Advances in targeting of miR-10-associated lncRNAs/circRNAs for the management of cancer (Review)

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Abstract. With advancements in sequencing technologies, an increasing number of aberrantly expressed long-non-coding RNAs (lncRNAs) and circular RNAs (circRNAs) have been identified in various types of cancer. lncRNAs and circRNAs are now well-established tumor-influencing factors in cancer, driving not only tumor proliferation and invasion, but also cancer progression, drug resistance and metastatic recurrence. The majority of lncRNAs and circRNAs influence cancer progression by targeting microRNAs (miRNAs/miRs). miR-10a and miR-10b, key members of the miR-10 family, have been shown to play important regulatory roles in cell proliferation, differentiation to cancer progression, and development. Manual evaluation and grouping according to different types of competing endogenous RNA and tumor was performed. The review outlined the current state of knowledge on the regulation of miR-10 family-related lncRNAs and circRNAs. The involvement of lncRNAs and circRNAs in the biogenesis, maturation and function of malignant tumors through the miR-10 family, and the key gene targets and signaling cascades that lncRNAs and circRNAs regulate through the miR-10 family were summarized. Based on the findings of this review, it can be hypothesized that lncRNAs and circRNAs targeting the miR-10 family may serve as diagnostic/prognostic markers and/or therapeutic targets for the management of cancer.

Contents

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1. Introduction

Cancer is one of the leading causes of death worldwide, and the steady year-on-year increase in cancer incidence and mortality cannot be ignored. According to data released by the World Health Organization in 2019, malignant tumors are the first or second leading cause of death in 112 of 183 countries (1). In 2022, there were 4.82 million new cancer cases in China and 2.37 million new cancer cases in the United States, and 3.21 million and 640,000 cancer deaths (2). Despite continuous progress in radiotherapy, chemotherapy and surgery (3), due to the lack of effective diagnostic markers and detection methods in the early stage, a number of patients receive a late diagnosis, and thus have a poor prognosis and a low 5-year survival rate (4). Therefore, it is important to formulate a cancer treatment strategy, to understand the occurrence and development of cancer, and to identify markers of early diagnosis and treatment.

Cancer is related to endogenous factors, such as gene mutations, epigenetic damage, chromosomal deletions and abnormal amplification, as well as exogenous factors, such as living habits and environmental pollution (5,6). It has previously been reported that oncogene activation leads to abnormal tumor cell proliferation (7). In addition, recent studies reported that non-coding RNAs (ncRNAs) are expressed in a variety of malignant tumors. and affect the occurrence, development and sensitivity of malignant tumors to molecular targeted therapy (2,4). ncRNAs are an emerging class of genomically encoded transcripts (5,8). In recent years, with the innovation of sequencing technologies, several ncRNAs have been detected in eukaryotic genomes, and are estimated to account for >80% of the genome (8,9). Approximately 50,000 ncRNAs have been identified in the last 10 years; however, most of them have not been reported on in depth (10). ncRNAs include microRNAs (miRNAs/miRs), long non-coding RNAs (lncRNAs), small interfering RNAs and circular RNAs (circRNAs) (9). Currently, miRNAs, lncRNAs and circRNAs are research hotspots in the field of biomedical science, as

they mediate a variety of cellular processes, including chromatin remodeling, gene transcription, epigenetic regulation, post-transcriptional modification, cell cycle regulation, cell differentiation and signal transduction (8,11,12). In terms of epigenetic changes, dysregulation of circRNAs, lncRNAs and miRNAs has been shown to affect the development and progression of cervical and thyroid cancer (13,14). In addition, ncRNAs can act as oncogenic molecules and tumor suppressors in cancer, including thyroid cancer (15,16), prostate cancer (PCa) (17), melanoma (18) and osteosarcoma (19), by interacting with coding proteins. Notably, lncRNAs and circRNAs can regulate cancer development by acting as miRNA sponges involved in the transcription of proteins. In the present review, PubMed (https://pubmed.ncbi.nlm.nih.gov) and Google Scholar (https://scholar.google.com) were used to search the keywords 'miR-10a', 'miR-10b', 'cancer' or 'tumor' and 'circRNA' or 'lncRNA'. The present review aimed to describe the specific regulatory mechanisms of lncRNA and circRNA in cancer through the miR-10 family.

2. ncRNAs

lncRNAs. lncRNAs are long non-coding transcription factors that are >200 nucleotides in length (20). lncRNAs do not encode proteins due to the lack of an open reading frame (ORF); however, they can serve as important regulatory RNAs (21). The vast majority of lncRNAs are transcribed by RNA polymerase II, form 5' cap structures and undergo polyadenylation at the 3' region (21,22). They are classified into the following four groups based on their relative position to the protein-coding genes: i) intergenic lncRNAs; ii) intronic lncRNAs; iii) overlapping lncRNAs; and iv) antisense lncRNAs (23). Most of these lncRNAs can interact with RNA or DNA molecules through base pairing, forming functional networks that consist of DNA, protein and RNA, and that are involved in the inheritance and transcription of genes (20,21,23). Furthermore, there is growing evidence that lncRNAs act as oncogenic factors in malignant tumors, inhibiting or promoting cell proliferation, apoptosis, metastatic differentiation and cell invasion in cancer cells (14-18), as well as regulating the metabolic reprogramming of cancer cells (22).

circRNAs. circRNAs are a specific class of RNA widely found in mammalian cells that were initially hypothesized to be a 'junk' byproduct of RNA transcription, and thus did not receive much attention from researchers (24). circRNAs are linear precursor mRNAs reverse spliced in the nucleus to form closed RNA loops without 5'-3' polar or polyadenine tails, and range in length from 100s to 1,000s of bases (25,26). Due to their specific structure, circRNAs are protected from degradation by nucleic acid exonucleases, and have a longer half-life and stability than their parental mRNAs (27). Based on this conserved property, circRNAs may serve as promising biomarkers for the identification of malignancies (28). Unlike lncRNAs, circRNAs have an ORF in their sequence, thus circRNAs can be translated into proteins via internal ribosome entry site-driven or N6-adenylation-mediated initiation sites (29). In addition, circRNAs can bind to mRNAs to act as miRNA sponges or interact with RNA-binding proteins to directly regulate transcription (30).

miRNAs. miRNAs are small, single-stranded, conserved ncRNAs that are 18-25 nucleotides in length (31). miRNAs are transcribed into early miRNAs (pri-miRNAs) in cells, and pri-miRNAs are cleaved into precursor miRNAs (pre-miRNAs) in the nucleus, transported out of the nucleus, and then cleaved into small mature double-stranded RNAs in the cytoplasm, eventually forming the RNA-induced silencing complex (32-34). The majority of miRNAs direct mRNA inhibition or activation through degradation or translational repression. Thus, miRNAs can act as oncogenic factors or tumor suppressors (Fig. 1) (33,35). miRNAs regulate the expression of >1/3 of the encoded proteins *in vivo*, and are closely associated with cell proliferation, apoptosis and differentiation (33,35,36). In addition, miRNAs are involved in soft tissue construction, embryonic and organ development, and malignant transformation of disease in humans (36), with miR-10a and miR-10b being the most widely studied. For example, Yang *et al* (37) found that miR-10a and miR-10b can act as oncogenic factors in gastric cancer, and are also novel genetic markers for the diagnosis and treatment of gastric cancer.

Association between lncRNAs/circRNAs and miRNAs. The classical biological theory of miRNAs is that they bind to target 3'-UTRs in their target mRNAs by complementary base-pairing as negative regulators (38). miRNA response elements (MREs) are present in the majority of lncRNAs and circRNAs; MREs can compete with mRNAs to bind miRNAs to promote tumor malignancy, lymph node metastasis, tumor proliferation and tumor metastasis (Fig. 1) (39-41). This theory was developed in 2011 by Salmena *et al* (42) and is now supported by a large body of experimental data (43-48). Furthermore, this mode of regulation underlies the theory that networks of endogenous RNA molecules compete with each other.

3. miR-10 family

The miR-10 gene family is involved in the regulation of a number of malignant diseases (49). The miR-10 family has two primary members: miR-10a and miR-10b. miR-10a is located on human chromosome 17 and miR-10b is located on human chromosome 2. The nucleotide sequences of the two miRNAs are highly consistent, indicating that their biological functions may be similar (50). Members of the miR-10 family are involved in tumor proliferation (51), lymph node metastasis (52), direct tumor invasion (53), tumor cell apoptosis (25) and other malignant behaviors in cancer. In addition, the miR-10 family can serve as early diagnostic and prognostic markers for bladder cancer (BCa) (37), PCa (17), acute myeloid leukemia (AML) (54) and intraductal papillary mucinous neoplasm (55). The lncRNAs and circRNAs that target the miR-10 family in cancer are shown in Fig. 2. miR-10 family-related lncRNAs and circRNAs are involved in numerous cancer processes including epithelial-mesenchymal transition (EMT), the tumor immune response, drug resistance and sensitivity to radiotherapy (Table I). It can be hypothesized that the miR-10 family-associated lncRNAs and circRNAs may be novel molecular targets for the diagnosis, prognosis and treatment of oncological diseases.

