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Review Article

Crosstalk between lncRNAs and Wnt/β-catenin signaling pathways in lung cancers: From cancer progression to therapeutic response

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ABSTRACT

Lung cancer (LC) is considered to have the highest mortality rate around the world. Because there are no early diagnostic signs or efficient clinical alternatives, distal metastasis and increasing numbers of recurrences are a challenge in the clinical management of LC. Long non-coding RNAs (lncRNAs) have recently been recognized as a critical regulator involved in the progression and treatment response to LC. The Wnt/β-catenin pathway has been shown to influence LC occurrence and progress. Therefore, discovering connections between Wnt signaling pathway and lncRNAs may offer new therapeutic targets for improving LC treatment and management. In this review, the purpose of this article is to present possible therapeutic approaches by reviewing particular relationships, key processes, and molecules associated to the beginning and development of LC.

1. Introduction

Lung cancer (LC), a significant worldwide health issue, is responsible for around 18% of cancer-related fatalities [1]. Because of its complicated and obscure biological processes that result in unregulated cell growth and metastasis, it leads to the primary cause of cancer-related deaths. LC is commonly categorized into two primary types: small cell lung cancer (SCLC) and non-small cell lung cancer (NSCLC), SCLC accounts for 15%, and NSCLC accounts for 15% of all primary lung cancer cases [2,3]. Lung adenocarcinoma (LUAD), lung squamous cell carcinoma, and large cell carcinoma are the three main pathophysiological subtypes of NSCLC [4]. The prevalence of occurrences involving primary lung cancer has grown in the past few decades. In 2020, lung cancer is the second most commonly diagnosed cancer and the leading cause of cancer death [1]. Active and passive smoking, chronic exposure to carcinogens, ambient air pollution and unidentified risk factors are the most common risk factors for LC [5,6]. As clinical research is rapidly advancing, there are many various therapeutic options accessible for LC include surgical and non-surgical treatments. The non-surgical treatment includes molecularly focused treatment, immunotherapy, chemotherapy, and interventional therapy. Despite the development of innovative and multimodal treatments, the overall prognosis of individuals with LC remains insufficient [7]. Numerous researches have proposed that the low longevity rate of LC patients may be related to the high variability in tumors and lack of an early diagnosis. Thus, to improve LC patient diagnosis and therapy, it is imperative to investigate the biological properties of LC and identify novel molecular targets.

It was determined that the Wnt signaling pathway is a highly conserved pathway which is essential for determining cell fate and embryonic patterning in all multicellular organisms [8]. This signaling pathway is primarily divided into two distinct types: the canonical signaling pathway, which depends on β-catenin, and the non-canonical signaling pathway, which does not. The latter is further subdivided into the Wnt/calcium and planar cell polarity (PCP) pathways, both of which influence cancer growth and dissemination [9]. The canonical signaling pathway is initiated by Wnt molecules such as Wnt1 and Wnt3a, engaging with a frizzled receptor (Fz) and lipoprotein receptor-related protein (LRP)5/LRP6. Then, Fz binds into and produces a cytoplasmic protein called Disheveled (DVL) in mammals, which causes a concentration of β-catenin in the cytoplasm, its movement into the nucleus, and the transcription of the target gene. The non-canonical pathway is far less researched than the canonical pathway. There are both canonical

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and non-canonical routes in this evolutionarily conserved signaling system.

According to study, as much as 80 % of the human genetic code maybe converted into non-coding RNAs (ncRNAs) [10]. Among them, lncRNAs are the most prevalent and functionally diverse class [11]. Long non-coding RNAs (lncRNAs) is a transcript with one over 200 nucleotides in length that is not transcribed to proteins [12]. In the last decade, mounting data show that lncRNAs have been proven to remain multiple regulatory functions, such as altering cytoplasmic mRNA stability and translation, interfering with signaling pathways, regulating chromatin function, controlling membrane-free nuclease formation and action [13]. Specifically, numerous kinds of lncRNAs can function as endogenous RNAs (ceRNAs) that compete with sponge microRNAs (miRNAs) to lessen the regulatory impact of miRNAs on target mRNAs. The dysregulation of lncRNAs and the associated ceRNA network are being mentioned to take part in the genesis and progress of various malignant cancer, including LC. For instance, lncRNA SFTA1P was reported to be abnormally expressed in LC, and to inhibit the growth of xenografts and metastasis of LUAD cells by sponging miR-4766–5p via modulating the large tumor suppressor kinase 1 (LATS1)/ Yes-associated protein (YAP) signaling pathway [14,15]. LncRNA PCAT1 was overexpressed in NSCLC tissues, and exerted an oncogenic role and immunosuppression by activating sex-determining region Y-related high-mobility group box 2 (SOX2) to repressing the cyclic GMP-AMP synthase (cGAS)/ stimulator of interferon genes (STING) signaling mediated T-cell activation [16]. Many lncRNAs regulate downstream genes or proteins to influence important pathways in LC biological activities. Although we have made a number of advances in the study of the correlation between lncRNA and the Wnt/β-catenin pathway in many malignancies, the underlying processes of LC pathogenesis and progression are not yet fully understood. Therefore, further studies on the effects of lncRNA interaction with the Wnt/β-actin pathway are necessary. This article emphasizes the sequence of events such as lncRNA involving the Wnt/β-catenin pathway in LC impacting the evolution of lung carcinogenesis. It is hoped that these findings may provide unique and perhaps novel concepts for targeted therapy in LC patients.

2. The Wnt/β-catenin pathways related to LC

Several Wnt ligands have high levels in LC, such as Wnt1, Wnt2b, Wnt3a and Wnt5a. Reports indicated that up-regulated Wnt1 ligand can lead to resistance to immune checkpoint inhibitors (ICIs) and promote the advanced stages of LC treated with ICIs [17]. The Wnt2b gene was repressed under the influence of DDX56 down-regulating the mature miR-378a-3p, which was connected to early recurrence in squamous cell lung carcinoma [18]. Furthermore, the interaction of golgi-phosphoprotein 3 (GOLPH3) and cytoskeletonssociaated protein 4 (CKAP4) enhanced the secretion of Wnt3a by binding Wnt3a in exosomes, which subsequently drivered the Wnt/β-catenin pathway and induced distal metastasis in NSCLC [19]. Also, it was revealed that Wnt5a expression could affect the response of NSCLC cell lines to radiotherapy. The combination of Wnt5a knockdown and irradiation leaded to decrease translocation of β-catenin from the cytoplasm to the nucleus, which had a connection to the neoplastic features of NSCLC, such as tumor proliferation and apoptosis [20]. β-catenin proteins played important roles in tumor formation, and inhibiting LC pathogenesis and delaying LC progression [21]. Further, eIF3a promoted the transcriptional activation of β -catenin and mediates its nuclear accumulation to form a complex with T cell factor 4 (TCF4) contributed to the maintenance of NSCLC stem cell-like characteristics [22]. In addition, it was reported that glycogen synthetic kinase-3β (GSK3β) plays a correlated with dose role in gefitinib resistance, and serine-arginine protein kinase 1 (SRPK1) increases glycogen synthetic kinase-3 (GSK3) phosphorylation at Ser9 to trigger the Wnt pathway [23]. The Wnt signaling has many downstream targeted genes, including axis inhibition protein 2 (AXIN2), c-Myc and cyclinD1. C-Myc, a traditional transcription mediators of PD-L1, was overexpressed in multiple types of human carcinomas, including SCLC [24]. According to one study, more than half of Asian patients had AXIN2 promoter methylation, which enhanced nuclear accumulation of β-catenin and was associated with poor survival [25]. In brief, Wnt signaling is emphasized as a viable target for LC precision medicine since it has a role in the development, progression, and medication resistance of LC.

3. Interactions between lncRNAs and Wnt signaling pathway in LC

New evidence suggested that a variety of substances may act as regulators or remarkably influence gene expression, regulating the Wnt/β-catenin pathway by altering the expression of a number of lncRNAs [26]. It was established that β-catenin proteins were important regulators that set off the Wnt/β-catenin cascade. The role of the lncRNA/Wnt/β-catenin axis in the etiology of malignant tumors has recently been successfully studied. We draw the conclusion that LC is influenced by the major lncRNA/Wnt/ β-catenin axis (Table 1 and Fig. 1).

