significantly overexpressed in HCC tissues and cell lines. It has been demonstrated that HOTAIR can sponge miR-23b-3p, thereby upregulating zinc finger E-box-binding homeobox 1 (ZEB1), a transcription factor that induces epithelial–mesenchymal transition (EMT). As ZEB1 expression is positively correlated with poor patient survival, the HOTAIR–miR-23b-3p–ZEB1 axis represents another critical oncogenic pathway contributing to HCC progression [21].

(3) HOTTIP

HOTTIP functions as an oncogene and is markedly upregulated in HCC. Overexpression of HOTTIP promotes cell proliferation and tumorigenesis, whereas its downregulation suppresses cell migration and distant metastasis [22]. Clinically, elevated HOTTIP expression is associated with distant metastasis and poor survival outcomes in HCC patients. In HCC cell lines, increased miR-125b expression reduces HOTTIP levels [23]. Moreover, the miR-192/miR-204–HOTTIP–GLS1 axis has been identified as an important pathway in hepatocarcinogenesis. Specifically, miR-192 and miR-204 inhibit HOTTIP expression through an AGO2-mediated RNA interference mechanism, thereby suppressing glutaminase (GLS1) expression and ultimately attenuating HCC growth [24].

(4) UCA1

Urothelial carcinoma–associated 1 (UCA1), located on chromosome 19p13.12, contains three introns and encodes two different transcripts of approximately 1.4 kb and 2.2 kb, respectively. In the UCA1–miR-216b–FGFR1–ERK axis, upregulated UCA1 directly binds to miR-216b, thereby increasing FGFR1 expression and subsequently activating the ERK signaling pathway. UCA1 expression is significantly elevated in HCC cell lines, where it promotes cell metastasis, whereas its downregulation inhibits cell proliferation, invasion, and migration. Acting as an endogenous antagonist, UCA1 reduces the expression of miR-216b, which otherwise suppresses cell proliferation, colony formation, and induces G0/G1 cell cycle arrest [25].

Additionally, UCA1 directly targets miR-203, a tumor suppressor that downregulates the protein level of Snail2. In HCC, UCA1 promotes epithelial–mesenchymal transition (EMT) by sponging miR-203 and activating Snail2, a potent driver of EMT [26].

(5) XIST

The expression levels of X-inactive specific transcript (XIST) and its upstream regulator just proximal to XIST (JPX) are markedly decreased in HCC tissues [27]. In the XIST/miR-92b/Smad7 axis, XIST suppresses miR-92b expression, which directly targets Smad7. In HCC, miR-92b is upregulated, whereas Smad7 is downregulated. As Smad7 is a negative regulator of the TGF-β/β-catenin signaling pathway, XIST upregulation increases Smad7 protein levels and thereby inhibits HCC cell proliferation and migration. Furthermore, JPX positively regulates XIST expression and is itself a direct target of miR-155-5p. miR-155-5p directly suppresses PTEN and SOX6, both of which function as tumor suppressors in HCC by inhibiting HepG2 cell growth. In the JPX–XIST–miR-155-5p–PTEN/SOX6 axis, XIST acts as a molecular sponge for miR-155-5p, thereby enhancing the expression of PTEN and SOX6 [27].

Additionally, miR-181a has been reported to be upregulated in HCC patients with metastasis and directly targets PTEN. A negative correlation exists between XIST and miR-181a. In the XIST–miR-181a–PTEN axis, XIST promotes PTEN expression by sequestering miR-181a, leading to the suppression of HCC cell proliferation [28].

(6) CCAT1

Colon cancer–associated transcript-1 (CCAT1), located on chromosome 8q24.21, is approximately 2 kb in length [28]. In HCC tissues, CCAT1 expression is significantly elevated and correlates with tumor size, microvascular invasion, elevated alpha-fetoprotein levels, and poor diagnostic sensitivity. Functionally, CCAT1 promotes HCC cell proliferation and migration. In the CCAT1–let-7–c-Myc/HMGA2 axis, CCAT1 acts as a molecular sponge for let-7, thereby relieving its inhibitory effects and leading to the upregulation of c-Myc and HMGA2, which drive tumor progression [29].

In addition, CCAT1 has been identified as a target of miR-490-3p, which directly suppresses cyclin-dependent kinase 1 (CDK1). By sponging miR-490-3p, CCAT1 attenuates its inhibitory effect on CDK1, thereby enhancing CDK1 expression and promoting tumorigenesis in HCC [30].

(7) GAS5

Growth arrest–specific transcript 5 (GAS5) is significantly downregulated in HCC tissues, whereas miR-21 is markedly upregulated [31]. Hu et al. demonstrated that overexpression of GAS5 decreases miR-21 levels, thereby restoring the expression of PTEN and PDCD4, two well-known tumor suppressors. Consequently, GAS5 exerts tumor-suppressive effects in HCC by modulating the GAS5–miR-21–PTEN/PDCD4 axis [31].

4. Perspective

In recent years, research on the biological significance and clinical utility of non-coding RNAs in cancer has advanced rapidly. Tumorigenesis is a multifactorial and highly complex process involving diverse molecular pathways. The development of tumors is not only regulated by numerous miRNAs and lncRNAs but also modulated by competitive endogenous RNA (ceRNA) networks composed of these molecules. The interplay between miRNAs and lncRNAs plays pivotal roles in tumor initiation, progression, invasion, and metastasis.

Elucidating the mechanisms underlying the crosstalk between miRNAs and lncRNAs is crucial for improving the diagnosis, treatment, and prognosis of malignant tumors through the modulation of specific signaling pathways. With continuing research, an increasing number of molecular mechanisms have been uncovered, but the combined effects of miRNAs and lncRNAs remain to be fully understood. Several key questions require further investigation. First, can high-quality and comprehensive databases of lncRNAs be established? Second, are there rigorous, efficient, and accurate methods to systematically explore miRNA–lncRNA interactions? Finally, is it possible to simultaneously modulate the expression of both miRNAs and lncRNAs to effectively inhibit tumor progression?

Although these challenges will persist for the foreseeable future, investigating the critical roles of non-coding RNAs in HCC and exploring their therapeutic potential remains a promising and worthwhile direction.

5. Conclusion

The interplay between miRNAs and lncRNAs plays a pivotal role in the initiation and progression of hepatocellular carcinoma by regulating key oncogenic and tumor-suppressive pathways. A deeper understanding of this crosstalk may provide novel biomarkers and therapeutic targets, offering new opportunities for the diagnosis and treatment of HCC.

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