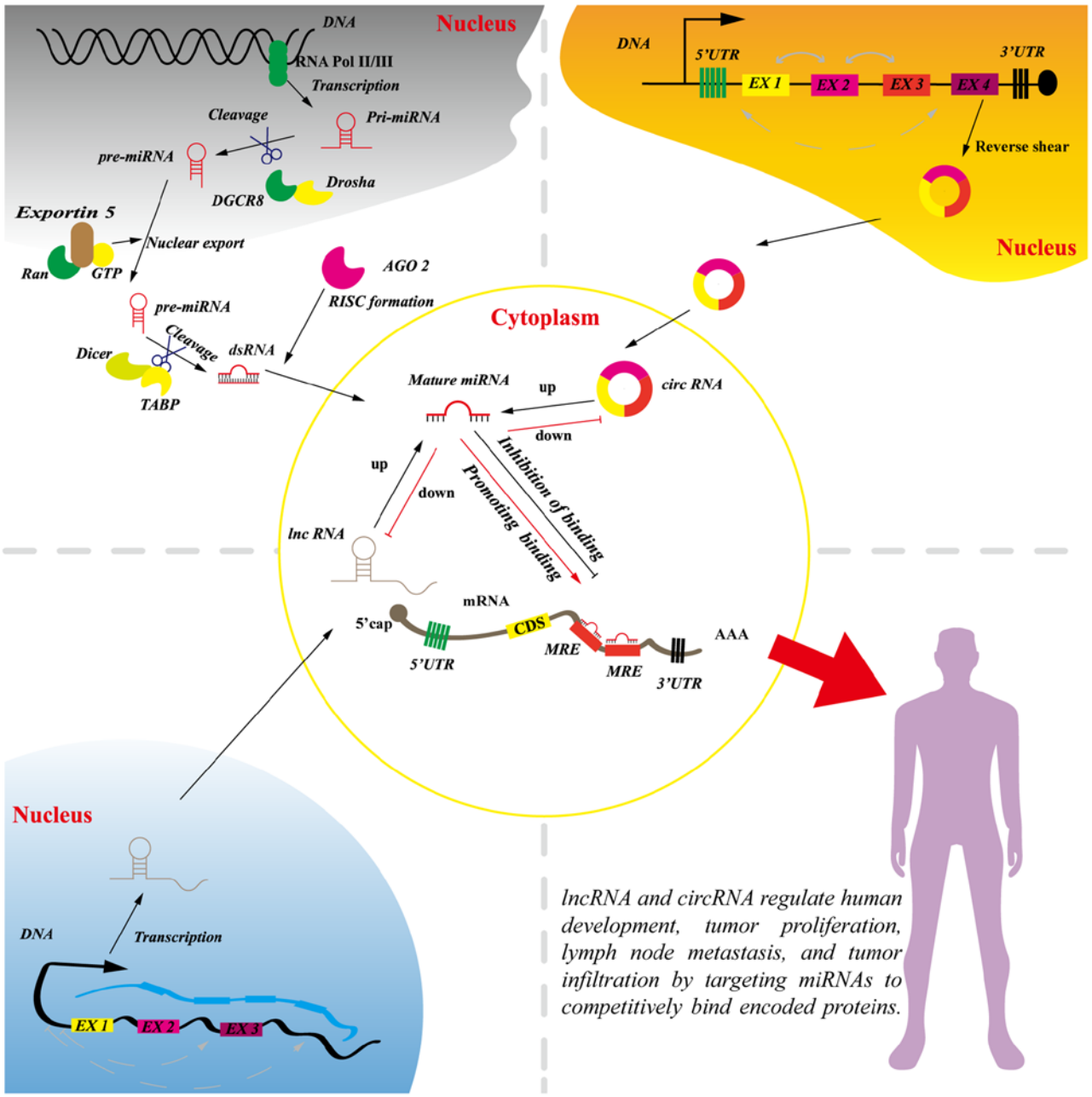

Figure 1. Mechanisms by which miRNAs regulate mRNAs. lncRNAs and circRNAs participate in cancer development and progression by competitively binding with miRNAs to regulate mRNAs. miRNA, microRNA; lncRNA, long non-coding RNA; circRNA, circular RNA.

4. lncRNAs and cancer

Lung cancer. The global mortality rate from lung cancer has remained high in recent years, with 2.2 million new cases worldwide in 2020, of which, ~1.79 million are predicted to die from the cancer (56). Early screening and surgery for lung cancer greatly improve post-operative cure and survival rates for patients (56,57). In addition, it has been shown that lncRNAs can act as ceRNAs targeting the miR-10 family members involved in lung cancer. For example, Zhu *et al* (58) detected high expression of the lncRNA LINRIS in non-small cell lung cancer (NSCLC) samples and cell lines, and patients with high LINRIS expression had significantly lower 5-year survival rates. Silencing lncRNA LINRIS significantly inhibited the viability of NSCLC cells, whereas overexpression of miR-10a reversed this phenomenon. Another study (59) found that lncRNA KAT7 expression was decreased in NSCLC tissues and tumor cells. The low expression of lncRNA KAT7 was associated with the survival of patients. Functionally, lncRNA KAT7 promoted the methylation of miR-10a. Furthermore, *in vitro* experiments showed that overexpression of miR-10a increased the proliferation of NSCLC cells. Conversely, over-expression of lncRNA KAT7 inhibited the effect of miR-10a on NSCLC proliferation (59). These findings suggested that lncRNAs targeting the miR-10 family may act as key genes in the progression of NSCLC.

Esophageal cancer (ESCA). It is estimated that 17,650 individuals were diagnosed with ESCA in the United States in 2019, and ~16,080 of them died from the disease (60).

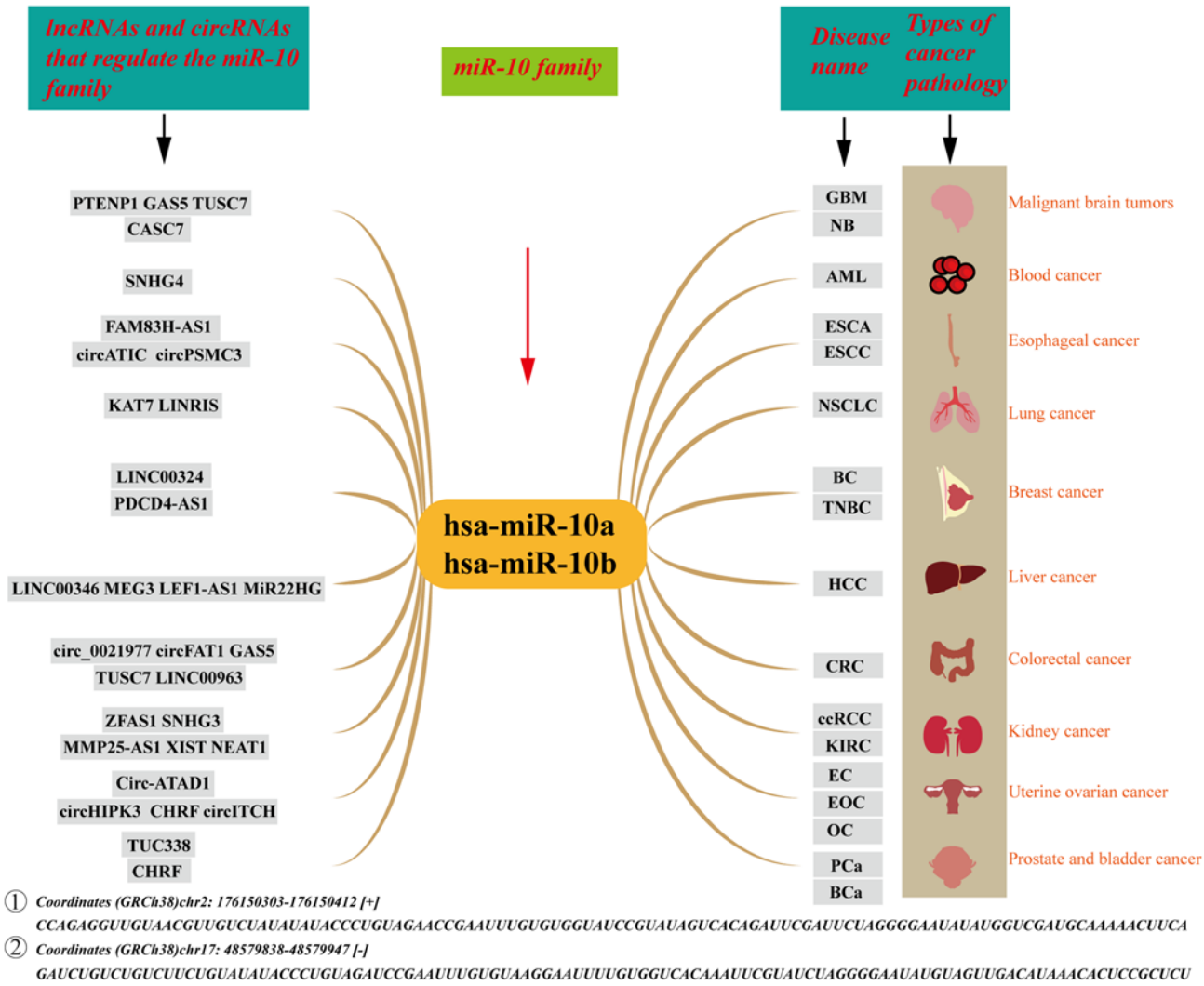

Figure 2. lncRNAs and circRNAs associated with the miR-10 family in cancer. mRNAs, lncRNAs and circRNAs can regulate gene expression by competitively binding with miRNAs, forming a ceRNA regulatory network with miRNAs. The present review found that miR-10a and miR-10b competitively combined with various lncRNAs and circRNAs to regulate various biological functions of cancer. Sequence 1 is the position and base sequence of hsa-miR-10a on the chromosome; sequence 2 is the position and base sequence of hsa-miR-10b on the chromosome. miR, microRNA; lncRNA, long non-coding RNA; circRNA, circular RNA.

Although early screening and prevention efforts have helped to improve the survival of patients with ESCA, the 5-year overall survival (OS) rate of patients with advanced ESCA remains <20% (60,61). The high mortality and low survival rates of ESCA are currently a major challenge for clinical practitioners and exploring novel biomarkers for prognostic analysis is an important topic in ESCA research (62). Feng *et al* (63) detected a trend toward decreased expression of the lncRNA FAM83H-AS1 in ESCC tissues and cells. lncRNA FAM83H-AS1 was negatively associated with TNM stage, lymph node metastasis and pathological stage. Mechanistically, lncRNA FAM83H-AS1 acted as a sponge for miR-10a-5p to activateCCDC88A. Functionally, the knockdown of lncRNA FAM83H-AS1 significantly inhibited the ability of Kyse150 and TE1 ESCA cells to proliferate, migrate and invade. By contrast, miR-10a-5p inhibitors partially rescued the inhibitory effects of lncRNA FAM83H-AS1 knockdown on cell proliferation, migration and invasion. Downregulated lncRNA FAM83H-AS1 promoted E-cadherin expression in tumor growth factor-β-induced Eca109 ESCA cells, and inhibited N-cadherin, vimentin, Snail and Twist1 expression at the transcriptional level, whereas overexpression of lncRNA FAM83H-AS1 had the opposite effect (63). These results suggested that lncRNA FAM83H-AS1 can mediate EMT, and serve as a novel diagnostic marker and therapeutic target for ESCA.