3.1. LncRNA CBR3-AS1

The lncRNA CBR3-AS1, having a transcriptional length of 749 nt, was first discovered to be substantially expressed in prostate cancer [27]. A prior investigation verified the facilitative role of CBR3-AS1 members in various tumor types. In recent studies, CBR3-AS1 expression was shown to be increased in NSCLC. It has proved linked to a poor long-term outcome in patients. In vitro cellular experiments, CBR3-AS1 suppression drove a significant reduction in proliferation, migration, and invasiveness, but an increase in apoptosis [28,29]. CBR3-AS1 was previously revealed to modify the expression of β-catenin proteins in A549 cells. Further CBR3- AS1 could triggered Wnt/β - catenin signaling cascade by inducing nuclear location of β-catenin in LUAD. CBR3-AS1 was knocked down, resulting lowered the activation of Wnt/ β-catenin signaling and suggested that decreasing CBR3-AS1 may alter the concentration of β -catenin in the cytoplasm or nucleus. This mechanism encouraged the Wnt signaling pathway to become active, enhancing the expression of Wnt target genes such c-Myc, cyclinD1, LGR5, and MMP-7 as well as modifying cell proliferation, migration and invasion [30].

3.2. LncRNA CASC15

LncRNA CASC15 was originally regarded as a key tumor suppressor in carcinoma but was later discovered to play an oncogenic role in many other cancers, such as breast cancer [31], laryngeal squamous cell carcinoma [32], osteosarcoma [33], thyroid carcinoma [34], esophageal squamous cell carcinoma [35] and nasopharyngeal carcinoma [36]. Overexpression of CASC15 was observed in LC tissues, which indicates that CASC15 maybe a potential biomarker [37,38]. CASC15 overexpression has a connection with LC cell neoplasm and could determine longevity time of individuals with advanced-stage disorders [37]. Inhibition of CASC15 significantly weakened the migration and growth ability of A549 and H1299 cells. This is consistent with the findings of laryngeal squamous cell carcinoma in CASC15. According to a paper, inhibiting CASC15 had a negative impact on how lung cancer developed in vivo [39]. In LC, CAS-C15 promotes NSCLC cell proliferation and migration by increasing SOX4 and thereby maintaining the β-catenin protein. Accordingly, several research groups have recently discovered that CASC15 may promote carcinogenesis by activating the Wnt/β -catenin signaling pathway in melanoma and colon cancer, indicating that the CASC15/β-catenin axis exhibited a vital role in the initiation and advancement of cancer [40,41].

Table 1
LncRNAs directly target Wnt/β-catenin signaling pathway in LC.

LncRNA	Oncogene/ suppressor	Targeted miRNA	Regulation of Wnt/ β-catenin	Biological functions	References
CBR3-AS1	Oncogene	Not available	Activation	Promote the proliferation, migration and invasion of LUAD cells	[30]
CASC15	Oncogene	Not available	Activation	Promote the proliferation and migration of NSCLC cells	[37]
MALAT1	Oncogene	miR-1297	Activation	Suppress the apoptosis and cisplatin(DDP) sensitivity of LUAD cells	[42]
DANCR	Oncogene	miR-216a	Activation	Promote the proliferation, stemness, invasion of NSCLC cells	[43]
LINC00514	Oncogene	Not available	Activation	Promote the proliferation, migration, invasion and epithelial-mesenchymal transition (EMT) of NSCLC cells	[44]
PKMYT1AR	Oncogene	MiR-485-5p	Activation	Promote the proliferation, migration, cell stemness and Xenograft tumor formation of NSCLC cells	[45]
PCAT6	Oncogene	MiR-326	Activation	Promote the proliferation and invasion but suppress the apoptosis of LC cells	[46]
NEAT 1	Oncogene	Not available	Activation	Promote the proliferation, invasion and migration but suppress apoptosis and anlotinib sensitivity of NSCLC cells	[47,48]
ITGB1-DT	Oncogene	Not available	Activation	Promote the proliferation, migration, invasion and Xenograft tumor formation of LUAD cells	[49]
SNHG11	Oncogene	MiR-4436a	Activation	Promote the proliferation and migration of LC cells	[50]
ASB16-AS1	Oncogene	Not available	Activation	Promote the proliferation and inhibit apoptosis of NSCLC cells	[51]
SNHG20	Oncogene	MiR- 197	Activation	Promotes the proliferation and inhibits the apoptosis of NSCLC cells	[52]
AC026356.1	Oncogene	Not available	Activation	Promote the stemness, proliferation and Xenograft tumor formation but increased apoptosis and cisplatin sensitivity of LUAD cells	[53]
LINC00669	Oncogene	Not available	Activation	Promote the proliferation, migration, invasion and Xenograft tumor formation but apoptosis of LUAD cells	[54]
DSCAM-AS1	Oncogene	MiR-577	Activation	Promote the proliferation, migration, invasion, and Xenograft tumor formation but suppress apoptosis of LUAD cells	[55]
UPLA1	Oncogene	Not available	Activation	Promote the proliferation, migration, invasion and Xenograft tumor formation of LUAD cells	[56]
JPX	Oncogene	MiR-33a-5p	Activation	Promote the proliferation, invasion, EMT and Xenograft tumor formation of LC cells	[57]
RPPH1	Oncogene	MiR-326	Activation	Promote the invasion, EMT and resistance to cisplatin/*cis*-diamminedichloridoplatinum (CDDP) of NSCLC cells	[58]
AOC4P	Suppressor	Not available	Inhibition	Suppress viability and invasion and induce apoptosis of NSCLC cells	[59]
LINC00476	Suppressor	Not available	Inhibition	Suppress the proliferation, migration, invasion and Xenograft tumor formation of NSCLC cells	[60]
DBH-AS1	Suppressor	MiR-155	Inhibition	Suppress the proliferation of NSCLC cells	[61]

3.3. LncRNA MALAT1

LncRNA MALAT1 as an oncogene with a high degree of conservation controls radiosensitivity and chemosensitivity in cancer cells, and fosters cancer progression [62,63]. Due to its association with tumor progression in NSCLC, MALAT1 was considered to be a prognostic cytokine that was first identified to be the survival marker [64]. For instance, increased MALAT1 expression in cisplatin (DDP) resistant A549/DDP cells encourages the activity of multiple drug resistance 1 (MDR1) by triggering signal transducer and activator of transcription protein 3 (STAT3) signaling, which decreases sensitivity to DDP and significantly worsens prognosis [65]. In A549/DDP cells, up-regulated expression of MALAT1 was linked to drug resistance thought β-catenin/MDR1 signaling. MALAT1 boosted drug resistance in A549/DDP cells via regulating β-catenin/MDR1 signaling via the miR- 1297/p300 axis [42]. Also miR-1297 had comparable effects in numerous experiments by regulating different signaling pathways. One study observed that miR-1297 could trigger the PTEN/AKT signaling pathway and Wnt/β-catenin signaling pathway to promote the proliferation of LC cells [66,67]. P300 is a transcriptional co-factor of β-catenin. It has been revealed that the P300/β-catenin complex is essential to activate β-catenin in transduction processes [68]. If p300 and β-catenin form a complex, this might improve the stability and nuclear distribution of p300 as well as enhancing β -catenin/LEF1 mediated transcriptional activity [69]. More research into Wnt/β-catenin signaling in LC is required since more molecular pathways have not yet been discovered.

