Biological functions and potential mechanisms of miR-143-3p in cancers (Review)

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Abstract. In recent years, microRNAs (miRNAs or miRs) have been increasingly studied for their role in cancer and have shown potential as cancer biomarkers. miR-143-3p and miR-143-5p are the mature miRNAs derived from pre-miRNA-143. At present, there are numerous studies on the function of miR-143-3p in cancer progression, but there are no systematic reviews describing the function of miR-143-3p in cancer. It is widely considered that miR-143-3p is downregulated in most malignant tumors and that upstream regulators can act on this gene, which in turn regulates the corresponding target to act on the tumor. In addition, miRNA-143-3p can regulate target genes to affect the biological process of tumors through various signaling pathways, such as the PI3K/Akt, Wnt/β-catenin, AKT/STAT3 and Ras-Raf-MEK-ERK pathways. The present review comprehensively described the biogenesis of miR-143-3p, the biological functions of miR-143-3p and the related roles and mechanisms in different cancer types. The potential of miR-143-3p as a biomarker for cancer was also highlighted and valuable future research directions were discussed.

Contents

1. Introduction
2. Biogenesis of miR-143-3p
3. Regulation and effect of miR-143-3p expression
4. Mechanism of miR-143-3p
5. Potential application of miR-143-3p in future practice
6. Discussion

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1. Introduction

The 2020 Global Cancer Statistics report stated that there were 19.3 million new cancer cases and almost 10 million cancer-related deaths in 2020 (1). Previous research has suggested that there will be 22.2 million new cases among all cancer types in 2030, and cancer will continue to be the principal cause of increasing morbidity and mortality in all regions of the world for decades to come (2,3). However, early screening for cancer is still not widespread, therefore numerous patients are diagnosed in the late stage, at which point radical treatments such as surgery are not ideal; thus, late diagnosis markedly reduces the survival rate of patients. Recently, an increasing number of new therapeutic methods, such as natural killer cell-based cancer immunotherapy (4), RAS-targeted cancer therapy (5) and immune checkpoint inhibitor (ICI) therapy, have been developed (6). Although the discovery of ICIs represents a major breakthrough in cancer therapy, there are still some patients who are unresponsive to treatment or who cannot tolerate the side effects. Therefore, identifying more effective treatments is crucial for improving patient prognosis.

In humans, >2,600 mature microRNA (miRNA or miR) sequences have been identified (7). MiRNAs are small non-coding RNAs that are ~19-25 nucleotides in length and negatively modulate gene expression by base pairing to the 3'-untranslated region (UTR) of target messenger RNAs (mRNAs). They are usually abnormally expressed in tumor cells and can modulate the apoptosis, proliferation, survival and metastasis of cancer cells (8-10). It is considered by numerous scholars (11-13) that abnormal expression of miR-143-3p in malignant tumors plays a crucial role in tumor progression and can also act as a diagnostic and prognostic marker; it has even been studied as a therapeutic target in the last few years. In the present review, the functions of miR-143-3p in malignant tumors and the potential underlying mechanisms involved were systematically described.

2. Biogenesis of miR-143-3p

Mature miRNAs are generated by successive pretreatment events: Pri-miRNAs are processed into 70-nucleotide pre-miRNAs in the nucleus, after which the pre-miRNAs are processed into mature miRNAs in the cytoplasm (14).

miR-143-3p is no exception. First, miRNA-143 is transcribed into pri-miRNA-143 by RNA polymerase II, which functions in the nucleus (15), after which the cellular RNase III Drosha processes pri-miRNA-143 into a 60-110 nucleotide structure (16). Then, pre-miRNA-143 is transported into the cytoplasm via the RanGTP/Exportin 5 pathway (17). Pre-miRNA-143 in the cytoplasm is cleaved by the RNase III Dicer to generate short miRNA-143 duplexes, which are then rapidly unwound by helicases to produce mature miRNA-143 (18). Mature miRNA-143 and proteins bind to form an RNA-induced silencing complex, which can lead to target mRNA degradation or translation suppression (17). Interestingly, under certain conditions, two different mature miRNAs can be generated from a pre-miRNA (15). In humans, removal of the opposite arm of pre-miR-143 can produce two diverse mature miRNAs, namely, *Homo sapiens* (has)-miR-143-5p and hsa-miR-143-3p. Although they are generated from the same primary transcript, their sequences differ widely, therefore they target different mRNAs to play different roles (18) (Fig. 1).

3. Regulation and effect of miR-143-3p expression

According to previous studies, miR-143-3p is often downregulated in malignant tumors of various systems, such as lung cancer (LC) (19), gastric cancer (GC) (20), bladder cancer (BC) (21), cervical cancer (CC) (22), thyroid cancer (TC) (23) and melanoma (12), and can impact the proliferation, migration and apoptosis of malignant tumor cells. The regulation and effect of miR-143-3p expression in various systems have been investigated in studies mainly focusing on the upstream factors and target genes that regulate miR-143-3p (Table I).

miR-143-3p in the respiratory system

LC. LC is the second most common cancer and a significant cause of cancer-related death; LC accounts for ~1/10 cancer cases (11.4%) and 1/5 cancer-related deaths (18.0%) (1). LC can be divided into two different types according to histopathological type: Non-small cell LC (NSCLC) and small cell LC (SCLC), with NSCLC predominating. It is considered by numerous scholars (19,24,25) that miR-143-3p acts as an important regulatory gene in NSCLC and lung adenocarcinoma (LUAD) to affect tumorigenicity and progression. miR-143-3p is often downregulated in NSCLC and LUAD and is regulated by its upstream regulators. For instance, the downregulation of long non-coding RNA (lncRNA)-TMPO-AS1 can result in high miR-143-3p expression, which leads to a decrease in CDK1 and ultimately participates in accelerating the apoptosis of LC cells (25).

In NSCLC, the lncRNA UCC is highly expressed and can act as a competing endogenous RNA (ceRNA) and compete with miR-143-3p to suppress miR-143-3p expression and subsequently regulate SOX5 expression. Mechanistically, the lncRNA UCC induces SOX5 expression through miR-143-3p, induces NSCLC cell proliferation and migration, and subsequently promotes epithelial-mesenchymal transition (EMT) in NSCLC (24). Another study revealed that miR-143-3p suppresses tumor growth, while LINC00667 and RRM2 promote tumor growth. The lncRNA LINC00667 can suppress miR-143-3p expression and indirectly regulate the expression of RRM2, a target gene of miR-143-3p. In conclusion, NSCLC growth is modulated by the LINC00667/miR-143-3p/RRM2 signaling pathway (26). In addition, the circTUBA 1C/miR-143-3p axis plays a significant role in NSCLC. CircTUBA 1C can negatively modulate miR-143-3p expression. Rescue experiments illustrated that knockdown of miR-143-3p could reverse the decrease in tumor growth induced by circTUBA 1C deficiency (27).

In LUAD, circSNK1G3 inhibits miR-143-3p expression. Downregulation of miR-143-3p induces homeobox (HOX) A10 (HOXA10) expression, thereby promoting LUAD cell growth and metastasis (19). On the other hand, miR-143-3p is targeted by the lncRNA PCAT19, and the expression of these two genes is positively correlated. LUAD cell proliferation, invasion and migration can be accelerated by downregulation of lncRNA PCAT19; nevertheless, the overexpression of miR-143-3p may attenuate these effects (28).

However, compared with that in patients with LC without brain metastases (BMs), the expression of miR-143-3p in patients with LC with BMs is markedly increased, and this increase in miR-143-3p increases the metastatic potential of LC cells and promotes angiogenesis (29). miR-143-3p expression is also upregulated in exosomes derived from granulocytic myeloid-derived suppressor cells (G-MDSCs) in LC tissues, and exosomes secreted by G-MDSCs can be transferred to LC cells to accelerate tumor progression (13). miR-143-3p is clearly associated with the malignant progression of cancer.

miR-143-3p in the digestive system

Hepatocellular carcinoma (HCC). Primary liver cancer was the third most common cause of cancer-related death worldwide according to a 2020 study, and HCC accounted for ~75-85% of these LC-related deaths (1). It has been suggested that miR-143-3p plays a major role in HCC progression. The findings of a previous study suggested that miR-143-3p is expressed at a low level in HCC and is a potential target of circ0003998. FOS-Like antigen 2 (FOSL2) is a downstream target of the circ0003998/miR-143-3p axis, and miR-143-3p can downregulate FOSL2 expression and affect the migration of HCC cells (30). miR-143-3p is also a target of the lncRNA SOx2-OT, and its expression levels are negatively correlated. The lncRNA SOx2-OT competitively binds with miR-143-3p to promote the expression of Musashi RNA binding protein 2 (MSI2) and consequently promote the malignant progression of HCC (31). Another study showed that the lncRNA metastasis-associated lung adenocarcinoma transcript 1 (MALAT1) is highly expressed in HCC tissues and binds to miR-143-3p to inhibit miR-143-3p expression; subsequently, miR-143-3p accelerates the progression of HCC by regulating zinc finger E-box binding homeobox 1 (ZEB1) expression (32). On the other hand, miR-143-3p may suppress HCC cell invasion and proliferation by directly modulating the target genes lncRNA PSMG3-AS1 and fibroblast growth factor 1 (FGF1) (33,34).

GC. In 2020, >1 million new cases of GC and 769,000 related deaths were reported worldwide; thus, GC remains an important cancer worldwide (1). In GC, miR-143-3p expression is influenced by various genes. As a ceRNA, the lncRNA CCDC144NL-AS1 interacts with miR-143-3p to negatively regulate its expression, thereby regulating MAP3K7 expression in GC. Mechanistically, the lncRNA CCDC144NL-AS1

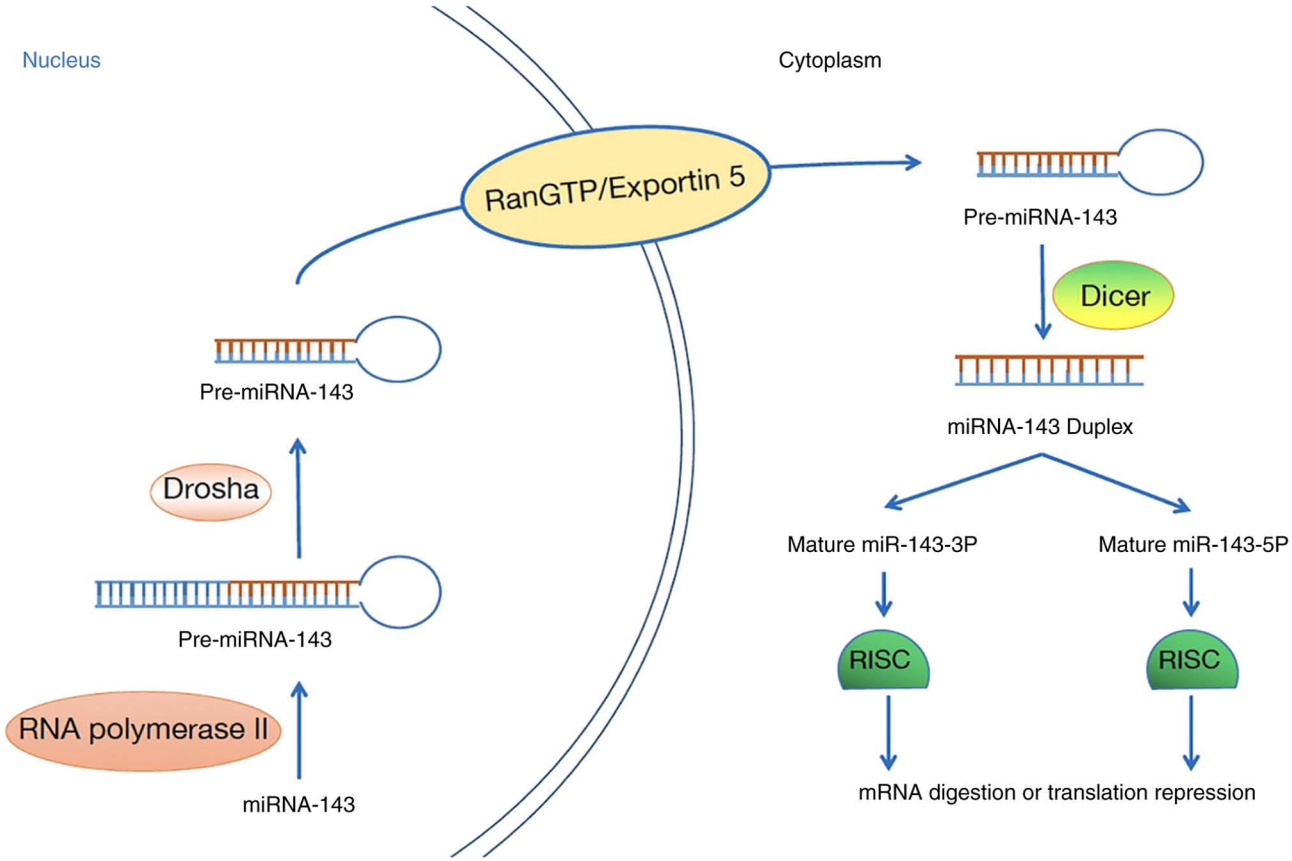

Figure 1. Schematic about miR-143-3p biogenesis. miR or miRNA, microRNA; RISC, RNA-induced silencing complex.

alters MAP3K7 levels by modulating miR-143-3p expression and subsequently acts as an oncogene in GC progression (35). LINC00200 also functions as a ceRNA, negatively regulating miR-143-3p expression, while miR-143-3p negatively regulates its target gene Serpin Family E Member 1 (SERPINE1). Through the aforementioned process, LINC00200 ultimately affects the proliferation of GC cells (36). Furthermore, miR-143-3p is considered the downstream target of circ_0006089, and circ_0006089 can actively modulate insulin-like growth factor 1 receptor (IGF1R) expression by inhibiting miR-143-3p, thus promoting the malignant progression of GC cells (20). Another study demonstrated that circ_0006089 knockdown could inhibit polypyrimidine tract-binding protein 3 (PTBP3) expression by increasing miR-143-3p expression, which subsequently inhibited GC cell proliferation (37). Similarly, circFOXO3 can directly interact with miR-143-3p and negatively regulate its expression, consequently upregulating ubiquitin-specific peptidase 44 (USP44) expression, ultimately facilitating GC cell proliferation and migration (38).