Liver cancer. Liver cancer continues to be a significant burden worldwide, with incidence rates increasing globally. It is estimated that one million individuals will develop liver cancer in 2025 (64). Hepatocellular carcinoma (HCC) accounts for 90% of all primary liver cancer cases (65). Patients with HCC often do not show early signs of clinical presentation, as such, patients are often diagnosed in the first instance with advanced-stage liver cancer, and thus have a greater risk of tumor recurrence and metastasis (64,66). lncRNA MEG3 expression has been revealed to be downregulated in HCC tissues and cells, whereas miR-10a-5p levels were shown to be upregulated. Overexpression of lncRNA MEG3 directly inhibited Hep2 HCC cell proliferation, migration and invasion,

Table I. Functions of lncRNAs/circRNAs targeting the miR-10 family in cancer.

A, miR-10a

First author, year	Upstream lncRNA/circRNA	Downstream mRNA	Tumor type	Function	Mechanism	(Refs.)
Liu, 2021	circFAT1	-	CRC	Proliferation Invasion, Migration	Overexpression of circFAT1 or knockdown of miR-10a inhibited cell proliferation, migration and invasion	(112)
Ren *et al*, 2017	TUSC7	-	CRC	Proliferation, Invasion	Overexpression of lncRNA TUSC7 inhibited cell proliferation and invasion of CRC cells, and overexpression of miR-10a reversed this effect	(73)
Zhu *et al*, 2022	LINRIS	-	NSCLC	Proliferation	Silencing of lncRNA LINRIS significantly inhibited cell viability	(58)
Zhou *et al*, 2020	CASC7	PTEN	NB	Proliferation	Abnormal increase in lncRNA CASC7 led to decreased cell proliferation via the miR-10a/PTEN axis	(99)
Yuan *et al*, 2020	SNHG4	PTEN	AML	Proliferation	lncRNA SNHG4 and miR-10a competitively bound to PTEN, inhibiting cell viability	(106)
Gao *et al*, 2021	KAT7	-	NSCLC	Proliferation	Overexpression of lncRNA KAT7 inhibited the effect of miR-10a on proliferation of NSCLC	(59)
Shang *et al*, 2018	TUSC7	-	GBM	Drug resistance	lncRNA TUSC7 overexpression in GBM cells inhibited TMZ resistance	(104)
Yang *et al*, 2021	Circ-ATAD1	-	EC	Invasion, Migration	Circ-ATAD1 attenuated miR-10a methylation to inhibit EC cell invasion and migration	(117)
Luo *et al*, 2018	circ-ITCH	-	EOC	Proliferation, Apoptosis	Transfection of circ-ITCH inhibited SKOV3 cell proliferation and enhanced apoptosis	(113)
Dong *et al*, 2019	ZFAS1	SKA1	ccRCC	Proliferation, Migration	Knockdown of lncRNA ZFAS1 or mRNA SKA1 effectively attenuated the ability of ccRCC cells to proliferate, migrate and invade	(78)

B, miR-10a-5p

First author, year	Upstream lncRNA/circRNA	Downstream mRNA	Tumor type	Function	Mechanism	(Refs.)
Wu *et al*, 2019	miR-22HG	NCOR2	HCC	Proliferation Migration, Invasion	lncRNA MIR22HG regulated the proliferation, migration and invasion of HCC cells by competitively binding to the coding protein NCOR2 and miR-10a-5p	(68)
Hao *et al*, 2019	PTENP1	PTEN	GBM	Proliferation Apoptosis	Co-culture of hUC-MSC-derived exosome lncRNA PTENP1 and U87 inhibited the proliferation of U87 cells and promoted cell apoptosis	(102)

Table I. Continued.

B, miR-10a-5p

First author, year	Upstream lncRNA/circRNA	Downstream mRNA	Tumor type	Function	Mechanism	(Refs.)
Zhang *et al*, 2019	MEG3	PTEN	HCC	Proliferation Apoptosis Migration Invasion	Overexpression of lncRNA MEG3 inhibited the proliferation, migration and invasion of HCC cells, and promoted apoptosis	(67)
Feng *et al*, 2020	FAM83H-AS1	CCDC88A	ESCC	EMT	Downregulation of lncRNA FAM83H-AS1 promoted TGF-β-induced EMT activation in esophageal cancer	(63)
Liu *et al*, 2022	NEAT1	SERPINE1	KIRC	Tumor immunity Proliferation Migration	lncRNA NEAT1 promoted cell proliferation and migration by activating SERPINE1 through miR-10a-5p. SERPINE1 expression was positively correlated with immune infiltration	(82)
Teng *et al*, 2019	circHIPK3	-	EOC	Proliferation, Migration, Invasion	Knockdown of circHIPK3 promoted cell proliferation, migration and invasion, and inhibited cell apoptosis	(114)
Tan *et al*, 2021	MMP-25-AS1	SERPINE1	KIRC	Tumor immunity	The lncRNA MMP25-AS1/hsa-miR-10a-5p/SERPINE1 axis was involved in tumor immunity	(83)
Liu *et al*, 2021	XIST	SERPINE1	KIRC	Tumor immunity	lncRNA XIST regulated SERPINE1 targeting of miR-10a-5p and regulated the levels of immune-related cells	(81)
Li *et al*, 2019	LINC00346	CDK1	HCC	-	The LINC00346-miR-10a-5p-CDK1/CCNE1 axis may be associated with the relapse-free survival time of HCC	(66)
Gao *et al*, 2021	LEF1-AS1	MSI1	HCC	Proliferation, Apoptosis Drug resistance	Knockout of lncRNA LEF1-AS1 inhibited proliferation of cisplatin-resistant cells, promoted apoptosis and increased sensitivity	(69)
Zhu *et al*, 2021	circPSMC3	PTEN	ESCA	Proliferation Apoptosis	Overexpression of circPSMC3 or knockdown of miR-10a-5p reduced the cell survival rate of GR cells and induced cell apoptosis	(70)

C, miR-10b

First author, year	Upstream lncRNA/circRNA	Downstream mRNA	Tumor type	Function	Mechanism	(Refs.)
Liu *et al*, 2019	CHRF	GSK3β	PCa	Proliferation EMT	lncRNA CHRF promoted GSK3β through miR-10b. lncRNA CHRF overexpression enhanced TGF-β1-induced EMT in PC3 cells	(95)
Yang *et al*, 2022	GAS5	-	CRC	Proliferation Migration	lncRNA GAS5 overexpression inhibited tumor growth rate and tumor weight in mice	(74)
Wu *et al*, 2021	LINC00963	FGF13	CRC	Migration Invasion EMT	LINC00963 can act as a sponge of miR-10b to inhibit the activation of FGFR13 Knockout of LINC00963 inhibited the EMT process	(75)
Ding *et al*, 2020	GAS5	Sirt1	GBM	Proliferation Apoptosis Migration Invasion	lncRNA GAS5 acted synergistically with miR-10b to downregulate Sirt1 expression, inhibit cell growth, migration and invasion, and promote apoptosis	(103)

Table I. Continued.

C, miR-10b

First author, year	Upstream lncRNA/circRNA	Downstream mRNA	Tumor type	Function	Mechanism	(Refs.)
Li *et al*, 2019	TUC338	-	BCa	Migration Invasion	Overexpression of lncRNA TUC338 significantly promoted cell invasion and migration	(97)
Tan *et al*, 2020	CHRF	STAT3	OC	Proliferation	Downregulation of lncRNA CHRF significantly reduced the growth of transplanted tumors in mice	(92)

D, miR-10b-3p

First author, year	Upstream lncRNA/circRNA	Downstream mRNA	Tumor type	Function	Mechanism	(Refs.)
Zhang *et al*, 2022	circATIC	RHCG	ESCA	Proliferation Migration Invasion	Overexpression of circATIC or RHCG inhibited the proliferation, migration and invasion of ESCA cells	(110)

E, miR-10b-5p

First author, year	Upstream lncRNA/circRNA	Downstream mRNA	Tumor type	Function	Mechanism	(Refs.)
Xu *et al*, 2021	SNHG3	BIRC5	ccRCC	Proliferation Migration Invasion	lncRNA SNHG3 activated the expression of BIRC5 by binding to miR-10b-5p, and promoted the proliferation, invasion and migration of ccRCC cells	(79)
Wang *et al*, 2020	LINC00324	E-cadherin	BC	Proliferation Migration Apoptosis EMT	LINC00324 inhibited EMT markers through miR-10b-5p, inhibited cell viability and migration, and promoted apoptosis	(89)
Lu *et al*, 2020	circ_0021977	P21/P53	CRC	Proliferation Migration Invasion	circ_0021977 competitively bound to miR-10b-5p to regulate p21 and p53, and inhibited the proliferation, migration and invasion of CRC cells	(111)
Wang *et al*, 2021	PDCD4-AS1	IQGAP2	TNBC	Proliferation Migration Invasion	Overexpression of lncRNA PDCD4-AS1 decreased cell proliferation, migration and invasion of breast cancer cells, and increased the cell apoptotic rate	(90)

CRC, colorectal cancer; NSCLC, non-small-cell lung cancer; NB, neuroblastoma; AML, acute myelocytic leukemia; GBM, glioblastoma multiforme; EC, endometrial cancer; EOC, epithelial ovarian cancer; ccRCC, clear cell renal cell carcinoma; HCC, hepatocellular carcinoma; ESCC, esophageal squamous cell; KIRC, kidney renal clear cell carcinoma; ESCA, esophageal carcinoma; PCa, prostate cancer; BCa, breast cancer; BC, bladder cancer; TNBC, triple-negative breast cancer; OC, ovarian cancer.

and increased cell cycle progression and apoptosis of Hep2 cells through inhibition of miR-10a-5p, activating PTEN via the AKT signaling pathway. In addition, it has been shown that PTEN is a miR-10a-5p target gene (67). Low expression of lncRNA miR-22HG was detected in HCC tissues and cells. lncRNA miR-22HG regulates the proliferation, migration and invasion of HCC cells by competitively binding to the protein NCOR2 and miR-10a-5p. In addition, lncRNA miR-22HG was significantly associated with patient OS and disease-free survival (68). Gao *et al* (69) detected abnormally elevated expression levels of lncRNA LEF1-AS1 in HCC clinical samples and HCC cancer cell lines. Mechanistically, overexpressed lncRNA LEF1-AS1 regulated activation of the AKT signaling pathway by MSI1 through miR-10a-5p. Functionally, knockdown of lncRNA LEF1-AS1 and MSI1 or overexpression of miR-10a-5p inhibited the proliferation of cisplatin (DDP)-resistant Huh7 HCC cells, promoted apoptosis and enhanced chemosensitivity of Huh7 cells to DDP (69). This may provide a novel direction for research on HCC drug resistance. In addition, to screen for lncRNAs that can serve as early markers of HCC, Li *et al* (66) constructed a ceRNA regulatory network using bioinformatics tools. Bioinformatics analysis identified the LINC00346/miR-10a-5p/CDK1/CCNE1 axis as having a possible association with HCC. Kaplan-Meier analysis showed that CDK1 and CCNE1 were associated with relapse-free survival time in HCC. Thus, lncRNAs targeting the miR-10 family may have significant potential as diagnostic/prognostic markers and therapeutic targets for the management of HCC.