3.4. LncRNA DANCR

LncRNA differentiation-antagonizing non-protein-coding RNA (DANCR) was initially discovered as a suppressor of epidermal ancestral differentiation [70]. In NSCLC, DANCR has been identified as a promoter of tumorigenesis by inhibiting several tumor suppressor miRNAs [43,71,72]. Wnt pathway activating genes are commonly upregulated in NSCLC tumorigenesis and metastasis whereas negative regulators are down-regulated [73,74]. DANCR expression was dramatically enhanced in diverse groups of NSCLC cell lines, except for H1944 cells. DANCR knockdown led to stem-like properties being reduced, long term clonogenic potential being inhibited, and impedance in cell migration, which are all classic Wnt/β -catenin inactivation features. In addition, DANCR knockdown reduced β-catenin protein expression and Wnt target genes Axin2 and c-Myc both mRNA and protein expression. Upregulation of miR-21-6a was reported to cell growth, invasion, and metastasis but induce apoptosis [75,76]. DANCR is high expressed in NSCLC tumors and cell lines. Silencing DANCR expression in NSCLC cells could inhibit long-term growth, migration and stemness by triggering Wnt/β-catenin signaling through miR-216a suppression [43].

3.5. LINC00514

LINC00514 has been shown to be overexpressed in cervical squamous cell carcinoma, gastric cancer, breast cancer and pancreatic cancer [77–80]. Zhu et al. [44] indicated that LINC00514 was significantly upregulated in NSCLC and related to poorer prognosis. They observed that silencing LINC00514 decreased the epithelial mesenchymal transition (EMT)-related proteins (E-Cadherin) and Wnt/β-catenin signaling related proteins (β-catenin, cyclinD), while enhancing N-Cadherin expression. Moreover, they revealed that LINC00514 facilitated cell proliferation, migration, and invasion by triggering the Wnt/β-catenin cascade.

3.6. LncRNA PKMYT1AR

The expression of lncRNA protein kinase, membrane associated tyrosine/threonine 1 associated lncRNA (PKMYT1AR) was up-regulated in NSCLC compared with cancer stem cells and peripheral blood serum [45]. Based on the research, PKMYT1AR serves as a sponge for miR-485-5p. MiR-485-5p may target the protein kinase, membrane

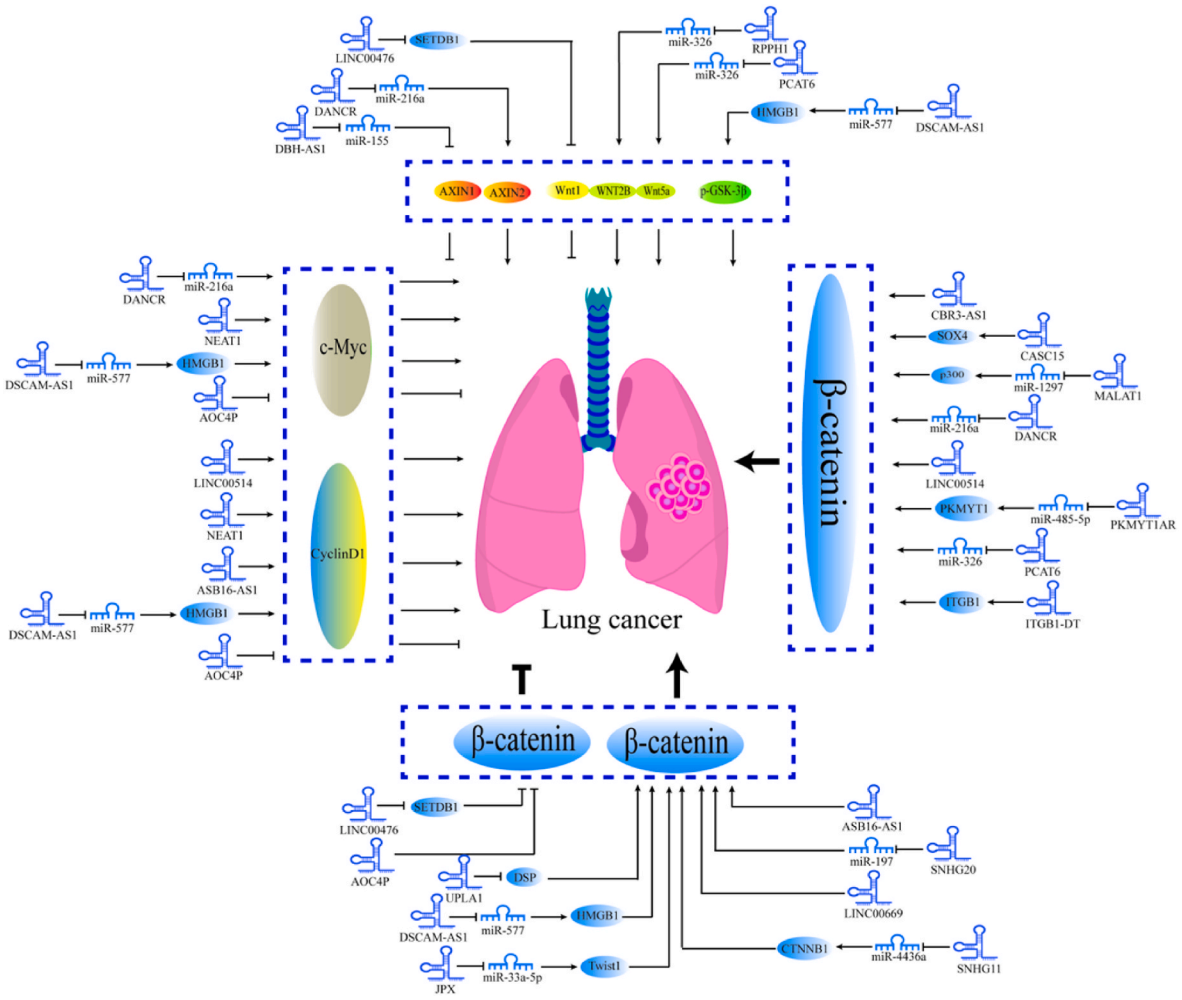

Fig. 1. LncRNAs related to the Wnt/β-catenin pathway in lung cancer.

associated tyrosine/threonine 1 (PKMYT1), a negative regulator of the typical cell cycle change by phosphorylating Tyr14/Tyr15, which suppressed the cyclin-dependent kinase 1 (CDK1)-cylinB combination [81]. Therefore, it was a vital target in the activating Wnt/β-catenin pathway and thereby suppress PKMYT1 protein levels. PKMYT1AR could promote the NSCLC cell proliferation, migration and xenograft tumor formation abilities by diminishing miR-485-5p function, increasing PKMYT1 protein expression and activating Wnt/β -catenin pathway.

3.7. LncRNA PCAT6

LncRNA PCAT6 is a sort of lncRNAs which has been discovered to have involved in different types of cancer, including LC. PCAT6 was highly expressed in lung cancer samples and cells induced by sevoflurane, and overexpression of PCAT6 reversed the suppression of sevoflurane on viability, proliferation, invasion and enhancement on apoptosis of LC cells. Depletion of PCAT6 was shown to significantly increase miR-326 levels in LC cells. Overexpression of miR-326 inhibits production of Wnt5a which acts as a positive activator of the Wnt/β-catenin pathway [46].

3.8. LncRNA NEAT1

Anlotinib an orally administered multi-targeting tyrosine kinase inhibitor (TKI) which is used in the patients with advanced refractory NSCLC [82]. LncRNA NEAT1 has been shown to exert an oncogenic role in NSCLC [83]. Gu et al. [48] [][][]observed that NEAT1 considerably induce the anlotinib resistance by hindering the tumor cell proliferation, migration, and invasion. NEAT1 was involved in the sponging of miR-760 and miR-361-5p. MiR-760 and miR-361-5p could considerately suppress Wnt/β-catenin signaling cascade. Therefore, anlotinib resistance is maintained by NEAT1, which regulates the Wnt/β -catenin pathway.

3.9. LncRNA ITGB1-DT

LncRNA ITGB1-DT has been associated with the progression of multiple malignant solid tumors, such as breast cancer, colon cancer, stomach adenocarcinoma and NSCLC [84–87]. ITGB1-DT is significantly elevated in LC, operates as an oncogene in LC, and is the independent risk factors for poor prognosis, and had diagnostic value for NSCLC patients. It has been seen that interfering with the ITGB1-DT expression can decrease the proliferation, migration, and invasion of LC cells, and drug-resistant A549/DDP by influencing the MAPK/ERK signaling pathway [88]. Besides, Chang et al. [88] uncovered that ITGB1-DT may induce epigenetic enhancement of ITGB1 expression, which is reverse transcribed from the ITGB1-DT gene. ITGB1, also known as integrin β1, was found to exert oncogenic roles in various cancers by regulating AKT/Wnt/β-catenin pathway [89,90]. Thus, by modulating ITGB1, ITGB1-DT activated the Wnt/β-catenin/MYC axis and formed a positive feedback loop, allowing ITGB1-DT silencing as a new potential therapeutic target for LUAD [49].