Colorectal cancer (CRC). It is estimated that CRC accounted for ~1/10 cancer cases and related deaths in 2020 (1). There is some evidence suggesting that miR-143-3p has great potential for predicting the prognosis of patients with CRC (39,40). Recent research has also suggested that miR-143-3p influences CRC cell development. In most cases, the expression level of miR-143-3p is low in CRC. MiR-143-3p can be targeted by circ-ACAP2, while frizzled class receptor 4 (FZD4) is the target of miR-143-3p in CRC cells. Circ-ACAP2 induces FZD4 expression through interaction with miR-143-3p, thereby promoting progression and radioresistance in CRC (41). miR-143-3p is also a target of the lncRNA TPMO-AS1. By targeting miR-143-3p, CRC cell invasion, proliferation and migration are influenced (42). In addition, LINC00908 acts as an oncogenic lncRNA that negatively regulates miR-143-3p. Kruppel-like factor 5 (KLF5) is a target of miR-143-3p. LINC00908 actively regulates KLF5 expression by secreting miR-143-3p, thereby modulating CRC cell apoptosis and proliferation (43). The Coiled-lncRNA Coil Domain Containing 144 N-Terminal-Like antisense 1 (CCDC144NL-AS1), a novel carcinogenic lncRNA, is highly expressed in the serum of patients with CRC. CCDC144NL-AS1 can act as a 'sponge' of hsa-miR-143-3p, thereby upregulating its target protein High Mobility Group AT-hook 2, and this interaction is correlated with tumor stage and size (44). On the other hand, miR-143-3p can also directly affect its target genes and participate in CRC progression. miR-143-3p plays a role as an anticancer gene by downregulating integrin alpha 6 (ITGA6)/ArfGAP with the SH3 domain and ankyrin repeat and PH domain 3 protein expression (45). When overexpressed, miR-143-3p can also directly target CTNND1 and suppress its expression, thereby suppressing CRC cell proliferation, migration and invasion (46).

Gallbladder carcinoma (GBC). GBC is the most common biliary malignancy and has a poor prognosis and a 5-year survival rate of ~5% (47). It has been previously reported that miR-143-3p can downregulate SPLGF expression by targeting ITGA6, thereby inhibiting tumor angiogenesis and GBC

Table I. Expression and role of microRNA-143-3p in cancers.

Cancer types	Expression level	Upstream factors	Targets	Functions	(Refs.)
LC	Downregulation	LncRNA-TMPO-AS1	CDK1	Promote survival and reduce apoptosis	(25)
NSCLC	Downregulation	LncRNA UCC	SOX5	Promote proliferation, migration and EMT	(24)
NSCLC	Downregulation	LINC00667	RRM2	Promote growth	(26)
NSCLC	Downregulation	CircT UBA1C		Promote proliferation, inhibit apoptosis	(27)
LUAD	Downregulation	CircSNK1G3	HOXA10	Promotes growth and metastasis	(19)
LUAD	Downregulation	LncRNA PCAT19		Promote migration and invasion	(28)
BM of LC	Upregulation		VASH1	Promote metastasis and enhance angiogenesis	(29)
HCC	Downregulation	Circ0003998	FOSL2	Promote EMT	(30)
HCC	Downregulation	LncRNA SOX2-OT	MSI2	Promote malignant progression	(31)
HCC	Downregulation	LncRNA MALAT1	ZEB1	Promote proliferation and invasion	(32)
HCC	Downregulation		lncRNA PSMG3-AS1	Promote proliferation	(33)
HCC	Downregulation		FGF1	Promote proliferation and invasion	(34)
GC	Downregulation	LncRNA CCDC144NL-AS1	MAP3K7	Promotes growth and metastasis	(35)
GC	Downregulation	LINC00200	SERPINE1	Promote proliferation, invasion and migration	(36)
GC	Downregulation	Circ_0006089	IGF1R	Promote proliferation, invasion and migration	(20)
GC	Downregulation	Circ_0006089	PTBP3	Promote proliferation, metastasis and inhibit apoptosis	(37)
GC	Downregulation	CircFOXO3	USP44	Promote proliferation and migration	(38)
CRC	Downregulation	Circ-ACAP2	FZD4	Promote proliferation, migration, invasion and radiation resistance, inhibit apoptosis	(41)
CRC	Downregulation	LncRNA TMPO-AS1		Promote proliferation, migration and invasion	(42)
CRC	Downregulation	LINC00908	KLF5	Promote proliferation	(43)
CRC	Downregulation	CCDC144NL-AS1	HMGA2	Correlated with tumor stage and size	(44)
CRC	Downregulation		ITGA6, ASAP3	Promote invasion and migration	(45)
CRC	Downregulation		CTNND1	Promote proliferation, migration and invasion	(46)
GBC	Downregulation		ITGA6	Promote proliferation and angiogenesis	(48)
GBC	Downregulation	LncRNA OIP5-AS1		Promote invasion, proliferation and migration	(49)
PDAC	Downregulation		KRAS	Promote proliferation, migration and invasion	(51)
Esophageal cancer	Downregulation	LncRNA CASC7	HK2	Promote proliferation	(52)
BC	Downregulation	LINC02470	SMAD3	Promote invasion	(21)
BC	Downregulation	LINC00511	PCMT1	Promote proliferation and migration	(53)
BC	Downregulation	circ_644	MSI2	Promote the malignant progression	(54)
BC	Downregulation	LncRNA SNHG1	HK2	Promote proliferation, migration and invasion	(55)
BC	Downregulation	LncRNA MAFG-AS1	COX-2	Promote proliferation, metastasis and invasion	(56)
BC	Downregulation	LncRNA PCAT6	PCAT6	Promote proliferation, migration and invasion	(57)
PCa	Downregulation	LncRNA-SChLAP1	DNMT3A	Promote survival, migration, and tumorigenicity	(58)

Table I. Continued.

Cancer types	Expression level	Upstream factors	Targets	Functions	(Refs.)
PCa	Downregulation	LncRNA-PVT1	NOP2	Promote transfer and progress	(59)
PCa	Downregulation	LncRNA PCSEAT	EZH2	Promote proliferation	(60)
PCa	Downregulation		AKT1	Promote EMT	(61)
CRPC	Downregulation	GR	JAG1, NOTCH2	Promote migration, invasion, proliferation and inhibit apoptosis	(62)
RCC		LncRNA SARCC	AKT, MMP-13, K-RAS, P-ERK	Inhibit invasion, migration and proliferation	(64)
ccRCC	Downregulation	LINC00667	ZEB1	Promote progression of ccRCC and induce chemotherapy resistance	(11)
RCC	Downregulation	LncRNA SLC16A1-AS1	SLC7A11	Promote survival, proliferation and migration	(65)
CC	Downregulation	LncRNA OIP5-AS1	SMAD3	Promote migration and invasion	(66)
CC	Downregulation	LncRNA OIP5-AS1	ITGA6	Promote proliferation and invasion	(67)
CC	Down regulation	LncRNA OIP5-AS1	ROCK1	Promote proliferation and inhibit apoptosis	(68)
CC	Downregulation	LncRNA MEOX2-AS1	VDAC1	Promote proliferation and metastasis	(22)
CC	Downregulation	LncRNA ACTA2-AS1	SMAD3	Promote proliferation, migration and inhibit apoptosis	(69)
CC	Downregulation	LncRNA HOTAIR	BCL2	Promote proliferation and inhibit apoptosis	(70)
CC	Downregulation	LncRNA TMPO-AS1	ZEB1	Promote proliferation, migration and invasion	(71)
CC	Downregulation		CDK1	Promote proliferation, migration and invasion	(72)
OC	Downregulation		RALBP1	Promote survival and inhibit apoptosis	(76)
OC	Downregulation		TAK1	Promote proliferation, migration and invasion	(77)
OC	Downregulation	LncRNA PCAT6	TAK1	Promote proliferation, migration and invasion	(78)
OC	Downregulation	LncRNA MALAT1	CMPK	Promote migration and invasion	(79)
OC	Downregulation	LncRNA CDKN2B-AS1	SMAD3	Promote growth, invasion and migration	(80)
Endometrial cancer	Downregulation	LNCRNA UCA1	FKLF5, RXFP1	Promote proliferation, invasion and inhibit apoptosis	(81)
TC	Downregulation		MSI2	Promote proliferation, invasion, migration and inhibit apoptosis	(23)
TC	Downregulation	LINC01296	MSI2	Promote proliferation, invasion, migration and inhibit apoptosis	(82)
LSCC	Downregulation		k-Ras	Promote growth, migration and invasion	(83)
LSCC	Downregulation		MAGE-A9	Promote proliferation, migration and invasion	(84)
NSCC	Downregulation		Bcl-2, IGF1R	Promote proliferation, migration and inhibit apoptosis	(85)
BC	Downregulation		MYBL2	Promote proliferation and inhibit apoptosis	(86)
BC	Downregulation		MAPK7	Promote proliferation and migration	(87)
Triple-negative BC	Downregulation		LIMK1	Promote proliferation	(88)

Table I. Continued.

Cancer types	Expression level	Upstream factors	Targets	Functions	(Refs.)
BC	Downregulation	lncRNA PSMG3-AS1	COL1A1	Promote proliferation and migration	(89)
BC	Downregulation	lncRNA LOXL1-AS1		Promote proliferation, migration, invasion and inhibit apoptosis	(90)
Neuroblastoma	Downregulation	CircRNA-ACAP2	HK2	Promote migration, invasion and inhibit apoptosis	(91)
AML	Downregulation		KAT6A	Promote survival and proliferation	(92)
OS	Downregulation		MAPK7	Promote proliferation, migration and invasion	(93)
OS	Downregulation		FOSL2	Promote proliferation, metastasis and inhibit apoptosis	(94)
OS	Downregulation	lncRNA PCAT6	ZEB1	Promote proliferation, migration and invasion	(95)
Melanoma	Downregulation		COX-2	Promote proliferation and inhibit apoptosis	(12)
Melanoma	Downregulation	Circ-FOXM1	FLOT2	Promote proliferation, invasion and inhibit apoptosis	(96)
OSCC	Down regulation	lncRNA UCA1	MYO6	Promote growth, migration and invasion	(97)
OSCC	Downregulation	CircPVT1	SLC7A11	Promote growth and metastasis	(98)
OSCC	Downregulation	LncRNA MALAT1	MAGEA9	Promote proliferation and migration	(99)

LC, lung cancer; NSCLC, non-small cell lung cancer; LUAD, lung adenocarcinoma; HCC, hepatocellular carcinoma; GC, gastric cancer; CRC, colorectal cancer; GBC, gallbladder carcinoma; PDAC, pancreatic ductal adenocarcinoma; BC, bladder cancer; Pca, prostate cancer; CRPC, castration resistant prostate cancer; RCC, renal cell carcinoma; CC, cervical cancer; OC, Ovarian cancer; TC, thyroid cancer; LSCC, laryngeal squamous cell carcinoma; BC, breast cancer; AML, acute myeloid leukemia; OS, osteosarcoma; OSCC, oral squamous cell carcinoma; LncRNA, long non-coding RNA; CDK1, cyclin-dependent kinase 1; TMPO-AS1, TMPO antisense RNA 1; RRM2, Ribonucleotide reductase M2 subunit; HOXA10, homeobox (HOX) A10; VASH1, vasohibin-1; FGF1, fibroblast growth factor 1; SERPINE1, Serpin Family E Member 1; IGF1R, insulin-like growth factor 1 receptor; PTBP3, polypyrimidine tract-binding protein 3; USP44, ubiquitin-specific peptidase 44; FZD4, frizzled class receptor 4; ITGA6, integrin alpha 6; ASAP3, ArfGAP with the SH3 domain and ankyrin repeat and PH domain 3; HK2, hexokinase 2; COX-2, Cyclooxygenase-2; PCAT6, prostate cancer-associated transcript 6; PVT1, Plasmacytoma variant translocation 1; EZH2, enhancer of zeste homolog 2; JAG1, Jagged1; SLC7A11, solute carrier family 7 membrane 11; OIP5-AS1, OPA-interacting protein 5 antisense transcript 1; VDAC1, voltage-dependent anion channel 1; HOTAIR, HOX transcript antisense RNA; ZEB1, zinc finger E-box binding homeobox 1; RALBP1, RalA-binding protein 1; TAK1, transforming growth factor (TGF)-β-activated kinase 1; MALAT1, Metastasis associated lung adenocarcinoma transcript 1; CDKN2B-AS1, cyclin-dependent kinase inhibitor 2B antisense RNA 1; UCA1, urothelial carcinoma associated 1; KLF5, Kruppel-like factor 5; RXFP1, relaxin like family peptide receptor 1; MSI2, Musashi RNA binding protein 2; MAGE-A9, melanoma-associated antigen-A9; MYBL2, MYB proto-oncogene like 2; MAPK7, mitogen activated protein kinase 7; LIMK1, LIM kinase-1; COL1A1, collagen type 1 alpha 1; KAT6A, Lysine acetyltransferase 6A; FOSL2, FOS-Like antigen 2; FLOT2, Flotillin 2; MYO6, Myosin VI; circPVT1, circRNA plasmacytoma variant translocation 1 (PVT1); MAGEA9, MAGE Family Member A9; CCDC144NL-AS1, Coiled-lncRNA Coil Domain Containing 144 N-Terminal-Like antisense 1; HMGA2, High Mobility Group AT-hook 2; circ_644, hsa_circ_0000644; EMT, epithelial-mesenchymal transition; ccRCC, clear cell renal cell carcinoma.

growth (48). In addition, miR-143-3p is a target of the lncRNA OIP5-AS1, which regulates GBC cell biological function by mediating miR-143-3p expression (49).

Pancreatic cancer. The prognosis of pancreatic cancer is very poor, and the 5-year survival rate is low, at 3-5% (50). It is often diagnosed in the advanced stage and responds poorly to treatment. Therefore, additional studies are needed to identify effective treatments for pancreatic cancer. Research has shown that miR-143-3p expression is downregulated in pancreatic ductal adenocarcinoma (PDAC). miR-143-3p targets KRAS to inhibit PDAC tumorigenesis (51).

Esophageal cancer. Esophageal cancer is a common tumor with a very poor prognosis and an extremely high mortality rate. According to 2020 statistics, it ranks sixth overall in terms of the global cancer mortality rate (1). It has been recently proposed that miR-143-3p plays a crucial role in modulating esophageal cancer cell proliferation. miR-143-3p can interact with the lncRNA CASC7 to regulate hexokinase 2 (HK2) expression to affect tumor glycolysis and cell proliferation (52).

miR-143-3p in the urinary system

BC. BC is the 10th most common cancer worldwide and is more common in men than in women (1). Research has shown that miR-143-3p is modulated mainly by lncRNAs in BC. Among them, LINC02470 is upregulated in BC cells to accelerate their invasion. LINC02470 inhibits miR-143-3p to induce SMAD3 expression. Subsequently, SMAD3 activates TGF-β-associated EMT processes, ultimately exacerbating the malignant behavior of BC cells (21). LINC00511 is also upregulated in BC. LINC00511 downregulation inhibits tumor growth *in vivo*, and LINC00511 competes with miR-143-3p and inhibits its expression. LINC00511 targets miR-143-3p/PCMT1 in addition to modulating BC cell migration, apoptosis and proliferation and facilitating BC cell occurrence and development (53). hsa_circ_0000644 (circ_644) also acts as a sponge of miR-143-3p in BC, upregulating the expression of MSI2 and promoting the malignant progression of cancer cells (54). In addition, the lncRNA SNHG1 inhibits miR-143-3p expression in BC cells. HK2 is a target of miR-143-3p. SNHG1 promotes BC cell migration, invasion and proliferation by repressing miR-143-3p and inducing HK2 expression (55). Another study showed that the lncRNA MFG-AS1 targets miR-143-3p to inhibit its expression, and Cyclooxygenase-2 (COX-2) is a target of miR-143-3p. The lncRNA MFG-AS1 can promote BC development by regulating the miR-143-3p/COX-2 axis (56). The lncRNA prostate cancer-associated transcript 6 (PCAT6) can also play a carcinogenic role in BC by repressing miR-143-3p expression and upregulating PDIA6 expression (57).

Prostate cancer (PCa). PCa is estimated to be the second most common cancer in men in China and is the most frequently diagnosed cancer in men in most other countries (1). In PCa, miR-143-3p is suppressed by lncRNA-Schlap1, and DNMT3a is a target of miR-143-3p and mediates the oncogenic effect of SChLAP1 in the development of PCa (58). In addition, the lncRNA Plasmacytoma variant translocation 1 enhances NOP2 expression by downregulating miR-143-3p, promoting the metastasis and progression of PCa (59). Similarly, the lncRNA PCSEAT facilitates PCa cell proliferation by competitively binding to miR-143-3p and inhibiting its expression, sequentially regulating enhancer of zeste homolog 2 expression (60). In addition, the overexpression of miR-143-3p in PCa may inhibit EMT by targeting AKT1 (61). Another study showed that the glucocorticoid receptor (GR), which downregulates miR-143-3p expression, is upregulated in castration-resistant PCa (CRPC) cells. miR-143-3p can target Jagged1 (JAG1) and NOTCH2. GR accelerates the malignant progression of CRPC through downregulating miR-143-3p expression and upregulating JAG1/NOTCH2 expression (62).

Renal cell carcinoma (RCC). RCC is considered to be one of the most aggressive malignant tumors; it accounts for ~3% of all cancers and is the main type of renal malignancy (63). In RCC, miR-143-3p is regulated mainly by lncRNAs. lncRNA-SARCC binds to and induces the degradation of the androgen receptor (AR) protein, inhibits AR function, and contributes to the increased expression of miR-143-3p, which inhibits downstream signaling molecules, such as K-RAS, P-ERK, AKT and MMP-13; thus, lncRNA-SARCC plays a role in inhibiting RCC progression (64). LINC00667 is upregulated in clear cell RCC (ccRCC) and enhances ZEB1 expression by targeting miR-143-3p, thereby accelerating the progression of ccRCC and inducing chemotherapy resistance (11). In addition, the lncRNA SLC16A1-AS1 is highly expressed in RCC and can induce ferroptosis, which is a form of programmed cell death that has antitumor effects on numerous cancers. SLC16A1-AS1 can also inhibit miR-143-3p expression. Solute carrier family 7 membrane 11 (SLC7A11) is a target of miR-143-3p. In brief, silencing of the lncRNA SLC16A1-AS1 induces ferroptosis via miR-143-3p/SLC7A11 signal transduction, which decreases the viability and suppresses the migration and proliferation of RCC cells (65).

miR-143-3p in the reproductive system

CC. CC is the fourth most common cancer in women. While there are highly effective primary and secondary prevention measures, including the HPV vaccine and screening, vaccination and CC screening are unavailable in some low-income countries (1). It is therefore necessary to find more effective treatments. It has commonly been assumed that miR-143-3p interacts with lncRNAs to modulate target expression levels to play a part in CC. In CC, the expression of the lncRNA OIP5-AS1 is markedly enhanced, which is associated with progression. lncRNA OIP5-AS1 may induce SMAD3, ITGA6 and ROCK1 expression by suppressing miR-143-3p expression, subsequently accelerating the invasion and migration of CC cells (66-68). In addition, the lncRNA MeOX2-AS1 is highly expressed in CC and can act as a sponge for miR-143-3p. MeOX2-AS1 knockdown inhibits the progression of CC by inhibiting voltage-dependent anion channel 1 expression through miR-143-3p (22). The lncRNA ACTA2-AS1 can also upregulate SMAD3 by acting as a ceRNA of miR-143-3p, thereby accelerating CC progression (69). Another study proposed that the lncRNA HOTAIR was upregulated in CC, while miR-143-3p was downregulated. HOTAIR facilitates the growth of CC cells by regulating BCL2 through miR-143-3p (70). The lncRNA TMPO-AS1 is also highly expressed in CC cells. TMPO-AS1 acts as a sponge of miR-143-3p to suppress its expression to increase ZEB1 expression, ultimately facilitating the migration, invasion and proliferation of CC cells (71). On the other hand, miR-143-3p overexpression can also repress

the migration, invasion and proliferation of CC cells by inhibiting CDK1 (72).

Ovarian cancer (OC). OC is the most lethal gynecologic malignancy, and the most common subtype is epithelial OC (EOC). The lack of specific symptoms prevents early detection of the disease, and most confirmed cases are diagnosed at an advanced stage. Therefore, biomarkers for the early diagnosis of OC are urgently needed (73). In malignant ovarian tumors, miR-143-3p is downregulated, and cystatin B (CSTB) is overexpressed. Transforming growth factor-β (TGF-β) is an essential cytokine that promotes or inhibits tumor growth. TGF-β1 regulates miR-143-3p so that it can directly bind to the CSTB 3'-UTR, resulting in a reduction in CSTB expression in OC cells, and knockdown of CSTB leads to a reduction in OC cell proliferation (74). In addition, loss of TGF-β reactivity may lead to downregulation of miR-143-3p, which consequently leads to upregulation of PNPO and accelerates the migration and invasion of EOC cells (73). The ribosomal protein L10 (RPL10) is upregulated in human ovarian malignancies. Although RPL10 is not targeted by miR-143-3p, its expression is modulated by this miRNA. miR-143-3p inhibits RPL10 expression, consequently decreasing the viability, suppressing the invasion and migration, and inducing the apoptosis of EOC cells (75). RalA-binding protein 1 (RALBP1) is a target gene of miR-143-3p, and the downregulation of miR-143-3p leads to increased expression of RALBP1, thereby promoting the occurrence of OC (76). Other evidence has demonstrated that the lncRNA PCAT6, as a ceRNA, regulates the TGF-β-activated kinase 1 (TAK1) expression by binding to miR-143-3p. miR-143-3p downregulates the expression of TAK1 and inhibits invasion, proliferation and migration in OC cells. Subsequent rescue assays also confirmed that upregulation of miR-143-3p reduced invasion, proliferation and migration induced by PCAT6 overexpression (77,78). Dual-luciferase reporter assays revealed that the lncRNA MALAT1 interacts with miR-143-3p, and that a low MALAT1 level leads to increased miR-143-3p expression, which in turn leads to decreased CMPK protein expression and suppressed migration and invasion in EOC (79). In addition, the lncRNA cyclin-dependent kinase inhibitor 2B antisense RNA 1 (CDKN2B-AS1) is highly expressed in OC and is positively related to the malignant progression of OC. The lncRNA CDKN2B-AS1 is a molecular sponge of miR-143-3p; its binding results in the de-repression of the expression of miR-143-3p, which targets SMAD3 and ultimately triggers OC progression (80).

Endometrial cancer. Endometrial cancer, the sixth most common cancer in women, led to 97,000 deaths worldwide in 2020 (1). It has been previously suggested that miR-143-3p interacts with the lncRNA urothelial carcinoma associated 1 (UCA1), and that the lncRNA UCA1 is highly expressed in endometrial cancer. The upregulated expression of UCA1 is associated with endometrial cancer progression and deterioration of the disease. UCA1 may upregulate KLF5 and relaxin family peptide receptor 1 expression by sponging miR-143-3p, thereby promoting the development of endometrial cancer (81). However, additional studies are necessary to verify the effect of miR-143-3p in endometrial cancer.

miR-143-3p in other malignant tumors. In addition to its role in other cancers, miR-143-3p is often downregulated in numerous other cancers and affects tumor progression. miR-143-3p is expressed at low levels in TC and can interact with LINC01296. Silencing LINC01296 promoted miR-143-3p expression. MSI2 is targeted by miR-143-3p. Silencing LINC01296 can repress the development of TC by suppressing its competitive binding with miR-143-3p and inhibiting MSI2 (82). MiR-143-3p can also directly target MSI2 in papillary thyroid carcinoma (PTC). Upregulation of miR-143-3p represses cell growth, invasion and migration by downregulating MSI2, thereby inhibiting the progression of PTC (23). miR-143-3p expression is downregulated in laryngeal squamous cell carcinoma (LSCC). K-Ras is a target of miR-143-3p in LSCC cells. miR-143-3p can suppress the growth, invasion and migration of LSCC cells by inhibiting K-Ras (83). MAGE-A9 is also a target of miR-143-3p, and downregulation of miR-143-3p contributes to LSCC progression by upregulating MAGE-A9 (84). In nasal SCC, miR-143-3p has an obvious tumor suppressive effect via negatively regulating Bcl-2 and IGF1R (85). In breast cancer, miR-143-3p is downregulated. miR-143-3p targets MYB proto-oncogene like 2 (MYBL2) and mitogen activated protein kinase 7 (MAPK7) and inhibits the expression of MYBL2 and MAPK7, sequentially accelerating proliferation and inhibiting breast cancer cell apoptosis (86,87). miR-143-3p can also inhibit LIMK1 expression to accelerate triple-negative breast cancer cell proliferation (88). In addition, the lncRNA PSMG3-AS1 modulates COL1A1 expression by repressing miR-143-3p, thereby affecting breast cancer development (89). The lncRNA LOXL1-AS1 affects breast cancer cell invasion, proliferation and migration by directly targeting miR-143-3p (90).

In addition to its role in these cancers, miR-143-3p also affects the progression of neuroblastoma, acute myeloid leukemia (AML), osteosarcoma (OS), melanoma and oral SCC (12,91-99).

4. Mechanism of miR-143-3p

According to previous studies (13,21,22,41,45), it is considered by numerous scholars that the mechanisms of action of miR-143-3p include the following: i) LncRNAs, circRNAs and cytokines interact with miR-143-3p to regulate its expression, thereby affecting its target gene expression; ii) miR-143-3p directly targets relevant target genes and plays a part in regulating the expression of target genes; and iii) it regulates tumor growth by affecting signaling pathways. The first two mechanisms of action have been aforementioned in various systemic cancers, and the associated signaling pathways are demonstrated in Fig. 2.

PI3K/Akt signaling pathway. The PI3K/Akt signaling pathway is a crucial signaling pathway in a variety of cancer types. It modulates tumor cell survival, metastasis and metabolism (99). Exosomal miR-143-3p derived from G-MDSCs in LC tissues promotes cancer progression by targeting ITM2B to stimulate the PI3K/Akt pathway (13). In addition, circ_0006089 knockdown suppresses PTBP3 expression and the PI3K/AKT pathway by increasing miR-143-3p expression, consequently repressing the proliferation of GC cells (37). In GBC, ITGA6 is upregulated and negatively associated with

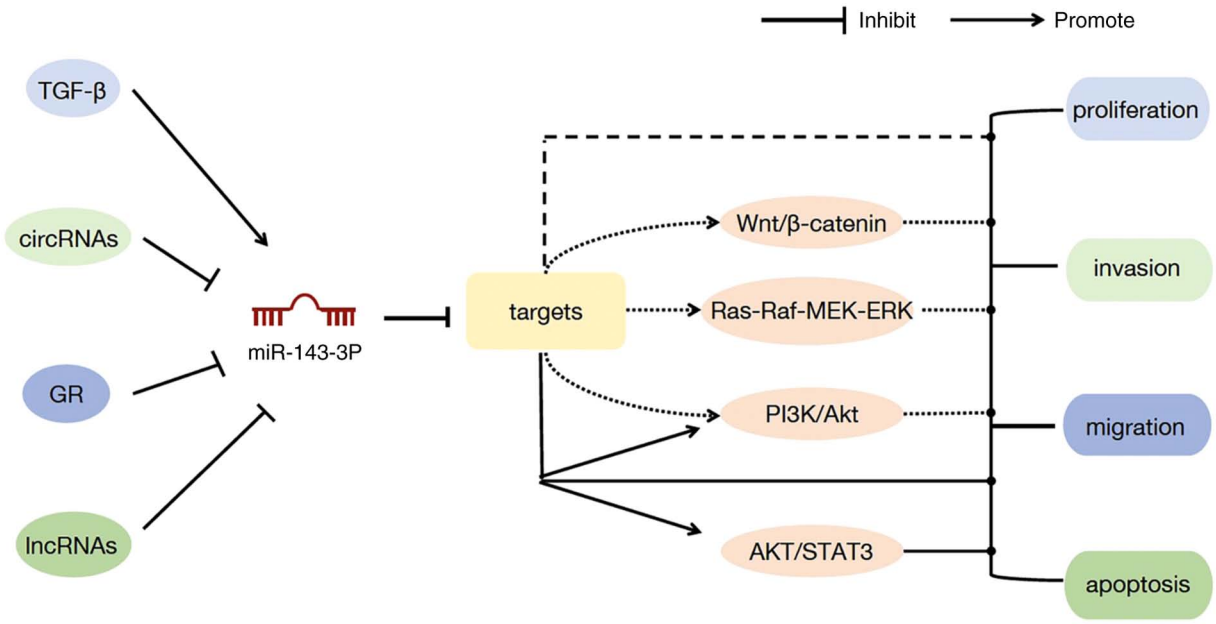

Figure 2. Mechanism of miR-143-3p. Solid line pathway: i) LncRNAs, circRNAs, GR and TGF-β upregulate/downregulate miR-143-3p levels to suppress the level of target genes (pink), and related target genes regulate tumor proliferation, invasion, migration and apoptosis by activating relevant signaling pathways (yellow) or directly. ii) Dashed pathway: miR-143-3p directly represses the expression of target genes to regulate tumor progression. iii) Punctate pathway: MiR-143-3p plays a corresponding part in tumors by inhibiting target genes to activate relevant signaling pathways. lncRNAs, long non-coding RNAs; circRNAs, circular RNAs; GR, glucocorticoid receptor; TGF-β, transforming growth factor-β.

miR-143-3p. miR-143-3p downregulates PLGF expression via the ITGA6/PI3K/AKT/STAT3 pathway, thereby inhibiting tumor angiogenesis and GBC growth (48).

Wnt/β-catenin signaling pathway. The Wnt/β-catenin signaling pathway comprises four parts: The membrane segment, the extracellular signaling pathway, the cytoplasmic segment, and the extracellular signaling pathway. These pathways are regulated mainly by Wnt proteins, which include Wnt1, Wnt3a and Wnt5a. A variety of serious diseases, including encompassing cancer and non-cancer diseases, often result from dysregulation of Wnt/β-catenin signaling (100). It has been suggested that FZD4 can participate in activating the Wnt/β-catenin pathway. In CRC, FZD4 is a target of miR-143-3p, and the accumulation of FZD4 or downregulation of miR-143-3p can induce the Wnt/β-catenin pathway to participate in the modulation of CRC progression and radioresistance (41).

AKT/STAT3 signaling pathway. The AKT/STAT3 signaling pathway has an essential function in the development of cancer and can enhance the antitumor immune response by modulating PD-L1 expression in cancer cells with abnormal EGFR activity (101). In addition, MACC1 can influence CC cell invasion, migration, apoptosis and cancer stemness by modulating the AKT/STAT3 pathway (102). In TC, LINC01296 can repress miR-143-3p expression through competitive binding with miR-143-3p, and downregulation of miR-143-3p can induce MSI2 and subsequently activate the AKT/STAT3 signaling pathway to promote the development of TC (82).

Ras-Raf-MEK-ERK signaling pathway. The Ras-Raf-MEK-ERK signaling pathway is the most well-studied MAPK pathway and has important functions in cell proliferation, survival, differentiation and development (103). In LSCC, K-Ras is the target of miR-143-3p. Downregulation of miR-143-3p induces K-ras expression and subsequently activates the K-ras/Raf/MEK/ERK signaling pathway to promote cell growth, invasion and migration in LSCC (83).

5. Potential application of miR-143-3p in future practice

As a diagnostic marker. Numerous studies (12,13,28,39,40) have revealed that miRNAs may serve as potential biomarkers in cancer. Among them, miR-143-3p inhibited human melanoma cell proliferation and facilitated their apoptosis, demonstrating that miR-143-3p may be a novel marker for early diagnosis (12). In LUAD, the lncRNA PCAT19 regulates invasion, proliferation and migration and may be an underlying diagnostic biomarker of LUAD. miR-143-3p is the target of lncRNA PCAT19, therefore miR-143-3p may also be a useful diagnostic marker of LUAD (28). On the other hand, miR-143-3p has been found to suppress OC cell invasion, migration and proliferation by targeting TAK1 *in vitro* and to inhibit ovarian tumorigenicity *in vivo*. These findings revealed that the miR-143-3p-TAK1 pathway has promising application potential in the clinical diagnosis of OC (77).

As a prognostic marker. Recent research has identified that miR-143-3p may be useful as a prognostic marker in cancer. In LUAD, the lncRNA PCAT19 is a self-controlled prognostic factor that can modulate LUAD cell proliferation, invasion and migration. miR-143-3p is the target of lncRNA PCAT19, thus miR-143-3p may also predict the prognosis of patients with LUAD (28). The lncRNA SOx2-OT is highly expressed in HCC and is positively associated with poor prognosis in patients with HCC. However, the lncRNA SOx2-OT negatively modulates miR-143-3p expression. Therefore, miR-143-3p may also

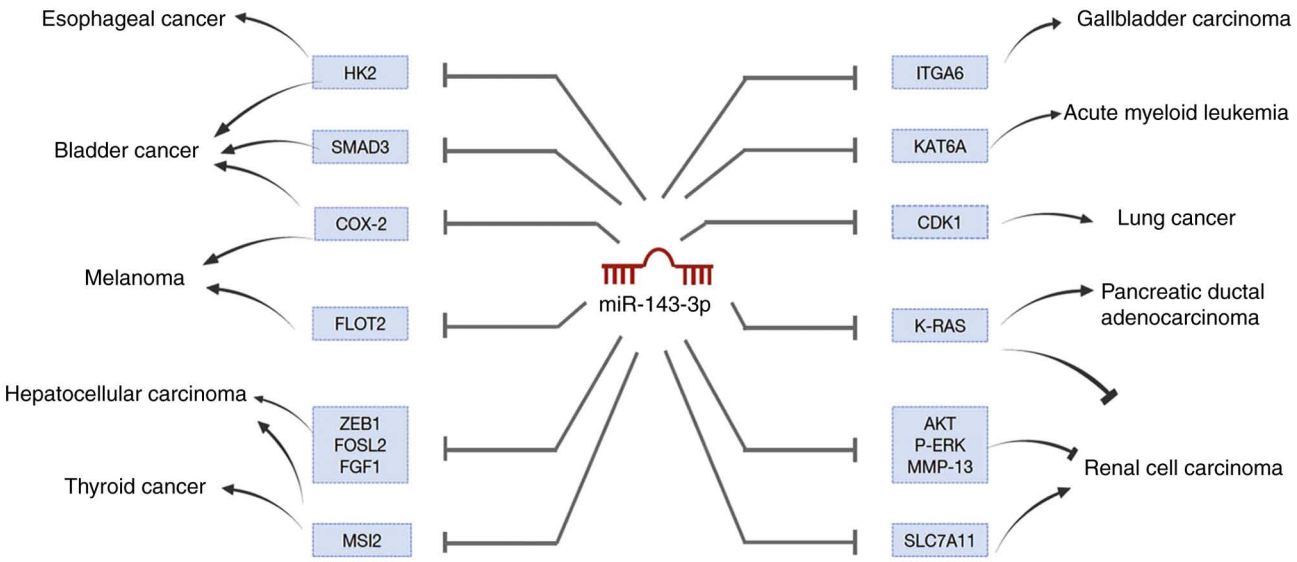

Figure 3. MiR-143-3p targets a large number of genes to inhibit or promote cancers' growth, including esophageal cancer, bladder cancer, melanoma, hepatocellular carcinoma, thyroid cancer, gallbladder carcinoma, acute myeloid leukemia, lung cancer, pancreatic ductal adenocarcinoma and renal cell carcinoma. miR, microRNA; CDK1, cyclin-dependent kinase 1; FGF1, fibroblast growth factor 1; ITGA6, integrin alpha 6; HK2, hexokinase 2; COX-2, Cyclooxygenase-2; SLC7A11, solute carrier family 7 membrane 11; ZEB1, zinc finger E-box binding homeobox 1; MSI2, Musashi RNA binding protein 2; KAT6A, lysine acetyltransferase 6A; FOSL2, FOS-Like antigen 2; FLOT2, Flotillin 2.

be related to the prognosis of patients with HCC (31). In addition, according to a 2-cohort study of 254 pretreated patients with rectal adenocarcinoma, miR-143-3p was overexpressed in tumors and was more highly expressed in stage II or III tumors than in stage I tumors (39). Another study proposed that the miR-143-3p expression level was greater in patients diagnosed with CRC aged >60 years than in those diagnosed with CRC aged <60 years (P=0.048), and the miR-143-3p level was greater in non-mucinous tumor subtypes than in mucinous tumor subtypes (P=0.025), indicating that miR-143-3p is highly expressed in patients diagnosed with CRC who are <60 years old (P=0.048). MiR-143-3p expression is associated with the age at diagnosis, pathological type and tumor stage of patients with CRC and will be helpful for early screening of young and elderly patients with CRC and for more accurate evaluation of patient prognosis (40). Another study reported that miR-143-3p is significantly downregulated in GBC, and this downregulation is associated with a poor prognosis in patients with GBC (48). Low miR-143-3p expression is also associated with more advanced clinical stages of LSCC and with a decreased overall survival rate (84). In summary, miR-143-3p can be used as a prognostic marker in multiple tumors.

As a therapeutic target. In a variety of cancers, miR-143-3p regulates tumor progression by modulating the expression of target genes. In melanoma, miR-143-3p targets COX-2 to repress proliferation and promote apoptosis in human melanoma cells (12). Exosomes of miR-143-3p derived from G-MDSCs in LC promote the proliferation of LC by targeting ITM2B in LC (13). In HCC, miR-143-3p represses HCC cell invasion and proliferation by influencing its target gene FGF1 (34). In CRC, miR-143-3p directly targets CTNND1 to affect cell invasion, proliferation and migration (46). In OC, miR-143-3p downregulates TAK1 expression and suppresses OC cell invasion, migration and proliferation *in vitro* (77). Moreover, miR-143-3p also has a similar role in AML, breast cancer and OS. These findings are sufficient to indicate that miR-143-3p is a potential therapeutic target in cancer.

6. Discussion

At present, diagnostic markers and treatment methods for cancer are still hot topics in the field of cancer research, and difficult problems are being gradually solved. In recent years, miRNAs have been found to play important roles in tumorigenesis and development. Among them, miR-143-3p is upregulated in LC with BMs and downregulated in numerous tumors; moreover, it can act as an oncogene to affect tumor progression in most cases. miR-143-3p has dual effects on RCC through its target genes but plays only positive roles in other cancers (Fig. 3) Moreover, miR-143-3p represents a favorable biomarker with great potential for early diagnosis, treatment and prognosis evaluation in patients with cancer. Moreover, it was discovered that miR-143-3p expression is modulated by upstream signaling factors; subsequently miR-143-3p acts on target genes to affect tumor cell invasion, migration, proliferation and apoptosis. Mechanistically, miR-143-3p, an upstream signaling factor, can act on different target genes in the same cancer (20,37). Different cancers can be regulated by an identical upstream signaling factor (25,42). After interacting with miR-143-3p, different upstream signaling factors can also target the same genes (52,55). The upstream signaling factor-miR-143-3p-target gene network is complex. Although miR-143-3p can directly inhibit target genes to affect tumor progression, it is likely that there are numerous upstream regulatory factors that have not been discovered, and the discovery of these upstream regulatory factors will be highly important for miR-143-3p-targeted cancer treatment. In addition, there are few studies on the signaling pathways associated with the effect of miR-143-3p, and

further exploration of the potential mechanism is needed, as this information will lead to additional ideas for cancer treatments related to miR-143-3p.

Furthermore, in 2015, researchers demonstrated that the exosomes of bone marrow mesenchymal stem cells (BMSCs) contained high levels of miR-143-3p (104), and the expression of miR-143-3p in hMSC exosomes was also significantly increased; these exosomes could promote the apoptosis of pancreatic cancer cells and suppress cell growth and invasion (50). In addition, MSC-derived exosomes are closely related to drug therapy, molecular targeted therapy, radiotherapy and chemotherapy for cancer (105). However, there is little research on miR-143-3p in exosomes at present, and numerous future studies are urgently needed to prove its importance in clinical practice.

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Availability of data and materials

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Authors' contributions

JW acquired the data and wrote the manuscript. YZ conceived the study. DL, QC and CB contributed to the revisions. All authors read and approved the final manuscript. Data authentication is not applicable.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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