Colorectal cancer (CRC). CRC is a global health problem, accounting for 10% of all cancer diagnoses and deaths each year (70). The number of CRC cases worldwide is expected to increase to 2.5 million by 2035 (71). Early tumor screening has reduced the morbidity and postoperative mortality rates of patients with CRC, but ~25% of patients with CRC are diagnosed with advanced-stage cancer in the first instance, and 25-50% of patients develop early metastases (72). A commitment to the development of diagnostic and prognostic markers for CRC is essential. Ren *et al* (73) found that lncRNA TUSC7 expression was suppressed in CRC cells and clinical cancer samples, and that low expression of lncRNA TUSC7 was associated with patient OS. In addition, lncRNA TUSC7 expression was lower in specimens classified as stage C/D than in those classified as stage A/B according to the Duke's classification system for colon cancer. Functionally, overexpression of lncRNA TUSC7 inhibited cell proliferation and invasion of SW480 and HT29 CRC cells, whereas overexpression of miR-10a reversed the effects of overexpression of lncRNA TUSC7 on CRC cells. In another study, significant downregulation of lncRNA GAS5 was detected in CRC samples and cells. Mechanistically, overexpression of lncRNA GAS5 inhibited the migration and proliferation of CRC cells through direct interaction with miR-10b. Animal model experiments showed that overexpression of lncRNA GAS5 resulted in significantly lower tumor-forming growth rates and tumor weights *in vivo*, in contrast to miR-10b overexpression, which restored tumor growth rates and increased tumor weight (74). Wu *et al* (75) found that LINC00963 expression was upregulated in CRC samples and CRC cells, and that it could inhibit CRC cell viability, colony formation, migration and invasion through a LINC00963/miR-10b/FGF13 axis. Knockdown of LINC00963 resulted in the downregulation of waveform protein and N-cadherin expression levels, and increased expression of E-cadherin. These findings suggested that LINC00963 may be involved in EMT in colon cancer (75). Thus, miR-10 family-related lncRNAs may serve as key factors in the progression of CRC.

Kidney cancer. Renal cell carcinoma (RCC) is the most common and deadly urological malignancy, accounting for 2.2% of all diagnosed cancers worldwide, of which 70-80% are clear cell RCC (ccRCC) (76,77). A study of ccRCC found that lncRNA ZFAS1 expression was upregulated in ccRCC samples and cell lines, and was strongly associated with tumor size, lymph node metastasis and patient OS. Knockdown of lncRNA ZFAS1 or mRNA SKA1 has been shown to effectively attenuate the ability of ACHN and Caki-1 ccRCC cells to proliferate, migrate and invade, whereas transfection with miR-10a inhibitors attenuated the effect of knockdown of lncRNA ZFAS1 or SKA1 on ccRCC cells (78). Furthermore, bioinformatics analysis showed that lncRNA SNHG3 was associated with a poorer prognosis in patients with ccRCC. lncRNA SNHG3 promoted the proliferation, invasion and migration of ccRCC cells by activating BIRC5 expression through binding to miR-10b-5p (79). In addition, it has been shown that lncRNAs are involved in the immune process of the tumor microenvironment (TME) (80). For example, Liu *et al* (81) showed that a lncRNA XIST/miR-10a-5p/SERPINE1 axis mediated the TME of ccRCC. Immune cell infiltration analysis revealed that the lncRNA XIST can regulate the levels of $CD4^+$ T cells, $CD8^+$ T cells, macrophages, dendritic cells, neutrophils and other immune-related cells through the regulation of miR-10a-5p targeting SERPINE1 expression (81). Another study showed that lncRNA NEAT1 similarly promoted the proliferation and migration of 786-O and ACHN kidney renal clear cell carcinoma (KIRC) cells by acting as a sponge of miR-10a-5p, thus indirectly activating SERPINE1. The area under the curve (AUC) of the receiver operating characteristic curve analysis indicated that SERPINE1 (AUC=0.789) and miR-10a-5p (AUC=0.892) had good prognostic performance. Furthermore, SERPINE1 expression was positively associated with the levels of immune infiltration of $CD4^+$ T cells, $CD8^+$ T cells, macrophages, dendritic cells and neutrophils (82). In another study, a ceRNA regulatory network consisting of the lncRNA MMP25-AS1/hsa-miR-10a-5p/SERPINE1 axis was identified. lncRNA MMP25-AS1 expression was significantly associated with sex, pathological stage, T-stage and M-stage. Immune infiltration analysis showed that the lncRNA MMP25-AS1/hsa-miR-10a-5p/SERPINE1 axis could affect the chemokines CCL4, CCL5, CXCL13 and XCL2. High SERPINE1 expression was also shown to be associated with tumor immune evasion in KIRC (83). Thus, the miR-10 family may be a core miRNA in the immune-related processes of kidney cancer, and the associated lncRNAs may serve as biomarkers/prognostic factors in kidney cancer, and could also participate in the regulation of the TME.

Breast cancer (BC). BC is one of the most common malignancies among women worldwide (84). More than 268,000 cases

are expected to be diagnosed each year in the United States, accounting for approximately one-third of all new cancer cases in women and 15% of all cancer-related deaths (85). Despite the progress made in the study of BC, it remains a major global health problem (86). Under normal conditions, EMT plays a key role in embryonic development, wound repair and tissue remodeling; however, aberrant EMT leads to lymph node metastasis and infiltration of cancerous tissue in BC (87,88). Low expression of LINC00324 in BC cells has been reported to be associated with a poorer patient prognosis and reduced OS. Functionally, LINC00324 inhibited cell viability, suppressed cell migration and promoted apoptosis in MCF-7 BC cells by directly interacting with miR-10b-5p through inhibition of the EMT marker E-cadherin. In addition, animal models have shown that LINC00324 overexpression limits the size of xenograft tumors (89). lncRNA PDCD4-AS1 expression was shown to be lower in clinical triple-negative BC (TNBC) samples and cells. Overexpression of the lncRNA PDCD4-AS1 attenuated the proliferation, migration and invasion of TNBC cells, and increased apoptosis. Coincidentally, IQGAP2 overexpression had the same effect as overexpression of lncRNA PDCD4-AS1, whereas miR-10b-5p downregulation reversed this result (90). These results suggested that miR-10 family-related lncRNAs may regulate cancer metastasis in BC by participating in the EMT process, and could also serve as novel therapeutic targets or diagnostic markers for BC.

Ovarian cancer (OC). OC accounts for 4% of new cancer cases and 5% of cancer deaths in women in the United States (91). DDP is the chemotherapy of choice for OC, and the primary reason for the ineffectiveness of chemotherapy is the development of resistance of tumor cells to DDP (91,92). lncRNA CHRF was found to be upregulated in DDP-resistant ES2 cells and in OC samples. In addition, lncRNA CHRF expression was significantly higher in patients with OC and liver metastases compared with samples from OC patients without liver metastases. Mechanistically, lncRNA CHRF regulated miR-10b and significantly increased the resistance of DDP-resistant cells to DDP via the EMT and STAT3 signaling pathways. Functionally, xenograft experiments showed that inoculation of DDP-resistant cells with lower levels of lncRNA CHRF expression resulted in a significant reduction in transplanted tumor growth in mice *in vivo*, an effect that could be attenuated by ectopic expression of miR-10b in xenograft tumors (92). These findings suggested that lncRNA CHRF and miR-10b may be molecular targets for reducing DDP resistance in OC.

PCa. PCa is the second most common type of cancer affecting men worldwide and the sixth leading cause of cancer-associated death in men (93). PCa is a heterogeneous disease that progresses slowly in the early stages of development, but is highly aggressive in the later stages (94). Therefore, it is particularly important to explore its pathogenesis. Studies have found that lncRNA CHRF participates in the proliferation of PCa cells by promoting activation of the GSK3β/AKT and NF-κB signaling cascades through miR-10b. In addition, overexpression of lncRNA CHRF can enhance TGF-β1-induced changes in the expression of EMT-related proteins in PC3 cells, whereas miR-10b overexpression reversed these changes (95). These findings suggested that CHRF targeting of miR-10b may affect tumor progression by regulating EMT in PCa.

BCa. Approximately 440,000 new cases of BCa are diagnosed each year, resulting in >130,000 deaths worldwide. The primary reasons for the low survival rates in patients with early and advanced BCa following surgery are the lack of early specific diagnostic markers and the limited availability of treatments for late-stage BCa (96). It has been shown that lncRNAs can be used to distinguish between patients with early-stage BCa malignancy and healthy patients. For example, Li *et al* (97) found that lncRNA TUC338 was abnormally elevated in the plasma of patients with early-stage BCa. Plasma lncRNA TUC338 levels were significantly downregulated after surgical resection, with an AUC value of 0.9239, indicating that lncRNA TUC338 was specific and sensitive for the diagnosis of BCa. Functional overexpression of lncRNA TUC338 and miR-10b have both been shown to significantly promote cell invasion and cell migration in HT-1197 and HT-1376 BCa cells. Thus, TUC338 targeting of miR-10b may be a novel molecular diagnostic marker for the detection of early-stage BCa.

Neuroblastoma (NB). NB is an embryonic malignant extracranial solid tumor that is relatively common in children and often has a poor prognosis (98). lncRNAs targeting miR-10a are involved in the progression of NB. For example, Zhou *et al* (99) detected abnormally reduced levels of lncRNA CASC7 epitopes by fluorescent quantitative (q)PCR in NB tissues and cells. lncRNA CASC7 resulted in reduced proliferation of NB cells, whereas overexpression of miR-10a activated PTEN to promote NB cell proliferation.

Glioblastoma multiforme (GBM). GBM is the most common primary malignant glioma, accounting for 48% of all malignant central nervous malignancies worldwide (100). Molecularly targeted therapy, radiotherapy, primary resection and postoperative supportive care can be used to treat GBM to some extent, but the postoperative and prognostic outcomes for patients remain limited (100,101). Therefore, there is an urgent need to investigate the pathogenesis of GBM and to explore potential therapeutic targets. In GBM, Hao *et al* (102) showed that lncRNA PTENP1 was reduced in tumor samples and miR-10a-5p expression was significantly increased. The expression of lncRNA PTENP1 was increased in U87 cells treated with human umbilical cord-mesenchymal stem cell (hUC-MSC)-derived exosomes. In addition, hUC-MSC-derived exosomes and U87 co-culture inhibited the proliferation of U87 cells and promoted cell apoptosis. Overexpression of lncRNA PTENP1 or inhibition of miR-10a-p reduced cell proliferation and promoted cell death in U87 cells not treated with hUC-MSCs. Luciferase experiments showed that when U87 cells were co-transfected with miR-10a-5p mimics and lncRNA PTENP1-WT or PTEN-WT plasmids, the luciferase activity was decreased. Thus, a lncRNA PTENP1/miR-10a-5p/PTEN axis may have potential clinical application in the management of glioma. Ding *et al* (103) found that overexpression or knockdown of lncRNA GAS5 in glioma cells altered the proliferation, migration, invasion and apoptosis of glioma cells. It was suggested that lncRNA GAS5 acts synergistically with miR-10b to downregulate

Sirt1 expression, inhibit PI3K/AKT and MEK/ERK signaling pathways, and to suppress the proliferation, migration and invasion, and promote the apoptosis of U251 and A172 GBM cells. In addition, it has been shown that miR-10a-related lncRNAs can increase chemosensitivity in GBM. For example, low expression levels of lncRNA TUSC7 were detected in GBM cells and temozolomide (TMZ)-resistant tissues. Furthermore, expression of lncRNA TUSC7 was lower in samples from TMZ-insensitive patients compared with that in TMZ-sensitive patients. Notably, overexpression of lncRNA TUSC7 suppressed TMZ resistance and expression of multidrug resistance protein 1 (MDR1) in U87TR GBM cells (104). Conversely, overexpression of miR-10a increased the expression of MDR1, increased the half-inhibitory concentration of TMZ and inhibited TMZ-induced cytotoxicity in U87TR cells (104). These data suggested that lncRNA TUSC7 may inhibit the chemosensitivity of U87TR cells to TMZ by targeting MDR1 through interaction with miR-10a (104).

AML. AML is a malignant cancer of the blood and bone marrow. Its incidence increases with age, and the 5-year OS rate in younger patients with *de novo* AML is 40-50% worldwide (105,106). Yuan and Wang (106) found reduced levels of lncRNA SNHG4 expression in The Cancer Genome Atlas AML dataset and AML tissues. Overexpression of lncRNA SNHG4 partially abolished the enhancing effect of upregulated miR-10a on AML cell proliferation, the same way overexpression of PTEN similarly attenuated the effect of miR-10a on the proliferation rate of AML cells (106). Taken together, lncRNA SNHG4 may serve as an oncogenic factor in AML.

5. circRNAs and tumors

Previous studies have demonstrated the role and regulatory mechanisms of miR-10 family-related lncRNAs in numerous types of cancer. These studies suggested that miR-10-related lncRNAs may be used as early and specific diagnostic and prognostic markers for cancer. In addition, they can be used as novel therapeutic targets for the management of cancer and for modulation of sensitivity to chemotherapy. Notably, in addition to miR-10-related lncRNAs, miR-10-related circRNAs are also important in cancer development and progression.

ESCA. Gefitinib (GR) is the most common and effective chemotherapeutic agent for the management of ESCA (107). It is estimated that 60-80% of patients with advanced ESCA have elevated EGFR expression levels in the cancerous tissue (107-109), and GR can inhibit EGFR expression and thus improve the prognosis of patients with ESCA. However, GR resistance in patients with advanced ESCA has become a major limiting factor affecting long-term patient prognosis, and the mechanisms of resistance are currently unclear. Therefore, exploring the molecular mechanisms of resistance to GR in ESCA is of significant importance for the clinical management of patients with ESCA (107,109). One study found that circPSMC3 was downregulated in ESCC tissues and gefitinib-resistant (GR) cells, while miR-10a-5p was upregulated. Overexpression of circPSMC3 or knockdown of miR-10a-p decreased the survival rate of GR ESCC cells (TE1/GR and KYSE450/GR) and induced apoptosis, while downregulation of PTEN reversed this effect (70). Luciferase assays were used to confirm that circPSMC3-WT activity could be inhibited by miR-10a-5p mimics, whereas circPSMC3-MUT was unaffected. Thus, circPSMC3 could overcome the chemosensitivity resistance to GR by sponging miR-10a-5p to promote downstream PTEN activation (70). miR-10-related circRNAs can regulate ESCA development and modulate radiotherapy sensitivity, in addition to increasing ESCA chemosensitivity to GR. For example, Zhang *et al* (110) detected reduced circATIC expression in ESCA tissues. circATIC expression was reduced in high TNM stage cancer and in patients with positive lymph node metastasis compared with that in low TNM stage cancer and in patients without lymph node metastasis group. Functionally, overexpression of circATIC or RHCG inhibited proliferation, migration and invasion of EC109 and KYSE150 ESCA cells, and promoted apoptosis of EC109 and KYSE150 cells under radiation exposure, whereas this was reversed by upregulation of miR-10b-3p. Furthermore, *in vivo* animal studies showed that circATIC overexpression promoted the inhibitory effect of radiation on tumor volume and weight. Taken together, targeting the miR-10 family of circRNAs may improve radiotherapy sensitivity in ESCA.

CRC. The value of lncRNA research in CRC has been demonstrated in previous studies. In addition, circRNAs in the serum can be used as a diagnostic marker for CRC. For example, circ_002197 expression has been reported to be reduced in clinical samples and cell lines from patients with colon cancer. Most notably, its expression was significantly lower in the preoperative blood of patients with CRC and lymph node metastases compared with that in patients without lymph node metastases. The AUC of circ_0021977 between plasma from patients with CRC and normal plasma was 0.873, the sensitivity was 85.71%, and the specificity was 77.78%. These findings suggested that the circ_0021977 diagnostic model had high specificity. In addition, low expression of circ_0021977 was significantly associated with a poor prognosis. *In vitro* and *in vivo* experiments showed that circ_0021977 competitively bound to miR-10b-5p to regulate p21 and p53 to inhibit proliferation, migration and invasion of CRC cells (111). This highlights its value for early diagnosis and treatment of patients with CRC. Similarly, Liu (112) found that circFAT1 expression was low in CRC tissues and cells, and its expression was negatively associated with miR-10a. Functional studies have shown that circFAT1 overexpression or miR-10a knockdown can inhibit the proliferation, invasion and migration of CRC cells. Thus, circRNAs targeting the miR-10 family may serve as novel diagnostic markers and therapeutic targets for the management of CRC.

OC. Luo *et al* (113) detected low expression levels of circ-ITCH in human OC epithelial cell lines. It was revealed that transfection of SKOV3 cells with circ-ITCH mimics inhibited proliferation and enhanced apoptosis, whereas knockdown of circ-ITCH promoted cell proliferation and inhibited apoptosis. Reverse transcription-qPCR detected a decrease in miR-10a expression levels following overexpression of circ-ITCH. In addition, rescue assays showed that overexpression of circ-ITCH could rescue SKOV3 cell proliferation and apoptosis caused by the overexpression of miR-10a (113). Another

study identified the presence of a circHIPK3/miR-10a-5p axis using bioinformatics tools. *In vitro* experiments revealed that the knockdown of circHIPK3 promoted proliferation, migration and invasion of epithelial ovarian cancer cells and inhibited apoptosis of A2780 and SKOV3 OC cells (114). These results suggested that circRNAs targeting miR-10a may have a tumor-suppressive role in OC.

Endometrial cancer (EC). EC is the second most common female malignancy after BC worldwide, and is the most common type of cancer in developing countries (115). In the United States and Europe, EC is the sixth and eighth leading cause of cancer-associated death in women, respectively (116). There is increasing evidence that circRNAs may be oncogenic factors in EC. Yang *et al* (117) showed that circ-ATAD1 expression was downregulated in EC, whereas miR-10a expression was upregulated in EC. Mechanistically, circ-ATAD1 attenuated miR-10a methylation, which resulted in the inhibition of EC cell invasion and migration.

6. Conclusion and future prospects

In the context of cancer, lncRNAs and circRNAs primarily function by sponging miRNAs. We found that lncRNAs and circRNAs regulate the malignant progression of diseases, including lung cancer, ESCA, liver cancer, CRC, kidney cancer, PCa, BCa, BC, OC, GBM, NB, AML and EC by activating or inhibiting miR-10a or miR-10b. The present review described the relevant mechanisms by which lncRNAs and circRNAs target the miR-10 family to regulate cancer. It was revealed that the expression levels of lncRNAs/circRNAs targeting the miR-10 family members are related to tumor size, local invasion, tumor metastasis and TME. They are also related to clinical factors, such as TNM stage, lymph node metastasis and pathological type. For example, the lncRNAs CHRF, FAM83H-AS1, LINC00963 and LINC00324 also participate in EMT in cancer by acting as a sponge of the miR-10 family members and affecting the metastatic ability of cancer cells. Additionally, lncRNAs and circRNAs regulate cancer progression through the modulation of various signaling pathways. For example, the lncRNAs MEG3, CHRF and GAS5 are involved in the regulation of PIK/AKT, MEK/ERK, GSK3β/AKT and NF-κB signaling pathways by targeting the miR-10 family. In addition, by evaluating the expression levels of miR-10a and miR-10b-related lncRNAs/circRNAs, it has been shown that lncRNAs and circRNAs can be used as diagnostic markers for several types of cancer. For example, circ_002197 and lncRNA TUC338 targeting miR-10 family members could be used to distinguish patients with malignant cancer from healthy individuals based on their expression in plasma, and could also be used to predict the prognosis of patients with cancer.

The present review highlights the role of miR-10-related lncRNAs/circRNAs in the early diagnosis and prognosis of cancer. For the treatment of malignant tumors, a combination of chemotherapy and radiotherapy is the standard mode of treatment for advanced malignancies (110,111); however, its effectiveness is limited due to intrinsic and acquired resistance (118). In addition, sensitivity to radiotherapy is key to the efficacy of treatment of malignant tumors, and as some tumors are not sensitive to radiotherapy, a proportion of patients will exhibit a poor prognosis, recurrence, tumor metastasis and/or resistance (119). circPSMC3, circATIC, lncRNAs TUSC7 and LEF1-AS1 can act as ceRNAs to regulate the miR-10 family members to reduce resistance to chemotherapy or improve sensitivity to radiotherapy in malignant tumors. Immunotherapy may be a treatment of last resort for advanced malignancies that are resistant to radiotherapy. In contrast to radiotherapy, immunotherapy kills tumor cells by facilitating the innate immune response through modulation of the TME (120,121). Coincidentally, the present study found that the lncRNAs XIST, NEAT1 and MMP25-AS1 are involved in regulating tumor immunity and escape in renal cancer by targeting the miR-10a-5p/SERPINE1 axis.

Although the roles of miR-10 family-related lncRNAs in cancer have been extensively studied, to the best of our knowledge, the mechanisms of miR-10 family-related lncRNAs in cancer, such as thyroid cancer, oral cancer, pancreatic cancer, bone tumors and vaginal cancer, have not been reported. In addition, only a few miR-10-related circRNAs have been reported so far in malignant tumors, due to a lack of studies targeting miR-10 family circRNAs. In addition, although researchers have found, through bioinformatics, that the lncRNA-targeted miR-10 family may be involved in renal tumor immunity, the specific regulatory mechanisms are not yet fully understood (70-72), and prediction by bioinformatics analysis alone is not rigorous; therefore, the regulatory mechanisms should be elaborated through cellular experiments or animal models. In conclusion, future research should continue to expand the lncRNAs and circRNAs targeting the miR-10 family and explore the mechanisms of their action in malignancies. Of note, targeted drugs and biomarkers based on miR-10 family-related lncRNAs and circRNAs may be the next direction in the translation of this field of research from the bench to bedside. In summary, targeting the miR-10 family of lncRNAs and circRNAs may be a new strategy for treating cancer.

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SG and WW contributed to the literature search and selected the studies for inclusion. SG, SL and WW drafted the manuscript and revised it critically for important intellectual content. FM and YQ conceived the topic of the present review and revised the manuscript. All authors read and approved the final version of the manuscript. Data authentication is not applicable.

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Competing interests

The authors declare that they have no competing interests.

References

1. Deo SVS, Sharma J and Kumar S: GLOBOCAN 2020 report on global cancer burden: Challenges and opportunities for surgical oncologists. Ann Surg Oncol 29: 6497-6500, 2022.
2. Xia C, Dong X, Li H, Cao M, Sun D, He S, Yang F, Yan X, Zhang S, Li N and Chen W: Cancer statistics in China and United States, 2022: Profiles, trends, and determinants. Chin Med J (Engl) 135: 584-590, 2022.
3. Wei C, Zhao L, Liang H, Zhen Y and Han L: Recent advances in unraveling the molecular mechanisms and functions of HOXA11-AS in human cancers and other diseases (review). Oncol Rep 43: 1737-1754, 2020.
4. Mao M, Zhang J, Xiang Y, Gong M, Deng Y and Ye D: Role of exosomal competitive endogenous RNA (ceRNA) in diagnosis and treatment of malignant tumors. Bioengineered 13: 12156-12168, 2022.
5. Bhan A, Soleimani M and Mandal SS: Long noncoding RNA and Cancer: A new paradigm. Cancer Res 77: 3965-3981, 2017.
6. Ye J, Li J and Zhao P: Roles of ncRNAs as ceRNAs in gastric cancer. Genes (Basel) 12: 1036, 2021.
7. Lee YR and Pandolfi PP: PTEN mouse models of cancer initiation and progression. Cold Spring Harb Perspect Med 10: a037283, 2020.
8. Wang J, Zhu S, Meng N, He Y, Lu R and Yan GR: ncRNA-encoded peptides or proteins and cancer. Mol Ther 27: 1718-1725, 2019.
9. Liu C, Wu Y and Ma J: Interaction of non-coding RNAs and Hippo signaling: Implications for tumorigenesis. Cancer Lett 493: 207-216, 2020.
10. Slack FJ and Chinnaiyan AM: The role of non-coding RNAs in oncology. Cell 179: 1033-1055, 2019.
11. Anastasiadou E, Jacob LS and Slack FJ: Non-coding RNA networks in cancer. Nat Rev Cancer 18: 5-18, 2018.
12. Zhang J, Li K, Zheng H and Zhu Y: Research progress review on long non-coding RNA in colorectal cancer. Neoplasma 68: 240-252, 2021.
13. Basera A, Hull R, Demetriou D, Bates DO, Kaufmann AM, Dlamini Z and Marima R: Competing endogenous RNA (ceRNA) networks and splicing switches in cervical cancer: HPV oncogenesis, clinical significance and therapeutic opportunities. Microorganisms 10: 1852, 2022.
14. Liu Y, Khan S, Li L, Ten Hagen TLM and Falahati M: Molecular mechanisms of thyroid cancer: A competing endogenous RNA (ceRNA) point of view. Biomed Pharmacother 146: 112251, 2022.
15. Liu H, Deng H, Zhao Y, Li C and Liang Y: LncRNA XIST/miR-34a axis modulates the cell proliferation and tumor growth of thyroid cancer through MET-PI3K-AKT signaling. J Exp Clin Cancer Res 37: 279, 2018.
16. Lima CR, Geraldo MV, Fuziwara CS, Kimura ET and Santos MF: MiRNA-146b-5p upregulates migration and invasion of different Papillary thyroid carcinoma cells. BMC Cancer 16: 108, 2016.
17. Worst TS, Previti C, Nitschke K, Diessl N, Gross JC, Hoffmann L, Frey L, Thomas V, Kahlert C, Bieback K, *et al*: miR-10a-5p and miR-29b-3p as extracellular vesicle-associated prostate cancer detection markers. Cancers (Basel) 12: 43, 2019.
18. Wang J, Wang B, Chen LQ, Yang J, Gong ZQ, Zhao XL, Zhang CQ and Du KL: miR-10b promotes invasion by targeting KLF4 in osteosarcoma cells. Biomed Pharmacother 84: 947-953, 2016.
19. Zhu H, Kang M and Bai X: TCF21 regulates miR-10a-5p/LIN28B signaling to block the proliferation and invasion of melanoma cells. PLoS One 16: e0255971, 2021.
20. Wang L, Cho KB, Li Y, Tao G, Xie Z and Guo B: Long noncoding RNA (lncRNA)-mediated competing endogenous RNA networks provide novel potential biomarkers and therapeutic targets for colorectal cancer. Int J Mol Sci 20: 5758, 2019.
21. Xing C, Sun SG, Yue ZQ and Bai F: Role of lncRNA LUCAT1 in cancer. Biomed Pharmacother 134: 111158, 2021.
22. Tan YT, Lin JF, Li T, Li JJ, Xu RH and Ju HQ: LncRNA-mediated posttranslational modifications and reprogramming of energy metabolism in cancer. Cancer Commun (Lond) 41: 109-120, 2021.
23. Peng WX, Koirala P and Mo YY: LncRNA-mediated regulation of cell signaling in cancer. Oncogene 36: 5661-5667, 2017.
24. Tang X, Ren H, Guo M, Qian J, Yang Y and Gu C: Review on circular RNAs and new insights into their roles in cancer. Comput Struct Biotechnol J 19: 910-928, 2021.
25. Chen L, Wang C, Sun H, Wang J, Liang Y, Wang Y and Wong G: The bioinformatics toolbox for circRNA discovery and analysis. Brief Bioinform 22: 1706-1728, 2021.
26. Wu P, Mo Y, Peng M, Tang T, Zhong Y, Deng X, Xiong F, Guo C, Wu X, Li Y, *et al*: Emerging role of tumor-related functional peptides encoded by lncRNA and circRNA. Mol Cancer 19: 22, 2020.
27. Yu T, Wang Y, Fan Y, Fang N, Wang T, Xu T and Shu Y: CircRNAs in cancer metabolism: A review. J Hematol Oncol 12: 90, 2019.
28. Memczak S, Jens M, Elefsinioti A, Torti F, Krueger J, Rybak A, Maier L, Mackowiak SD, Gregersen LH, Munschauer M, *et al*: Circular RNAs are a large class of animal RNAs with regulatory potency. Nature 495: 333-338, 2013.
29. Kong S, Tao M, Shen X and Ju S: Translatable circRNAs and lncRNAs: Driving mechanisms and functions of their translation products. Cancer Lett 483: 59-65, 2020.
30. Hansen TB, Jensen TI, Clausen BH, Bramsen JB, Finsen B, Damgaard CK and Kjems J: Natural RNA circles function as efficient microRNA sponges. Nature 495: 384-388, 2013.
31. Saliminejad K, Khorram Khorshid HR, Soleymani Fard S and Ghaffari SH: An overview of microRNAs: Biology, functions, therapeutics, and analysis methods. J Cell Physiol 234: 5451-5465, 2019.
32. Wang S, Talukder A, Cha M, Li X and Hu H: Computational annotation of miRNA transcription start sites. Brief Bioinform 22: 380-392, 2021.
33. Ali Syeda Z, Langden SSS, Munkhzul C, Lee M and Song SJ: Regulatory mechanism of MicroRNA expression in cancer. Int J Mol Sci 21: 1723, 2020.
34. Catalanotto C, Cogoni C and Zardo G: MicroRNA in control of gene expression: An overview of nuclear functions. Int J Mol Sci 17: 1712, 2016.
35. He B, Zhao Z, Cai Q, Zhang Y, Zhang P, Shi S, Xie H, Peng X, Yin W, Tao Y and Wang X: miRNA-based biomarkers, therapies, and resistance in Cancer. Int J Biol Sci 16: 2628-2647, 2020.
36. Zhao Z, Sun W, Guo Z, Zhang J, Yu H and Liu B: Mechanisms of lncRNA/microRNA interactions in angiogenesis. Life Sci 254: 116900, 2020.
37. Yang L, Sun HF, Guo LQ and Cao HB: MiR-10a-5p: A promising biomarker for early diagnosis and prognosis evaluation of bladder cancer. Cancer Manag Res 13: 7841-7850, 2021.
38. Han TS, Hur K, Cho HS and Ban HS: Epigenetic associations between lncRNA/circRNA and miRNA in hepatocellular carcinoma. Cancers (Basel) 12: 2622, 2020.
39. Qi X, Zhang DH, Wu N, Xiao JH, Wang X and Ma W: ceRNA in cancer: Possible functions and clinical implications. J Med Genet 52: 710-718, 2015.
40. Zhu J, Zhang X, Gao W, Hu H, Wang X and Hao D: lncRNA/circRNA-miRNA-mRNA ceRNA network in lumbar intervertebral disc degeneration. Mol Med Rep 20: 3160-3174, 2019.
41. Cheng Y, Su Y, Wang S, Liu Y, Jin L, Wan Q, Liu Y, Li C, Sang X, Yang L, *et al*: Identification of circRNA-lncRNA-miRNA-mRNA competitive endogenous RNA network as novel prognostic markers for acute myeloid leukemia. Genes (Basel) 11: 868, 2020.
42. Salmena L, Poliseno L, Tay Y, Kats L and Pandolfi PP: A ceRNA hypothesis: The Rosetta Stone of a hidden RNA language? Cell 146: 353-358, 2011.
43. Moreno-Garcia L, López-Royo T, Calvo AC, Toivonen JM, de la Torre M, Moreno-Martínez L, Molina N, Aparicio P, Zaragoza P, Manzano R and Osta R: Competing endogenous RNA networks as biomarkers in neurodegenerative diseases. Int J Mol Sci 21: 9582, 2020.
44. Zhou T, Lin K, Nie J, Pan B, He B, Pan Y, Sun H, Xu T and Wang S: LncRNA SPINT1-AS1 promotes breast cancer proliferation and metastasis by sponging let-7 a/b/i-5p. Pathol Res Pract 217: 153268, 2021.
45. Tajima H, Kumazaki T, Gemma K, Iida E, Kawamata H, Murakami R, Goto S and Aoyama T: Rotational digital angiography of ulcer-like projection of pelvis. Radiat Med 14: 49-51, 1996.

46. Li C, Mu J, Shi Y and Xin H: LncRNA CCDC26 interacts with CELF2 protein to enhance myeloid leukemia cell proliferation and invasion via the circRNA_ANKIB1/miR-195-5p/PRR11 axis. Cell Transplant 30: 963689720986080, 2021.
47. Zeng X, Xiao J, Bai X, Liu Y, Zhang M, Liu J, Lin Z and Zhang Z: Research progress on the circRNA/lncRNA-miRNA-mRNA axis in gastric cancer. Pathol Res Pract 238: 154030, 2022.
48. Cao J, Zhang M, Zhang L, Lou J, Zhou F and Fang M: Non-coding RNA in thyroid cancer-functions and mechanisms. Cancer Lett 496: 117-126, 2021.
49. Tehler D, Hoyland-Kroghsbo NM and Lund AH: The miR-10 microRNA precursor family. RNA Biol 8: 728-734, 2011.
50. Liu F, Shi Y, Liu Z, Li Z and Xu W: The emerging role of miR-10 family in gastric cancer. Cell Cycle 20: 1468-1476, 2021.
51. Guo L, Li Y, Zhao C, Peng J, Song K, Chen L, Zhang P, Ma H, Yuan C, Yan S, *et al*: RECQL4, negatively regulated by miR-10a-5p, facilitates cell proliferation and invasion via MAFB in ovarian cancer. Front Oncol 10: 524128, 2020.
52. Lu Y, Wei G, Liu L, Mo Y, Chen Q, Xu L, Liao R, Zeng D and Zhang K: Direct targeting of MAPK8IP1 by miR-10a-5p is a major mechanism for gastric cancer metastasis. Oncol Lett 13: 1131-1136, 2017.
53. Zhu J, Du S, Zhang J, Huang G, Dong L, Ren E and Liu D: microRNA-10a-5p from gastric cancer cell-derived exosomes enhances viability and migration of human umbilical vein endothelial cells by targeting zinc finger MYND-type containing 11. Bioengineered 13: 496-507, 2022.
54. Vu TT, Stölzel F, Wang KW, Röllig C, Tursky ML, Molloy TJ and Ma DD: miR-10a as a therapeutic target and predictive biomarker for MDM2 inhibition in acute myeloid leukemia. Leukemia 35: 1933-1948, 2021.
55. Kuratomi N, Takano S, Fukasawa M, Maekawa S, Kadokura M, Shindo H, Takahashi E, Hirose S, Fukasawa Y, Kawakami S, *et al*: MiR-10a in pancreatic juice as a biomarker for invasive intraductal papillary mucinous neoplasm by miRNA sequencing. Int J Mol Sci 22: 3221, 2021.
56. Thai AA, Solomon BJ, Sequist LV, Gainor JF and Heist RS: Lung cancer. Lancet 398: 535-554, 2021.
57. Bade BC and Dela Cruz CS: Lung cancer 2020: Epidemiology, etiology, and prevention. Clin Chest Med 41: 1-24, 2020.
58. Zhu Y, Ma K, Ye Y, Tang J and Zhu J: Long non-coding RNA LINRIS is upregulated in non-small cell lung cancer and its silencing inhibits cell proliferation by suppressing microRNA-10a maturation. Bioengineered 13: 4340-4346, 2022.
59. Gao Y, Zhao H and Mu L: LncRNA-KAT7 negatively regulates miR-10a through an epigenetic pathway to participate in nonsmall cell lung cancer. Cancer Biother Radiopharm 36: 441-445, 2021.
60. Thrift AP: Global burden and epidemiology of Barrett oesophagus and oesophageal cancer. Nat Rev Gastroenterol Hepatol 18: 432-443, 2021.
61. Iriarte F, Su S, Petrov RV, Bakhos CT and Abbas AE: Surgical management of early esophageal cancer. Surg Clin North Am 101: 427-441, 2021.
62. He N, Xiang L, Chen L, Tong H, Wang K, Zhao J, Song F, Yang H, Wei X and Jiao Z: The role of long non-coding RNA FGD5-AS1 in cancer. Bioengineered 13: 11026-11041, 2022.
63. Feng B, Wang G, Liang X, Wu Z, Wang X, Dong Z, Guo Y, Shen S, Liang J and Guo W: LncRNA FAM83H-AS1 promotes oesophageal squamous cell carcinoma progression via miR-10a-5p/Girdin axis. J Cell Mol Med 24: 8962-8976, 2020.
64. Llovet JM, Kelley RK, Villanueva A, Singal AG, Pikarsky E, Roayaie S, Lencioni R, Koike K, Zucman-Rossi J and Finn RS: Hepatocellular carcinoma. Nat Rev Dis Primers 7: 6, 2021.
65. Anwanwan D, Singh SK, Singh S, Saikam V and Singh R: Challenges in liver cancer and possible treatment approaches. Biochim Biophys Acta Rev Cancer 1873: 188314, 2020.
66. Li H, Zhao X, Li C, Sheng C and Bai Z: Integrated analysis of lncRNA-associated ceRNA network reveals potential biomarkers for the prognosis of hepatitis B virus-related hepatocellular carcinoma. Cancer Manag Res 11: 877-897, 2019.
67. Zhang Y, Liu J, Lv Y, Zhang C and Guo S: LncRNA meg3 suppresses hepatocellular carcinoma in vitro and vivo studies. Am J Transl Res 11: 4089-4099, 2019.
68. Wu Y, Zhou Y, Huan L, Xu L, Shen M, Huang S and Liang L: LncRNA MIR22HG inhibits growth, migration and invasion through regulating the miR-10a-5p/NCOR2 axis in hepatocellular carcinoma cells. Cancer Sci 110: 973-984, 2019.
69. Gao J, Dai C, Yu X, Yin XB and Zhou F: Long noncoding RNA LEF1-AS1 acts as a microRNA-10a-5p regulator to enhance MSI1 expression and promote chemoresistance in hepatocellular carcinoma cells through activating AKT signaling pathway. J Cell Biochem 122: 86-99, 2021.
70. Zhu H, Du F and Cao C: Restoration of circPSMC3 sensitizes gefitinib-resistant esophageal squamous cell carcinoma cells to gefitinib by regulating miR-10a-5p/PTEN axis. Cell Biol Int 45: 107-116, 2021.
71. Dekker E, Tanis PJ, Vleugels JLA, Kasi PM and Wallace MB: Colorectal cancer. Lancet 394: 1467-1480, 2019.
72. Fan A, Wang B, Wang X, Nie Y, Fan D, Zhao X and Lu Y: Immunotherapy in colorectal cancer: Current achievements and future perspective. Int J Biol Sci 17: 3837-3849, 2021.
73. Ren W, Chen S, Liu G, Wang X, Ye H and Xi Y: TUSC7 acts as a tumor suppressor in colorectal cancer. Am J Transl Res 9: 4026-4035, 2017.
74. Yang RQ, Jin ZZ, Jiang SY and Jin YJ: LncRNA GAS5 interacts with MicroRNA-10b to inhibit cell proliferation and migration and induces apoptosis in colorectal cancer. Comput Math Methods Med 2022: 4996870, 2022.
75. Wu Y, Cong L, Chen W, Wang X and Qiu F: lncRNA LINC00963 downregulation regulates colorectal cancer tumorigenesis and progression via the miR-10b/FGF13 axis. Mol Med Rep 23: 211, 2021.
76. Li F, Aljahdali IAM, Zhang R, Nastiuk KL, Krolewski JJ and Ling X: Kidney cancer biomarkers and targets for therapeutics: Survivin (BIRC5), XIAP, MCL-1, HIF1α, HIF2α, NRF2, MDM2, MDM4, p53, KRAS and AKT in renal cell carcinoma. J Exp Clin Cancer Res 40: 254, 2021.
77. Yong C, Stewart GD and Frezza C: Oncometabolites in renal cancer. Nat Rev Nephrol 16: 156-172, 2020.
78. Dong D, Mu Z, Wei N, Sun M, Wang W, Xin N, Shao Y and Zhao C: Long non-coding RNA ZFAS1 promotes proliferation and metastasis of clear cell renal cell carcinoma via targeting miR-10a/SKA1 pathway. Biomed Pharmacother 111: 917-925, 2019.
79. Xu Z, Ye J, Bao P, Wu Q, Xie F and Li P: Long non-coding RNA SNHG3 promotes the progression of clear cell renal cell carcinoma via regulating BIRC5 expression. Transl Cancer Res 10: 4502-4513, 2021.
80. Luo Y, Yang J, Yu J, Liu X, Yu C, Hu J, Shi H and Ma X: Long non-coding RNAs: Emerging roles in the immunosuppressive tumor microenvironment. Front Oncol 10: 48, 2020.
81. Liu RJ, Xu ZP, Li S, Yu JJ, Xu B and Chen M: Identification a ceRNA (XIST/miR-10a-5p/SERPINE1) axis as a prognostic biomarker in kidney renal clear cell carcinoma. Research Square, 2021.
82. Liu RJ, Xu ZP, Li SY, Yu JJ, Feng NH, Xu B and Chen M: BAP1-related ceRNA (NEAT1/miR-10a-5p/SERPINE1) promotes proliferation and migration of kidney cancer cells. Front Oncol 12: 852515, 2022.
83. Tan P, Chen H, Huang Z, Huang M, Du Y, Li T, Chen Z, Liu Y and Fu W: MMP25-AS1/hsa-miR-10a-5p/SERPINE1 axis as a novel prognostic biomarker associated with immune cell infiltration in KIRC. Mol Ther Oncolytics 22: 307-325, 2021.
84. Liang Y, Zhang H, Song X and Yang Q: Metastatic heterogeneity of breast cancer: Molecular mechanism and potential therapeutic targets. Semin Cancer Biol 60: 14-27, 2020.
85. Coughlin SS: Epidemiology of breast cancer in women. Adv Exp Med Biol 1152: 9-29, 2019.
86. Matamala N, Vargas MT, González-Cámpora R, Miñambres R, Arias JI, Menéndez P, Andrés-León E, Gómez-López G, Yanowsky K, Calvete-Candenas J, *et al*: Tumor microRNA expression profiling identifies circulating microRNAs for early breast cancer detection. Clin Chem 61: 1098-1106, 2015.
87. Lu Y, Ding Y, Wei J, He S, Liu X, Pan H, Yuan B, Liu Q and Zhang J: Anticancer effects of traditional Chinese medicine on epithelial-mesenchymal transition (EMT) in breast cancer: Cellular and molecular targets. Eur J Pharmacol 907: 174275, 2021.
88. Liu Z, Huang L, Sun L, Nie H, Liang Y, Huang J, Wu F and Hu X: Ecust004 suppresses breast cancer cell growth, invasion, and migration via EMT regulation. Drug Des Devel Ther 15: 3451-3461, 2021.
89. Wang B, Zhang Y, Zhang H, Lin F, Tan Q, Qin Q, Bao W, Liu Y, Xie J and Zeng Q: Long intergenic non-protein coding RNA 324 prevents breast cancer progression by modulating miR-10b-5p. Aging (Albany NY) 12: 6680-6699, 2020.

90. Wang D, Wang Z, Zhang L and Sun S: LncRNA PDCD4-AS1 alleviates triple negative breast cancer by increasing expression of IQGAP2 via miR-10b-5p. Transl Oncol 14: 100958, 2021.
91. Berek JS, Renz M, Kehoe S, Kumar L and Friedlander M: Cancer of the ovary, fallopian tube, and peritoneum: 2021 Update. Int J Gynaecol Obstet 155 (Suppl 1): S61-S85, 2021.
92. Tan WX, Sun G, Shangguan MY, Gui Z, Bao Y, Li YF and Jia ZH: Novel role of lncRNA CHRF in cisplatin resistance of ovarian cancer is mediated by miR-10b induced EMT and STAT3 signaling. Sci Rep 10: 14768, 2020.
93. Zhu Y, Mo M, Wei Y, Wu J, Pan J, Freedland SJ, Zheng Y and Ye D: Epidemiology and genomics of prostate cancer in Asian men. Nat Rev Urol 18: 282-301, 2021.
94. Adamaki M and Zoumpourlis V: Prostate cancer biomarkers: From diagnosis to prognosis and precision-guided therapeutics. Pharmacol Ther 228: 107932, 2021.
95. Liu S, Wang L, Li Y, Cui Y, Wang Y and Liu C: Long non-coding RNA CHRF promotes proliferation and mesenchymal transition (EMT) in prostate cancer cell line PC3 requiring up-regulating microRNA-10b. Biol Chem: Jul 9, 2019 (Epub ahead of print).
96. Li WJ, Li G, Liu ZW, Chen ZY and Pu R: LncRNA LINC00355 promotes EMT and metastasis of bladder cancer cells through the miR-424-5p/HMGA2 axis. Neoplasma 68: 1225-1235, 2021.
97. Li G, Zhang Y, Mao J, Hu P, Chen Q, Ding W and Pu R: lncRNA TUC338 is a potential diagnostic biomarker for bladder cancer. J Cell Biochem 120: 18014-18019, 2019.
98. Ye M, Gao R, Chen S, Wei M, Wang J, Zhang B, Wu S, Xu Y, Wu P, Chen X, *et al*: Downregulation of MEG3 and upregulation of EZH2 cooperatively promote neuroblastoma progression. J Cell Mol Med 26: 2377-2391, 2022.
99. Zhou X, Lu H, Li F, Han L, Zhang H, Jiang Z, Dong Q and Chen X: LncRNA cancer susceptibility candidate (CASC7) upregulates phosphatase and tensin homolog by down-regulating miR-10a to inhibit neuroblastoma cell proliferation. Neuroreport 31: 381-386, 2020.
100. Tan AC, Ashley DM, López GY, Malinzak M, Friedman HS and Khasraw M: Management of glioblastoma: State of the art and future directions. CA Cancer J Clin 70: 299-312, 2020.
101. Uddin MS, Mamun AA, Alghamdi BS, Tewari D, Jeandet P, Sarwar MS and Ashraf GM: Epigenetics of glioblastoma multiforme: From molecular mechanisms to therapeutic approaches. Semin Cancer Biol 83: 100-120, 2022.
102. Hao SC, Ma H, Niu ZF, Sun SY, Zou YR and Xia HC: hUC-MSCs secreted exosomes inhibit the glioma cell progression through PTENP1/miR-10a-5p/PTEN pathway. Eur Rev Med Pharmacol Sci 23: 10013-10023, 2019.
103. Ding Y, Wang J, Zhang H and Li H: Long noncoding RNA-GAS5 attenuates progression of glioma by eliminating microRNA-10b and Sirtuin 1 in U251 and A172 cells. Biofactors 46: 487-496, 2020.
104. Shang C, Tang W, Pan C, Hu X and Hong Y: Long non-coding RNA TUSC7 inhibits temozolomide resistance by targeting miR-10a in glioblastoma. Cancer Chemother Pharmacol 81: 671-678, 2018.
105. Saleh K, Khalifeh-Saleh N and Kourie HR: Acute myeloid leukemia transformed to a targetable disease. Future Oncol 16: 961-972, 2020.
106. Yuan Z and Wang W: LncRNA SNHG4 regulates miR-10a/PTEN to inhibit the proliferation of acute myeloid leukemia cells. Hematology 25: 160-164, 2020.
107. Petty RD, Dahle-Smith A, Stevenson DAJ, Osborne A, Massie D, Clark C, Murray GI, Dutton SJ, Roberts C, Chong IY, *et al*: Gefitinib and EGFR gene copy number aberrations in esophageal cancer. J Clin Oncol 35: 2279-2287, 2017.
108. Kang J, Guo Z, Zhang H, Guo R, Zhu X and Guo X: Dual inhibition of EGFR and IGF-1R signaling leads to enhanced antitumor efficacy against esophageal squamous cancer. Int J Mol Sci 23: 10382, 2022.
109. Fu X, Cui G, Liu S and Zhao S: Linc01014 regulates gefitinib resistance in oesophagus cancer via EGFR-PI3K-AKT-mTOR signalling pathway. J Cell Mol Med 24: 1670-1675, 2020.
110. Zhang K, Fan R, Zhao D, Liu Z, Yang Z, Liu J, Zhang S, Rao S, Wang Y and Wan L: CircATIC inhibits esophageal carcinoma progression and promotes radiosensitivity by elevating RHCG through sponging miR-10-3p. Thorac Cancer 13: 934-946, 2022.
111. Lu C, Jiang W, Hui B, Rong D, Fu K, Dong C, Tang W and Cao H: The circ_0021977/miR-10b-5p/P21 and P53 regulatory axis suppresses proliferation, migration, and invasion in colorectal cancer. J Cell Physiol 235: 2273-2285, 2020.
112. Liu M: CircFAT1 is overexpressed in colorectal cancer and suppresses cancer cell proliferation, invasion and migration by increasing the maturation of miR-10a. Cancer Manag Res 13: 4309-4315, 2021.
113. Luo L, Gao YQ and Sun XF: Circular RNA ITCH suppresses proliferation and promotes apoptosis in human epithelial ovarian cancer cells by sponging miR-10a-α. Eur Rev Med Pharmacol Sci 22: 8119-8126, 2018.
114. Teng F, Xu J, Zhang M, Liu S, Gu Y, Zhang M, Wang X, Ni J, Qian B, Shen R and Jia X: Comprehensive circular RNA expression profiles and the tumor-suppressive function of circHIPK3 in ovarian cancer. Int J Biochem Cell Biol 112: 8-17, 2019.
115. Raglan O, Kalliala I, Markozannes G, Cividini S, Gunter MJ, Nautiyal J, Gabra H, Paraskevaidis E, Martin-Hirsch P, Tsilidis KK and Kyrgiou M: Risk factors for endometrial cancer: An umbrella review of the literature. Int J Cancer 145: 1719-1730, 2019.
116. Urick ME and Bell DW: Clinical actionability of molecular targets in endometrial cancer. Nat Rev Cancer 19: 510-521, 2019.
117. Yang P, Yun K and Zhang R: CircRNA circ-ATAD1 is down-regulated in endometrial cancer and suppresses cell invasion and migration by downregulating miR-10a through methylation. Mamm Genome 32: 488-494, 2021.
118. Cui C, Yang J, Li X, Liu D, Fu L and Wang X: Functions and mechanisms of circular RNAs in cancer radiotherapy and chemotherapy resistance. Mol Cancer 19: 58, 2020.
119. Kim MK, Cho KJ, Kwon GY, Park SI, Kim YH, Kim JH, Song HY, Shin JH, Jung HY, Lee GH, *et al*: ERCC1 predicting chemoradiation resistance and poor outcome in oesophageal cancer. Eur J Cancer 44: 54-60, 2008.
120. Tan S, Li D and Zhu X: Cancer immunotherapy: Pros, cons and beyond. Biomed Pharmacother 124: 109821, 2020.
121. van den Bulk J, Verdegaal EM and de Miranda NF: Cancer immunotherapy: Broadening the scope of targetable tumours. Open Biol 8: 180037, 2018.