3.10. LncRNA SNHG11 (lncRNA NCRNA00101)

According to earlier research, lncRNA SNHG11 functions as a oncogene, preventing the growth of numerous tumors including gastric cancer, colorectal cancer, hepatocellular cancer and NSCLC.

[91–94]. SNHG11 was shown to be highly expressed and correlated with poor patient prognosis, TNM stage and tumor size in LC. Silencing SNHG11 reduced the cancer cells proliferation, invasion, and metastasis in vitro, while inhibiting tumor growth in vivo [50].

The ceRNAs are RNA transcripts that interact with one another by depressing other mRNAs that share the same miRNA response regions and reducing the production of targeted miRNAs [95]. SNHG11 could act as a sponge for miR-4436. Overexpression of miR-4436 reduces the production of catenin beta 1 (CTNNB1) protein, which positively regulates the Wnt/β -catenin signaling cascade. As a consequence, SNHG11 might thereby increase CTNNB1 protein by blocking miR-4436, which activates the Wnt/β-catenin axis in LC[50].

3.11. LncRNA ASB16-AS1

The LncRNA ASB16 antisense RNA 1 (ASB16-AS1), located on chromosome 17, has been identified as an oncogene that promotes the development and incidence of several types of cancer [96,97]. The expression of ASB16-AS1 was found to be high expression in NSCLC. Tan et al. [51] that ASB16-AS1 could promote proliferation but inhibits apoptosis of LC cells in vitro. Further evidence suggests that ASB16-AS1 overexpression caused the levels of β-catenin and cyclinD1 to reduce considerably but p21 to increase by triggering the Wnt/β-catenin signaling cascade.

3.12. LncRNA SNHG2

The research has revealed that lncRNA SNHG20 expression was higher in NSCLC cell lines and tissues than the non-tumor cells and tissues [98]. SNHG20 might be functioning as a sponge for miR-197 and inhibit its downstream activity. Wang et al. [52] found out that SNHG20 could promote proliferation but inhibit apoptosis by bounding to miR-197 in a targeted manner. In addition, miR-197 downregulation led to the remarkably down-regulation in TCF and LEF1, while significantly enhancement in nuclear translocation of β-catenin after transfection experiments. As a result, the SNHG-20/miR- 197/Wnt/β-catenin axis may have therapeutic potential for NSCLC through influencing the Wnt/β-catenin signaling cascade.

3.13. LncRNA AOC4P

LncRNA amine oxidase, copper containing 4, pseudogene (AOC4P) is a novel lncRNA which has received much attention. Before that, AOC4P is found to be a novel tumor suppressor in ovarian cancer and hepatocellular carcinoma (HCC) [99,100]. Low expression of AOC4P is positively linked to poor prognostic outcomes in HCC. Li et al. [59] discovered that overexpressing AOC4P inhibited the NSCLC cells proliferation, invasion and tumor growth, while induced apoptosis both in vitro and in vivo. The Wnt/β-catenin is widely recognized as a significant oncogenic pathway in cancer progression [101]. AOC4P overexpression could hinder the Wnt/β-catenin signaling cascade, leading to decreased β-catenin, cyclinD1, and c-Myc tumor suppressors as Wnt/β-catenin pathway target genes [59]. These findings showed that AOC4P suppressed tumor growth in NSCLC. In conclusion, these lncRNAs are reported to have a substantial connection with prognosis, and it is necessary to further investigate the mechanism by which lncRNAs influence the Wnt/β-catenin pathway. Fortunately, the cited study offers encouraging suggestions for LC diagnostic or therapeutic tacks.

4. Biological functions of lncRNAs/wnt/β-catenin axis in LC

LncRNAs exert diverse biological functions of LC through the regulation of Wnt/β-catenin cascades. As it turned out, we described the processes associated with LC actions in Fig. 2, hoping for full insight into the molecular of lncRNAs in LC.

4.1. Cancer cell stemness

Cancer stem cells (CSCs), a distinct class of cancer cells, have a limitless capacity for self-renewal and differentiation, which aids in the development of treatment resistance and the start, progression, and metastasis of malignant tumors [102,103]. Existing research find that lncRNAs promote cancer stem cell maintenance in lung cancer through activating Wnt signaling pathway. Yu et al. [43] illustrated that DANCR knockdown could attenuate malignant phenotype and stem-like characteristics in A549 and H19755 cell lines by sponging miR-216a. DANCR increased NSCLC stemness through the activation of CSC regulators Wnt and Sox2 and the action was found inversely correlated with activating Wnt/β-catenin signaling through suppressing miR-216a. Moreover, m6A-mediated up-regulation of lncRNA-AC026356.1 induced cell stemness by the mediation of upstream molecule METTL14 and IGF2BP2. It suggests that there might be a METTL14/IGF2BP2/AC026356.1 loop that regulates cancer stemness via activating Wnt signaling pathway in LUAD [53].

4.2. Proliferation and apoptosis

A variety of cancer treatments, including radiation therapy and cytotoxic chemotherapeutic drugs, have as their primary objective the inhibition of cancer cell growth and proliferation, which is an efficient technique to halt the course of cancer. Wnt/β -catenin are essential signals that govern cell growth and proliferation. By preventing β-catenin from nuclear translocation and decreasing downstream transcription factor T cell factor 1 (TCF- 1), LINC00669 activates Wnt/β-catenin to promote cell proliferation but inhibit apoptosis [54]. Also, DSCAM-AS1 knockdown inhibited cell proliferation but promoted apoptosis by sponging miR-577 and reducing HMGB1 protein expression under the control of the Wnt/β-catenin pathway in NSCLC [55].

4.3. Migration and invasion

Invasion and metastasis are the primary causes of shorter life expectancy for patients, as they are the basic characteristics of malignant tumors. LncRNAs that promote lung cancer cell migration and invasion are up-regulated in lung cancer tissues. For instance, LINC00514 increases the migration and invasion of NSCLC. On the one hand, LINC00514 silencing reduced TCF/LEF1 activity, demonstrating yet another way that LINC00514 knockdown inactivated Wnt signaling in NSCLC cells; on the other hand, down-regulation of LINC00514 inhibited migration and invasiveness abilities of cells and tumor xenograft growth [44]. The Wnt/β-catenin pathway is recognized as carcinogenic. Functionally, the lncRNA LUAD-associated transcript-1 (UPLA1) promotes cell invasion and proliferation in LUAD. Mechanistically, By binding to desmoplakin (DSP), UPLA1 increased Wnt/β-catenin signaling and was discovered to be negatively controlled by the transcription factor YY1 [56].

4.4. Epithelial mesenchymal transition

Epithelial cells can turn into mesenchymal cells through a process known as EMT. During this process, epithelial cells gradually shed their epithelial identity in favor of more mesenchymal characteristics, such as a spindle-like shape and a lack of intercellular adhesion. The partial or complete activation of EMT is necessary for the malignant growth of many malignant tumors in addition to its function in pathophysiological

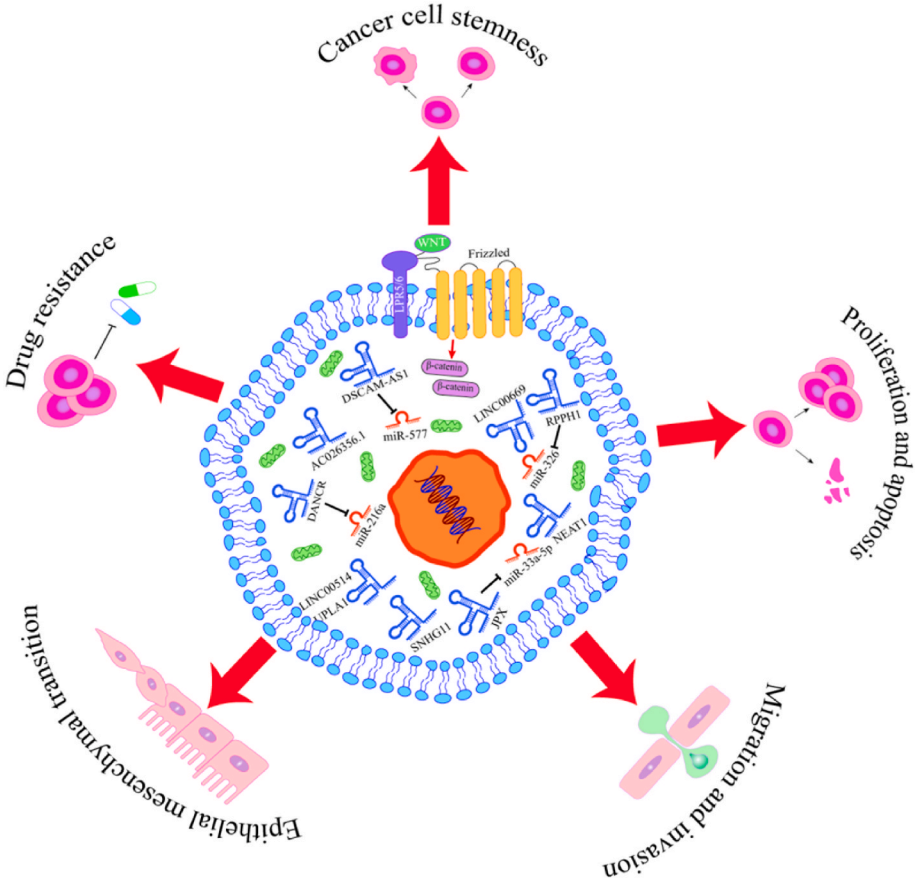

Fig. 2. The mechanism and function of lncRNAs in lung cancer progression.

processes including embryonic development and wound healing. By attaching to the appropriate ligand of the tumor cell and initiating a series of cascade events in the cell, the tumor microenvironment releases particular signals that activate the EMT program. TGF, WNTs, and NOTCH are examples of signaling pathways associated with EMT [104]. The up-regulation of LINC00514 was observed in non-small cell lung cancer lines and clinical samples, which activated Wnt pathway and triggered EMT by positively regulating the expression of Wnt [44]. Pan et al. [57] found that lncRNA JPX promoted cell proliferation, EMT and cell invasion in lung cancer by inhibiting the tumorigenesis and metastasis processes triggered through miR-33a-5p/Twist1 axis by activating Wnt/β-catenin signaling. It has been found that SNHG11 was high expressed in LC and played the role of tumor oncogene. SNHG11 can directly bind with β -catenin to activate classic Wnt pathway by facilitating the EMT process [50].

4.5. *Drug resistance*

These lncRNAs that impede drug resistance are frequently found to exhibit high expression levels in drug resistant lung cancer cells. This finding holds significant importance in the selection and application of targeted therapeutics. RPPH1 promoted tumor development by inducing invasion and EMT in lung cancer cells. In cellular experiments, RPPH1 might improve A549 and H1299 resistance to cisplatin/*cis*-diamminedichloridoplatinum (CDDP) therapy by enhancing cell viability and colonial formation. Furthermore, in vitro research revealed that RPPH1 promoted NSCLC advancement via regulating miR-326 and WNT2B [58]. In addition to this, lncRNAs also influence drug resistance by regulating cell proliferation and apoptosis. The existence of cancer stemness is the main cause of drug resistance. The AC026356.1 has been observed to enhance resistant to erlotinib treatment by increasing stemness of stem-like LC cells in the context of LC therapy. Researchers have demonstrated that m6A-mediated AC026356.1 expression was substantially up-regulated in stem cells and chemoresistant LC cells. As a result, silencing AC026356.1 markedly suppressed proliferation and migration but increased apoptosis in A549/DDP cells [53]. By suppressing proliferation but increasing apoptosis, NEAT 1 knockdown sensitizes NSCLC cells to chemotherapy. Mechanistically, NEAT 1 knockdown enhances the anti-apoptotic and cytotoxic effect of anlotinib, making NSCLC cells more sensitive to anlotinib through down-regulation of the Wnt/β-catenin signaling cascade [48]. Anlotinib paired with NEAT 1 knockdown therapy offers unique insights into creating a combination therapeutic strategy to chemotherapy insensitivity in LC patients. Nevertheless, the mechanism through which lncRNA acts as a positive regulator in the treatment of LC still require more in-depth analysis.

5. Clinical applications of lncRNAs and wnt/β-catenin signaling in LC

In order to find more positive and useful clinical applications for LC patients, we enlarge on the crucial roles played by lncRNAs and the Wnt/β-catenin cascade in LC. Accordingly, we further concentrate on the potential targets of lncRNAs and Wnt/β-catenin pathway as well as diagnostic biomarkers and prognostic biomarkers.

5.1. *Effective diagnostic biomarkers*

The majority of cancer biomarkers used today are proteins or peptides whose alterations in tissue or blood levels indicate the development of the tumor [105]. But the invasive and uncomfortable nature of biopsy or blood makes it difficult to use. So, it is vital to create

non-protein biomarkers that are very sensitive and non-intrusive. Due to the absence of recognizable symptoms in the early stages of LC, the majority of patients only have limited survival spans after they are detected. As a result, specialists have recently investigated more accurate LC diagnostic biomarkers [106,107]. Based on reports, increased expression of ASB16-AS1 contributed to promoting proliferation while inhibiting apoptosis in NSCLC through the Wnt/β-catenin signaling pathway [51]. It was previously reported that NEAT1 functioned as an oncogene in NSCLC through regulating the Wnt signaling pathway[[47]. Additional investigation is needed to better understand the diagnostic functions of lncRNAs in LC.

5.2. Possible prognosis biomarkers

Effective prognostic biomarkers are now essential for estimating the likelihood of LC recurring in clinical practice. HIF1A-AS2, a KRAS responding lncRNA, was found to be elevated in LC and its high expression was correlated with poor patient prognosis. HIF-1A-AS2 and MYC formed a double-regulatory loop that could pro-mote cell survival, tumor growth and metastasis [108]. Increased levels of LINC00669 was generally linked with a bad prognosis. When the expression level of LINC00669 was down, the cell proliferation was weakened while apoptosis was enhanced by inactivating Wnt/β-catenin pathway in NSCLC [54]. These findings offer a wide range of indicators to properly estimate the likelihood of recurrence in LC patients.

6. Conclusion

LC is a prevalent illness that poses a major global health risk to people. The Wnt/β-catenin signaling pathway is a widely acknowledged oncogenic pathway that is implicated in the beginning and progression of abroad variety of human malignancies, including LC. In several studies, lncRNAs have been shown to affect the Wnt/β-catenin signaling pathway in LC. In this review, we summarized the essential signaling cascade of the Wnt/β-catenin signaling pathway and emphasized the critical functions of lncRNAs in regulating this pathway in LC.

As previously indicated, mounting data suggests that the Wnt/β-catenin signaling system is crucial to the initiation and spread of human malignancies. The Wnt/β-catenin signaling pathway has been implicated in multiple roles, including normal embryonic development, tissue differentiation, tissue homology, and carcinogenesis [109]. By activating β-catenin, Wnt signaling controls several processes of cancer cells, including invasion, proliferation, flexibility and cell cycle determination [110]. This review suggests that the lncRNA-regulated Wnt/β-catenin pathway functions as a carcinoma promoter as well as suppressor. Therefore, for lncRNAs to be used in therapeutic settings going forward, a deeper comprehension of the intricate mechanisms behind their impact on the Wnt/β-catenin pathway is required.

It has been established that lncRNAs, a class of ncRNA transcripts, are functional components engaged in a variety of biological processes in human malignancies, including angiogenesis, metastasis, apoptosis, and cell survival. According to research, lncRNAs could regulate several important pathways connected to cancer, including the STAT3, PI3K-AKT-mTOR, and Notch pathways [111–113]. In this paper, we compiled 21 lncRNAs linked to the Wnt/β-catenin pathway in LC, 18 of which are up-regulated and 3 of which are down-regulated. Mechanistically, in the nucleus, lncRNAs participate in the nuclear localization or degradation of β-catenin to regulate the Wnt/β-catenin signaling pathway. In the cytoplasm, lncRNAs can act as sponges to compete for binding miRNAs or interact with proteins, including Wnt/β-catenin pathway components and their regulatory proteins, thereby affect their stability and activity. As described in our paper, the SNHG20 activates the Wnt/β-catenin pathway by sponging miR-197 and promoting the nuclear localization of β-catenin in NSCLC but suppresses the activity of TCF and LEF1 [52].

According to a clinical standpoint, a number of studies have pointed out that potential therapeutic and prognostic biomarkers might be molecules connected to the lncRNA-Wnt/β-catenin regulatory axis such as the FOXO1/lncRNA LYPLAL1-DT/hnRNPK/β-catenin signaling axis in triple-negative breast cancer (TNBC) [114]. It should be acknowledged that there are still a lot of restrictions. First, it is important to assess if a lncRNA expression pattern is sufficiently stable to serve as a predictive biomarker. Establishing a consistent analytical process including sample processing, recognition techniques, and standard values is also crucial. In addition, irregular Wnt/β-catenin signaling pathways are often not exclusively caused by lncRNAs. The Wnt/β-catenin pathway and other signaling path-ways also communicate with one another. Applying this treatment technique clinically is quite difficult because of the need to ensure the physiological functioning of Wnt/β-catenin pathway while targeting it selectively. Thus, it is necessary to do more research to see whether these compounds can be used clinically to treat LC. There are still many unidentified lncRNAs in the regulatory network of the Wnt/β-catenin signaling pathway, and our understanding of how lncRNAs regulate the Wnt/β-catenin pathway is still lacking. Research has shown that lncRNAs have no effect on the activity of the Wnt/β -catenin pathway by a single biochemical mechanism. To gain greater understanding of the Wnt/β -catenin pathway and LC development, more research into the unique genetic signals controlling this process is crucial.

In summary, our analysis outlined the intricate underlying processes and demonstrated the role of lncRNAs in the Wnt/β-catenin signaling pathway regulation network to offer new insights into the initiation of LC. These findings also suggest that a novel approach to identifying and treating LC might involve focusing on these lncRNAs or Wnt/β -catenin pathway elements.

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CRediT authorship contribution statement

Ting Wu: Writing – review & editing, Writing – original draft, Validation, Conceptualization. **YiRan Dong:** Visualization, Validation. **XinZhi Yang:** Visualization, Validation. **Liang Mo:** Visualization, Validation, Supervision, Funding acquisition, Conceptualization. **Yong You:** Visualization, Validation, Supervision, Funding acquisition, Conceptualization.

Declaration of competing interest

(1) Guarantee that there are no duplicate publications or multiple submissions.
(2) We guarantee that there is no conflict of interest due to financial interests or other relationships.
(3) All authors have read the article and agreed to publish it, all authors are eligible for authorship, and all authors agree that the article represents their real research results and that they are responsible for the article.
(4) The corresponding author of the article is responsible for contacting the first author to revise and finally review the review draft.

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Abbreviation

LC	Lung cancer
LncRNAs	Long non-coding RNAs
SCLC	Small cell lung cancer
NSCLC	Non-small cell lung cancer
LUAD	Lung adenocarcinoma
PCP	Planar cell polarity
Fz	Frizzled receptor
LRP	Lipoprotein receptor-related protein
DVL	Disheveled in mammals
NcRNAs	Non-coding RNAs
Ce-RNAs	Endogenous RNAs
MiRNAs	MicroRNAs
LATS1	Large tumor suppressor kinase 1
YAP	Yes-associated protein
SOX2	Sex-determining region Y-related high-mobility group box 2
cGAS	Cyclic GMP-AMP synthase
STING	Stimulator of interferon genes
ICIs	Immune checkpoint inhibitors
GOLPH3	Golgi phosphoprotein 3
CKAP4	Cytoskeleton-associated protein 4
TCF4	T cell factor 4
GSK3β	Glycogen synthase kinase-3β
SRPK1	Serine-arginine protein kinase 1
GSK3	Glycogen synthase kinase-3
AXIN2	Axis inhibition protein 2
SOX4	Sex-determining region Y-related high-mobility group box 4
DDP	Cisplatin
MDR1	Multiple drug resistance 1
STAT3	Signal transducer and activator of transcription protein 3
DANCR	Differentiation-antagonizing non-protein-coding RNA
EMT	Epithelial mesenchymal transition
E− Cadherin	EMT-related proteins
PKMYT1AR	Protein kinase, membrane associated tyrosine/threonine 1 associated lncRNA
PKMYT1	Protein kinase, membrane associated tyrosine/threonine 1
TKI	Tyrosine kinase inhibitor
CTNNB1	Catenin beta 1
ASB16-AS1	ASB16 antisense RNA 1
AOC4P	Amine oxidase, copper containing 4, pseudogene
CSCs	Cancer stem cells
TCF- 1	T cell factor 1
DSP	Desmoplakin
UPLA1	LUAD-associated transcript-1
CDDP	Cisplatin/cis-diamminedichloridoplatinum

References

[1] H. Sung, J. Ferlay, R.L. Siegel, M. Laversanne, I. Soerjomataram, A. Jemal, F. Bray, Global cancer statistics 2020: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries, CA Cancer J Clin 71 (3) (2021) 209–249, https://doi.org/10.3322/caac.21660.

[2] V.S. Gadepalli, S.P. Deb, S. Deb, R.R. Rao, Lung cancer stem cells, p53 mutations and MDM2, Subcell. Biochem. 85 (2014) 359–370, https://doi.org/10.1007/978-94-017-9211-0_19.

[3] A.K. Ganti, A.B. Klein, I. Cotarla, B. Seal, E. Chou, Update of incidence, prevalence, survival, and initial treatment in patients with non-small cell lung cancer in the US, JAMA Oncol. 7 (12) (2021) 1824–1832, https://doi.org/10.1001/jamaoncol.2021.4932.

[4] R.S. Herbst, D. Morgensztern, C. Boshoff, The biology and management of non-small cell lung cancer, Nature 553 (7689) (2018) 446–454, https://doi.org/10.1038/nature25183.

[5] L.A. Tse, O.W.-K. Mang, I.T.-S. Yu, F. Wu, J.S.-K. Au, S.C.-K. Law, Cigarette smoking and changing trends of lung cancer incidence by histological subtype among Chinese male population, Lung Cancer 66 (1) (2009) 22–27, https://doi.org/10.1016/j.lungcan.2008.12.023.

[6] I. Brüske-Hohlfeld, Environmental and occupational risk factors for lung cancer, Methods Mol. Biol. 472 (2009), https://doi.org/10.1007/978-1-60327-492-0_1.

[7] C. Gridelli, A. Rossi, D.P. Carbone, J. Guarize, N. Karachaliou, T. Mok, F. Petrella, L. Spaggiari, R. Rosell, Non-small-cell lung cancer, Nat Rev Dis Primers 1 (2015) 15009, https://doi.org/10.1038/nrdp.2015.9.

[8] O. Silva-García, J.J. Valdez-Alarcón, V.M. Baizabal-Aguirre, Wnt/β-Catenin signaling as a molecular target by pathogenic bacteria, Front. Immunol. 10 (2019) 2135, https://doi.org/10.3389/fimmu.2019.02135.

[9] X.-Y. Hu, P.-F. Hou, T.-T. Li, H.-Y. Quan, M.-L. Li, T. Lin, J.-J. Liu, J. Bai, J.-N. Zheng, The roles of Wnt/β-catenin signaling pathway related lncRNAs in cancer, Int. J. Biol. Sci. 14 (14) (2018) 2003–2011, https://doi.org/10.7150/ijbs.27977.

[10] P. Zhang, W. Wu, Q. Chen, M. Chen, Non-coding RNAs and their integrated networks, J Integr Bioinform 16 (3) (2019), https://doi.org/10.1515/jib-2019-0027.

[11] L. Ma, V.B. Bajic, Z. Zhang, On the classification of long non-coding RNAs, RNA Biol. 10 (6) (2013) 925–933, https://doi.org/10.4161/rna.24604.

[12] B. Maher, ENCODE: the human encyclopaedia, Nature 489 (7414) (2012) 46–48.

[13] L. Statello, C.-J. Guo, L.-L. Chen, M. Huarte, Gene regulation by long non-coding RNAs and its biological functions, Nat. Rev. Mol. Cell Biol. 22 (2) (2021), https://doi.org/10.1038/s41580-020-00315-9.

[14] B. Zhu, M. Finch-Edmondson, K.W. Leong, X. Zhang, M. V, Q.X.X. Lin, Y. Lee, W. T. Ng, H. Guo, Y. Wan, M. Sudol, R. DasGupta, LncRNA SFTA1P mediates positive feedback regulation of the Hippo-YAP/TAZ signaling pathway in non-small cell lung cancer, Cell Death Dis. 7 (1) (2021) 369, https://doi.org/10.1038/s41420-021-00761-0.

[15] W. Zhang, M. Bai, K. Liu, J. Tan, J. Ma, J. Zhao, P. Hou, LncRNA surfactant associated 1 activates large tumor suppressor kinase 1/Yes-associated protein pathway via modulating hypoxic exosome-delivered miR-4766-5p to inhibit lung adenocarcinoma metastasis, Int. J. Biochem. Cell Biol. 153 (2022) 106317, https://doi.org/10.1016/j.biocel.2022.106317.

[16] Y. Gao, N. Zhang, Z. Zeng, Q. Wu, X. Jiang, S. Li, W. Sun, J. Zhang, Y. Li, J. Li, F. He, Z. Huang, J. Zhang, Y. Gong, C. Xie, LncRNA PCAT1 activates SOX2 and suppresses radioimmune responses via regulating cGAS/STING signalling in non-small cell lung cancer, Clin. Transl. Med. 12 (4) (2022) e792, https://doi.org/10.1002/ctm2.792.

[17] C. Mehlman, P. Takam Kamga, A. Costantini, C. Julié, C. Dumenil, J. Dumoulin, J. Ouaknine, V. Giraud, T. Chinet, J.-F. Emile, E. Giroux Leprieur, Baseline hedgehog pathway activation and increase of plasma Wnt1 protein are associated with resistance to immune checkpoint inhibitors in advanced non-small-cell lung cancer, Cancers 13 (5) (2021), https://doi.org/10.3390/cancers13051107.

[18] Q. Wu, X. Luo, M.G. Terp, Q. Li, Y. Li, L. Shen, Y. Chen, K. Jacobsen, T.G. Bivona, H. Chen, R. Zeng, H.J. Ditzel, DDX56 modulates post-transcriptional Wnt signaling through miRNAs and is associated with early recurrence in squamous cell lung carcinoma, Mol. Cancer 20 (1) (2021) 108, https://doi.org/10.1186/s12943-021-01403-w.

[19] J.-W. Song, J. Zhu, X.-X. Wu, T. Tu, J.-Q. Huang, G.-Z. Chen, L.-Y. Liang, C.-H. Zhou, X. Xu, L.-Y. Gong, GOLPH3/CKAP4 promotes metastasis and tumorigenicity by enhancing the secretion of exosomal WNT3A in non-small-cell lung cancer, Cell Death Dis. 12 (11) (2021) 976, https://doi.org/10.1038/s41419-021-04265-8.

[20] J. Li, S. Xu, H. Dong, X. Wu, L.-H. Wang, X. Xu, Altered Wnt5a expression affects radiosensitivity of non-small cell lung cancer via the Wnt/β-catenin pathway, Exp. Ther. Med. 23 (1) (2022) 5, https://doi.org/10.3892/etm.2021.10927.

[21] T. Valenta, G. Hausmann, K. Basler, The many faces and functions of β-catenin, EMBO J. 31 (12) (2012) 2714–2736, https://doi.org/10.1038/emboj.2012.150.

[22] J.-Y. Zheng, T. Zhu, W. Zhuo, X.-Y. Mao, J.-Y. Yin, X. Li, Y.-J. He, W. Zhang, C. Liu, Z.-Q. Liu, eIF3a sustains non-small cell lung cancer stem cell-like properties by promoting YY1-mediated transcriptional activation of β-catenin, Biochem. Pharmacol. 213 (2023) 115616, https://doi.org/10.1016/j.bcp.2023.115616.

[23] J.-Q. Huang, L.-X. Duan, Q.-Y. Liu, H.-F. Li, A.-P. Hu, J.-W. Song, C. Lin, B. Huang, D. Yao, B. Peng, Y. Sun, Y. Wen, L. Yang, X. Xu, L.-Y. Gong, Serine-arginine protein kinase 1 (SRPK1) promotes EGFR-TKI resistance by enhancing GSK3β Ser9 autophosphorylation independent of its kinase activity in non-small-cell lung cancer, Oncogene 42 (15) (2023) 1233–1246, https://doi.org/10.1038/s41388-023-02645-2.

[24] S.K. Nair, S.K. Burley, X-ray structures of Myc-Max and Mad-Max recognizing DNA. Molecular bases of regulation by proto-oncogenic transcription factors, Cell 112 (2) (2003) 193–205.

[25] R.C. Tseng, R.K. Lin, C.K. Wen, C. Tseng, H.S. Hsu, W.H. Hsu, Y.C. Wang, Epigenetic silencing of AXIN2/betaTrCP and deregulation of p53-mediated control lead to wild-type beta-catenin nuclear accumulation in lung tumorigenesis, Oncogene 27 (32) (2008) 4488–4496, https://doi.org/10.1038/onc.2008.83.

[26] R. Beroukhim, C.H. Mermel, D. Porter, G. Wei, S. Raychaudhuri, J. Donovan, J. Barretina, J.S. Boehm, J. Dobson, M. Urashima, K.T. Mc Henry, R. M. Pinchback, A.H. Ligon, Y.-J. Cho, L. Haery, H. Greulich, M. Reich, W. Winckler, M.S. Lawrence, B.A. Weir, K.E. Tanaka, D.Y. Chiang, A.J. Bass, A. Loo, C. Hoffman, J. Prensner, T. Liefeld, Q. Gao, D. Yecies, S. Signoretti, E. Maher, F.J. Kaye, H. Sasaki, J.E. Tepper, J.A. Fletcher, J. Tabernero, J. Baselga, M.-S. Tsao, F. Demichelis, M.A. Rubin, P.A. Janne, M.J. Daly, C. Nucera, R. L. Levine, B.L. Ebert, S. Gabriel, A.K. Rustgi, C.R. Antonescu, M. Ladanyi, A. Letai, L.A. Garraway, M. Loda, D.G. Beer, L.D. True, A. Okamoto, S.L. Pomeroy, S. Singer, T.R. Golub, E.S. Lander, G. Getz, W.R. Sellers, M. Meyerson, The

landscape of somatic copy-number alteration across human cancers, Nature 463 (7283) (2010) 899–905, https://doi.org/10.1038/nature08822.
[27] Z. Fang, C. Xu, Y. Li, X. Cai, S. Ren, H. Liu, Y. Wang, F. Wang, R. Chen, M. Qu, Y. Wang, Y. Zhu, W. Zhang, X. Shi, J. Yao, X. Gao, J. Hou, C. Xu, Y. Sun, A feed-forward regulatory loop between androgen receptor and PlncRNA-1 promotes prostate cancer progression, Cancer Lett. 374 (1) (2016) 62–74, https://doi.org/10.1016/j.canlet.2016.01.033.
[28] Y. Guan, J. Yang, X. Liu, L. Chu, Long noncoding RNA CBR3 antisense RNA 1 promotes the aggressive phenotypes of non-small-cell lung cancer by sponging microRNA-509-3p and competitively upregulating HDAC9 expression, Oncol. Rep. 44 (4) (2020) 1403–1414, https://doi.org/10.3892/or.2020.7719.
[29] S. Liu, N. Zhan, C. Gao, P. Xu, H. Wang, S. Wang, S. Piao, S. Jing, Long noncoding RNA CBR3-AS1 mediates tumorigenesis and radiosensitivity of non-small cell lung cancer through redox and DNA repair by CBR3-AS1/miR-409-3p/SOD1 axis, Cancer Lett. 526 (2022), https://doi.org/10.1016/j.canlet.2021.11.009.
[30] M. Hou, N. Wu, L. Yao, LncRNA CBR3-AS1 potentiates Wnt/β-catenin signaling to regulate lung adenocarcinoma cells proliferation, migration and invasion, Cancer Cell Int. 21 (1) (2021) 36, https://doi.org/10.1186/s12935-020-01685-y.
[31] P. Shen, Y. Yu, Y. Yan, B. Yu, W. You, LncRNA CASC15 regulates breast cancer cell stemness via the miR-654-5p/MEF2D axis, J. Biochem. Mol. Toxicol. 36 (5) (2022) e23023, https://doi.org/10.1002/jbt.23023.
[32] M. Zhu, C. Zhang, P. Zhou, S. Chen, H. Zheng, LncRNA CASC15 upregulates cyclin D1 by downregulating miR-365 in laryngeal squamous cell carcinoma to promote cell proliferation, J Otolaryngol Head Neck Surg 51 (1) (2022) 8, https://doi.org/10.1186/s40463-022-00560-2.
[33] H. Wang, P. Zhang, [Corrigendum] lncRNA-CASC15 promotes osteosarcoma proliferation and metastasis by regulating epithelial-mesenchymal transition via the Wnt/β-catenin signaling pathway, Oncol. Rep. 47 (4) (2022), https://doi.org/10.3892/or.2022.8278.
[34] D. Bai, C. Guo, A. Wang, G. Pang, J. Gao, C. Wang, D. Zhao, J. Yang, J. Ren, LncRNA CASC15 promotes the proliferation of papillary thyroid carcinoma cells by regulating the miR-7151-5p/WNT7A axis, Pathol. Res. Pract. 225 (2021) 153561, https://doi.org/10.1016/j.prp.2021.153561.
[35] B. Qin, M. Dong, Z. Wang, J. Wan, Y. Xie, Y. Jiao, D. Yan, Long non-coding RNA CASC15 facilitates esophageal squamous cell carcinoma tumorigenesis via decreasing SIM2 stability via FTO-mediated demethylation, Oncol. Rep. 45 (3) (2021) 1059–1071, https://doi.org/10.3892/or.2020.7917.
[36] M.Y. Xue, H.X. Cao, Long non-coding RNA CASC15 promotes nasopharyngeal carcinoma cell proliferation and metastasis by downregulating miR-101-3p, Eur. Rev. Med. Pharmacol. Sci. 25 (3) (2021) 1166, https://doi.org/10.26355/eurrev_202102_24815.
[37] J. Sun, Y. Xiong, K. Jiang, B. Xin, T. Jiang, R. Wei, Y. Zou, H. Tan, T. Jiang, A. Yang, L. Jia, L. Wang, Hypoxia-sensitive long noncoding RNA CASC15 promotes lung tumorigenesis by regulating the SOX4/β-catenin axis, J. Exp. Clin. Cancer Res. 40 (1) (2021) 12, https://doi.org/10.1186/s13046-020-01806-5.
[38] D.J. Yu, M. Zhong, W.L. Wang, Long noncoding RNA CASC15 is upregulated in non-small cell lung cancer and facilitates cell proliferation and metastasis via targeting miR-130b-3p, Eur. Rev. Med. Pharmacol. Sci. 25 (4) (2021) 1765, https://doi.org/10.26355/eurrev_202102_25053.
[39] Y. Bai, G. Zhang, R. Cheng, R. Yang, H. Chu, CASC15 contributes to proliferation and invasion through regulating miR-766-5p/ KLK12 axis in lung cancer, Cell Cycle 18 (18) (2019) 2323–2331, https://doi.org/10.1080/15384101.2019.1646562.
[40] N. Jing, T. Huang, H. Guo, J. Yang, M. Li, Z. Chen, Y. Zhang, LncRNA CASC15 promotes colon cancer cell proliferation and metastasis by regulating the miR-4310/LGR5/Wnt/β-catenin signaling pathway, Mol. Med. Rep. 18 (2) (2018) 2269–2276, https://doi.org/10.3892/mmr.2018.9191.
[41] L. Sheng, R. Wei, Long non-coding RNA-CASC15 promotes cell proliferation, migration, and invasion by activating wnt/β-catenin signaling pathway in melanoma, Pathobiology 87 (1) (2020) 20–29, https://doi.org/10.1159/000502803.
[42] Q. Cheng, S. Zhang, B. Zhong, Z. Chen, F. Peng, Asiatic acid re-sensitizes multidrug-resistant A549/DDP cells to cisplatin by down regulating long non-coding RNA metastasis associated lung adenocarcinoma transcript 1/β-catenin signaling, Bioengineered 13 (5) (2022) 12972–12984, https://doi.org/10.1080/21655979.2022.2079302.
[43] J.E. Yu, J.A. Ju, N. Musacchio, T.J. Mathias, M.I. Vitolo, Long noncoding RNA DANCR activates wnt/β-catenin signaling through MiR-216a inhibition in non-small cell lung cancer, Biomolecules 10 (12) (2020), https://doi.org/10.3390/biom10121646.
[44] Y. Zhu, H. Wu, X. Yang, Z. Xiong, T. Zhao, X. Gan, LINC00514 facilitates cell proliferation, migration, invasion, and epithelial-mesenchymal transition in non-small cell lung cancer by acting on the Wnt/β-catenin signaling pathway, Bioengineered 13 (5) (2022) 13654–13666, https://doi.org/10.1080/21655979.2022.2084246.
[45] Y. He, X. Jiang, L. Duan, Q. Xiong, Y. Yuan, P. Liu, L. Jiang, Q. Shen, S. Zhao, C. Yang, Y. Chen, LncRNA PKMYT1AR promotes cancer stem cell maintenance in non-small cell lung cancer via activating Wnt signaling pathway, Mol. Cancer 20 (1) (2021) 156, https://doi.org/10.1186/s12943-021-01469-6.
[46] G. Su, Z. Yan, M. Deng, Sevoflurane inhibits proliferation, invasion, but enhances apoptosis of lung cancer cells by wnt/β-catenin signaling via regulating lncRNA PCAT6/miR-326 Axis, Open Life Sci. 15 (2020) 159–172, https://doi.org/10.1515/biol-2020-0017.
[47] S.J. Sun, Q. Lin, J.X. Ma, W.W. Shi, B. Yang, F. Li, Long non-coding RNA NEAT1 acts as oncogene in NSCLC by regulating the Wnt signaling pathway, Eur. Rev. Med. Pharmacol. Sci. 21 (3) (2017) 504–510.
[48] G. Gu, C. Hu, K. Hui, T. Chen, H. Zhang, X. Jiang, NEAT 1 knockdown enhances the sensitivity of human non-small-cell lung cancer cells to anlotinib, Aging (Albany NY) 13 (10) (2021) 13941–13953, https://doi.org/10.18632/aging.203004.
[49] R. Chang, X. Xiao, Y. Fu, C. Zhang, X. Zhu, Y. Gao, ITGB1-DT facilitates lung adenocarcinoma progression via forming a positive feedback loop with ITGB1/wnt/β-catenin/MYC, Front. Cell Dev. Biol. 9 (2021) 631259, https://doi.org/10.3389/fcell.2021.631259.
[50] S. Liu, N. Yang, L. Wang, B. Wei, J. Chen, Y. Gao, lncRNA SNHG11 promotes lung cancer cell proliferation and migration via activation of Wnt/β-catenin signaling pathway, J. Cell. Physiol. 235 (10) (2020) 7541–7553, https://doi.org/10.1002/jcp.29656.
[51] L.J. Tan, J.T. Liu, M. Yang, T. