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Review

A review on the role of MYC-induced long non-coding RNA in human disorders

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ABSTRACT

MINCR (MYC-Induced Long Non-Coding RNA) is classified as an lncRNA. It has a significant correlation with MYC gene. MINCR has important roles in the carcinogenesis. It has been approved that this lncRNA can act as molecular sponge for miR-28–5p, miR-708–5p, miR-876–5p and miR-146a-5p. Dysregulated levels of MINCR has been observed in different types of cancer, especially hepatocellular carcinoma. In addition to malignant conditions, schizophrenia and neurodegenerative diseases such as Alzheimer's disease and amyotrophic lateral sclerosis are associated with dysregulation of expression patterns of MINCR. This review outlines MINCR molecular mechanisms of action in different disorders.

1. Introduction

Long non-coding RNAs (lncRNAs) are a class of RNA that usually do not encode protein [9,31]. These molecules are longer than 200 bp in length, mostly transcribed by RNA POL II (exemptions are seen in plants), and may undergo polyadenylation and capping [11,31]. LncRNAs have important biological functions in the process of gene expression [10,27]. Whether it is at transcription or post-transcriptional level, or in a form of interaction with proteins, there is no doubt that these molecules can regulate enormous number of genes [8,24,25].

In recent years, it has been shown that if dysregulated, lncRNAs have adverse outcomes in the carcinogenesis and can contribute to malignancy in almost any type of cancer [13,14].

MYC-Induced Long Non-Coding RNA (MINCR) was firstly discovered by Doose and colleagues in 2015 in Burkitt lymphoma cells [6]. They have shown that after activation of MYC in hT-RPE-MycER and P493–6 cells, rise of MINCR levels occurs [6]. In addition, it has been approved that promoter region of MINCR is bound by MYC, and a significant correlation between MYC and MINCR exists [6].

According to NCBI, MINCR is located on 8q24.3 and is highly transcribed in fat tissue and thyroid gland. Based on ensembl, MINCR has 7 transcripts. Dysregulated level of MINCR has been approved in different cancers such as hepatocellular carcinoma, non-small cell lung cancer and colorectal cancer, alongside with non-malignant conditions like schizophrenia and Alzheimer's disease. This review summarizes involvement of MINCR in three different levels: cell lines studies, animal studies and finally clinical studies.

2. Role of MINCR in cancers

2.1. Cell line studies

As signified in its name, MINCR is in a close correlation with MYC gene. Regarding its role on the proliferation and cell growth, oncogenic role of MYC in different types of cancer has been approved [5]. In addition to its obvious correlation with MYC in Burkitt lymphoma, it has been shown that knockdown of MINCR in non-small lung cancer cell lines PC9, HCC827 and A549 contributes to the subsequent down-regulation of c-Myc resulting in reduced proliferation and apoptosis activation [3].

Wnt/β-catenin signaling pathway is of great importance in embryonic development and cell survival and proliferation [19]. First member

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of Wnt family was discovered in 1982 [21]. In an off-state situation, a complex is formed between AXIN, the APC protein, GSK-3β, CK-1α, and β-catenin. This complex inhibits this signaling pathway. Wnt binding to FZD and LRP5/6 receptors causes activation of pathway by inhibiting β-catenin phosphorylation [19]. Several studies support the correlation between MINCR and this signaling pathway, for instance in osteosarcoma cell lines, after forced inhibition of MINCR, activity levels of Wnt/β-catenin signaling diminishes and this phenomenon is associated with reduced proliferation, migration, invasion and induced apoptosis [1]. Identical molecular mechanism also applies for oral squamous cell carcinoma [20].

Similar to many lncRNAs, MINCR can act as molecular sponge and this sponging mechanism also can affect Wnt/β-catenin signaling pathway. For instance, in colorectal cancer cell lines SW620, HCT116, RKO and HT29, it has been demonstrated that MINCR sponges miR-708–5p. Interestingly, this miRNA targets 3′-UTR of CTNNB1, a gene responsible for β-catenin production. After knocking down MINCR, elevated levels of miR-708–5p causes CTNNB1 depletion and consequent inhibition of Wnt/β-catenin signaling pathway occurs [30]. Also, in hepatocellular carcinoma, depletion of MINCR causes miR-107 levels to rise and results in declined levels of β-catenin, which ultimately hampers malignant features of cells [17] (Fig. 1).

In summary, MINCR play an oncogenic role in different types of cancer and its downregulation is in favor of benign properties. Table 1 shows the role of MINCR in cancer cell lines.

3. Animal studies

Consistent with the observed oncogenic role of MINCR in cancer cell lines and its association with c-Myc, down-regulation of MINCR in cancer cells has been shown to reduce their tumorigenic potential in xenograft animal models. These experiments have been mostly performed in the BALB/c-nude mice (Table 2). Studies in animal models of osteosarcoma [1], colorectal cancer [30], glioma [18], non-small cell lung cancer [28] and gallbladder cancer [29] have verified this function of MINCR.

4. Studies in clinical samples

Over-expression of MINCR has been verified in a variety of tumoral tissues (Table 3). For instance, osteosarcoma have exhibited significant over-expression of MINCR [1]. Moreover, qRT-PCR assay has verified up-regulation of MINCR in oral squamous cell carcinoma tissues versus equivalent normal controls. More importantly, the overall survival rate of oral squamous cell carcinoma patients with high levels of MINCR has been lower than the other group as revealed by Kaplan-Meier analyses [20]. MINCR over-expression has also been detected in non-small cell carcinoma samples from TCGA datasets as well as another cohort of patients [3]. MINCR over- expression in this type of cancer has been closely related to poor survival of patients [28], emphasizing on the oncogenic role of MINCR.

Based on the results of qPCR, MINCR expression is also higher in hepatocellular cancer tissues than in adjacent tissues. Patients with high level of MINCR have exhibited lower overall survival rate compared with those with low level of MINCR [17].

MINCR has also been found to be markedly upregulated in gallbladder cancer tissues compared with adjacent normal tissues. High levels of MINCR in these tissues has been positively associated with

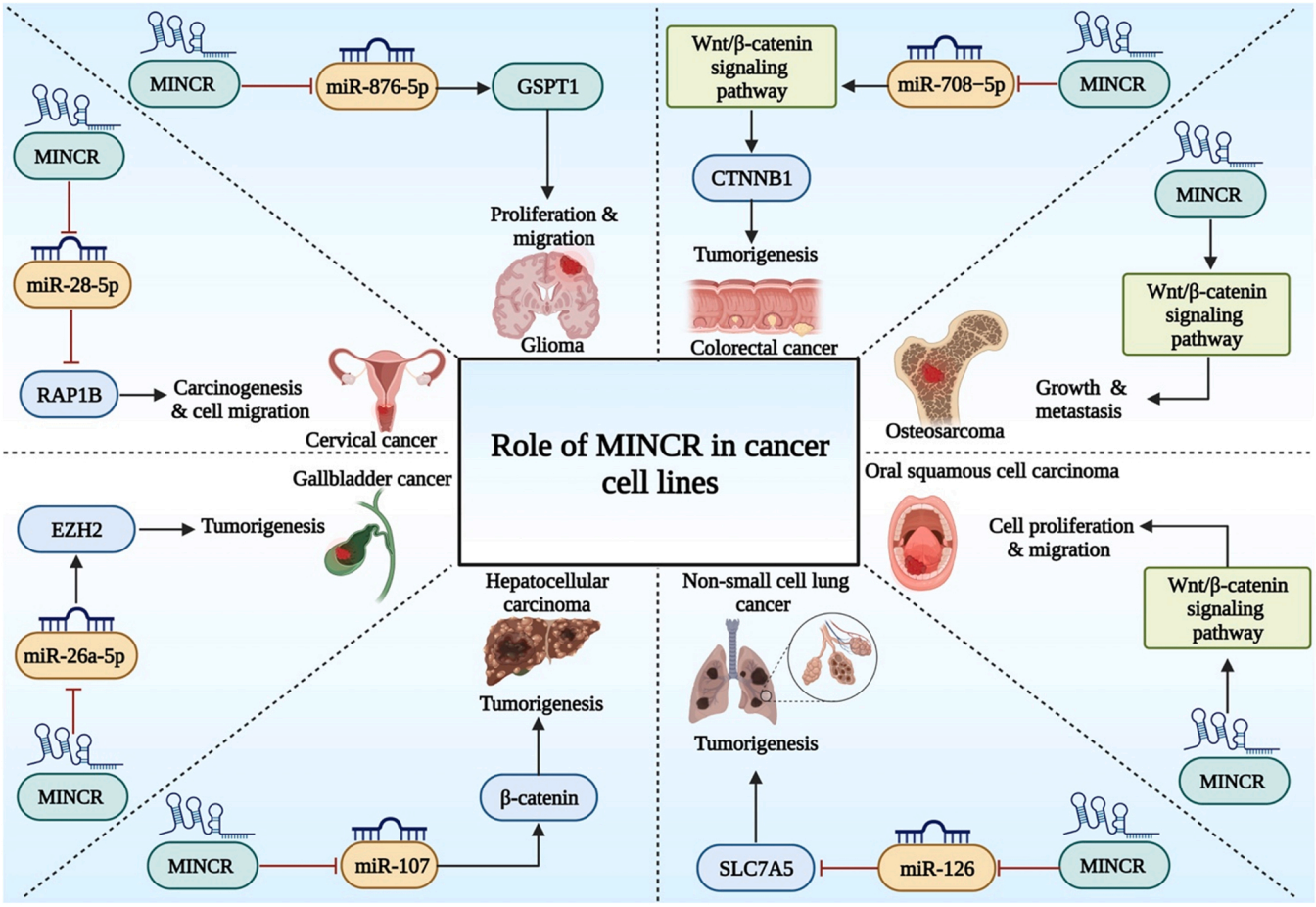

Fig. 1. Illustration of the different signaling pathways and the role of MINCR in cancer cell lines. MINCR can promote tumor cell proliferation, invasion, migration, and survival by targeting particular genes and sponging various types of miRs, such as miR-28–5p, miR-26a-5p, miR-107, miR-126, miR-708 − 5p, and miR-876–5p.

Table 1
Role of MINCR in cancer cell lines (Δ: knock-down or deletion, ↑: over-expression, →: results in, EMT: Epithelial-mesenchymal transition, HPV: human papilloma virus).

Tumor type	Targets/ Regulators and signaling pathways	Cell line	Function	References
Kidney renal clear cell carcinoma	-	786-O and 769-P	Higher expression of MINCR was observed in tumor cell lines	[12]
Osteosarcoma	Wnt/ β-catenin signaling pathway	HOS, MG-63 and Saos-2	Δ MINCR → ↓ Wnt/ β-catenin signaling pathway: ↓ proliferation ↑ apoptosis ↓ EMT process ↓ invasion ↓ migration	[1]
Cervical cancer	miR-28–5p/ RAP1B axis	C33-A cell transfected with E6 oncoprotein of HPV16	Δ MINCR (which acts as ceRNA for miR-28–5p) → ↑ miR-28–5p → ↓ RAP1B: ↓ migration	[23]
Colorectal cancer	miR-708–5p/ CTNNB1 axis | Wnt/ β-catenin signaling pathway	SW620, HCT116, RKO and HT29	Δ MINCR (which sponges miR-708–5p) → ↑ miR-708–5p → ↓ CTNNB1 → ↓ Wnt/β -catenin: ↓ proliferation ↓ EMT process ↓ invasion ↓ migration	[30]
Glioma	miR-876–5p/ GSPT1 axis	A172, U87, U251, and LN229	Δ MINCR (which sponges miR-876–5p) → ↑ miR-876–5p → ↓ GSPT1: ↓ proliferation ↓ invasion ↓ migration	[18]
Hepatocellular carcinoma	miR-107/ β-catenin axis	Bel-7402, HepG2, MHCC88H, SMMC-7221, Huh7 and Hep3B	Δ MINCR → ↑ miR-107 → ↓ β-catenin: ↓ proliferation ↑ apoptosis	[17]
	-	SMMC-7721, Huh7, HCC-LM3, HepG2, and MHCC-97 H	Δ MINCR: ↓ proliferation ↓ invasion ↓ migration	[2]
Non-small cell lung cancer	c-Myc	PC9, HCC827 and A549	Δ MINCR → ↓ c-Myc: ↓ proliferation ↑ cell cycle arrest ↑ apoptosis	[3]
	miR-126/ SLC7A5 axis	A549, H1299, H460, SPC-A1 and H1975w	Δ MINCR → ↑ miR-126 → ↓ SLC7A5: ↓ proliferation ↑ apoptosis ↓ migration	[28]
Oral squamous cell carcinoma	Wnt/ β-catenin	TSCCA, Tca8113 and SCC25	Δ MINCR → ↓ Wnt/ β-catenin: ↓ proliferation ↑ apoptosis ↓ invasion ↓ migration	[20]
Gallbladder	miR-26a-5p/EZH2 axis	NOZ and GBC-SD	Δ MINCR → ↑ miR-26a-5p → ↓ EZH2: ↓ proliferation ↑ apoptosis ↓ invasion ↓ EMT process	[29]

Table 2
Impact of MINCR in carcinogenesis in vivo (Δ: knock-down or deletion).

Tumor type	Animal models	Results	References
Osteosarcoma	BALB/c-nude mice	Δ MINCR: ↓ tumor weight ↓ tumor size	[1]
Colorectal cancer	BALB/c-nude mice	Δ MINCR: ↓ tumor weight ↓ tumor size	[30]
Glioma	Nude mice	Δ MINCR: ↓ tumor weight ↓ tumor size	[18]
Non-small cell lung cancer	BALB/c athymic nude mice	Δ MINCR: ↓ tumor growth	[28]
Gallbladder cancer	Nude mice	Δ MINCR: ↓ tumor growth ↓ tumor size	[29]

tumor volume and lymph node metastases and has been negatively correlated with overall survival [29]. Similarly, overexpression of MINCR in colorectal cancer tissues has been associated with the pathological stage of these patients [4].

5. Non-malignant conditions

While MINCR is upregulated in various cancers, it has been shown to be downregulated in amyotrophic lateral sclerosis patients. Pandini et al. have assessed expression levels of MINCR in SH-SY5Y cells using RNA-sequencing. Up-regulation of MINCR have caused huge changes in the expression of cancer-related genes and alterations in several essential processes, including cell cycle and growth factor signaling. In contrast, downregulation of MINCR has resulted in changes in the expression of a small quantity of genes contributing to various neurodegenerative disorders, particularly those being involved in RNA metabolism and inflammation [22]. In another study, Li et al. have demonstrated down-regulation of MINCR and BMPR2 whereas up-regulation of miR-146a-5p in osteoarthritis cartilage tissues compared with controls. This expression pattern has also been detected in IL-1β-induced chondrocytes versus normal chondrocytes. Functional experiments have shown that up-regulation of MINCR promotes cell proliferation and inhibits apoptosis and degeneration of extracellular matrix. MINCR has been demonstrated to bind with miR-146a-5p, a miRNA that targets BMPR2 (Fig. 2a). Mechanistically, MINCR silencing reverses the effect of miR-146a-5p under-expression in osteoarthritis. Up-regulation of miR-146a-5p also reverses the impact of BMPR2 up-regulation in these cells. Cumulatively, MINCR prevents progression

Table 3
Dysregulation of MINCR in clinical specimens (ANT: adjacent normal tissue, O-S: overall survival, TCGA: the cancer genome atlas).

Tumor type	Samples	Expression (tumor vs. normal)	Kaplan-Meier Analysis (MINCR dysregulation impact)	Univariate/ Multivariate cox regression	Association of MINCR expression with clinicopathologic characteristics	References
Osteosarcoma (OS)	91 OS + paired ANT	Upregulated	-	-	-	[1]
Colorectal cancer (CRC)	114 CRC + paired ANT (43 tumor + 36 polyp + 35 ANT)	Upregulated	-	-	Associated with clinical stage and type of samples (Tumor or polyp)	[4]
Glioma	8 gliomas + 8 healthy controls	Upregulated	-	-	-	[22]
Hepatocellular carcinoma (HCC)	52 HCC + paired ANT	Upregulated	Shorter O-S	-	-	[17]
	75 HCC + paired ANT	Upregulated	Poor survival rate	-	Associated with TNM stage, lymph node metastasis and cirrhosis	[28]
	161 HCC + paired ANT	Upregulated	Shorter O-S	Independent prognostic factor for HCC	Associated with TNM stage and histological grade	[15]
	70 HCC + paired ANT					[2]
Non-small cell lung cancer (NSCLC)	29 NSCLC + paired ANT + TCGA (1027 tumor and 108 normal tissues)	Upregulated	-	-	-	[3]
	35 NSCLC + paired ANT	Upregulated	Poor O-S	-	-	[28]
Oral squamous cell carcinoma (OSCC)	84 OSCC + paired ANT	Upregulated	Shorter O-S	-	Associated with lymph node metastasis, TNM stage and distant metastasis	[20]
Gallbladder (GBC)	35 GBC + paired ANT	Upregulated	Shorter O-S	-	Associated with lymph node metastasis and larger tumor size	[29]

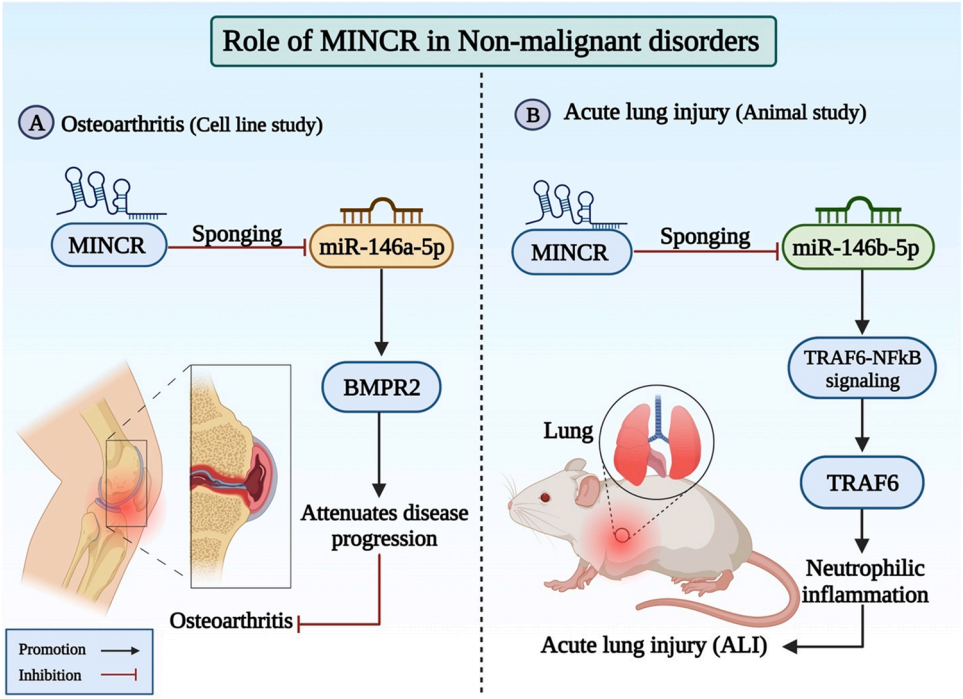

Fig. 2. Role of MINCR in non-malignant conditions. (A) Through sponging miR-146a-5p and increasing BMPR2 expression, the lncRNA MINCR suppresses the progression of osteoarthritis. (B) MINCR could promote the progression of ALI/ARDS by inducing cellular injury to the alveolar epithelial cells via the miR-146b-5p and TRAF6/NF-B pathways.

of osteoarthritis through targeting miR-146a-5p and increasing expression of BMPR2 [16]. On the contrary, MINCR has been found to be up-regulated in an animal model of LPS-induced acute lung injury and small airway epithelial cells. Short hairpin-mediated silencing of MINCR has enhanced cell viability and reduced apoptosis, therefore protecting against LPS-induced cell injury. This intervention can also suppress establishment of neutrophil extracellular traps and reduce neutrophil numbers, activity of myeloperoxidase, and release of inflammatory cytokines. The effects of MINCR on LPS-associated lung injury have been reversed by silencing of miR-146b-5p (Fig. 2). Moreover, MINCR silencing has reduced TRAF6 and p-P65 levels in LPS-induced small airway epithelial cells and lung tissue [7]. Table 4 shows the role of MINCR in non-malignant conditions.

6. Discussion

MINCR is an oncogenic lncRNA which activates c-Myc oncogene. This route of action is the most appreciated mechanism of function of

Table 4
Role of MINCR in non-malignant conditions (Δ: knock-down or deletion, ECM: extracellular matrix).

Disease type	Cell lines/Animal models	Number of clinical samples	Expression (case vs. control)	Function	References
Osteoarthritis	Chondrocytes	-	-	↑ MINCR (which sponges miR-146a-5p) → ↑ BMPR2: ↑ proliferation ↓ apoptosis ↓ ECM degeneration Note: overexpression of MINCR attenuates disease progression	[16]
Acute lung injury (ALI)	C57BL/6 mice	-	-	Transfection with LPS: Causes upregulation of MINCR Δ MINCR → ↑ miR-146b-5p → ↓ TRAF6: ↓ Neutrophilic inflammation ↓ apoptosis	[7]
Schizophrenia (SCZ)	-	GSE53987 dataset (48 SCZ brain samples + 55 normal brain samples) + GSE73129 (15 SCZ + 15 controls)	Upregulated		[26]
Amyotrophic lateral sclerosis (ALS)	-	10 ALS + 10 controls	Downregulated	-	[22]
Alzheimer's disease (AD)	-	5 AD + 4 controls	Downregulated	-	[22]

MINCR in the carcinogenesis. Similar to many other lncRNAs, MINCR can serve as a molecular sponge for miRNAs. miR-28–5p, miR-708–5p, miR-876–5p and miR-146a-5p are among miRNAs that are sponged by MINCR. The interactions between MINCR and other miRNAs should be investigated in future studies. Unraveling additional MINCR/miRNA/mRNA axes would help in identification of the mechanism of participation of MINCR in the pathological conditions. Moreover, these axes can be used as potential therapeutic targets and biomarker in human disorders. This would lead to design of effective therapies for cancer.

Moreover, MINCR contributes to the etiopathology of osteoarthritis, acute lung injury, schizophrenia, amyotrophic lateral sclerosis and Alzheimer's disease. In fact, MINCR represents a shared point between cancer and neurodegenerative disorders.

Based on the impact of MINCR in the regulation of expression the c-Myc, it is expected that up-regulation of MINCR in the tumoral tissues affects response of malignant cells to a variety of anti-cancer therapies. However, this issue has not been investigated yet. Moreover, the role of epigenetic factors in the regulation of MINCR expression has not been clarified.

MINCR over-expression has been remarkably associated with poor clinical outcome in a wide range of malignancies highlighting the importance of MINCR as a prognostic marker. This finding is also supported by the observed association between up-regulation of MINCR and malignant characteristics such as propensity to metastasize to distant locations. Therefore, MINCR-targeted therapies are expected to be effective in reducing tumor burden.

Collectively, MINCR is an oncogenic lncRNA which affects activity of cancer-related signaling pathways mainly through enhancing expression of c-Myc. Future studies are needed to examine the efficacy and safety of MINCR-targeting therapies for treatment of cancer.

Ethics approval and consent to Participant

Not applicable.

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CRediT authorship contribution statement

SGF wrote the draft and revised it. MT designed and supervised the study. AA, BMH and AE collected the data and designed the figures and tables. All the authors read the submitted version and approved it.

Declaration of Competing Interest

The authors declare they have no conflict of interest.

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Consent of publication

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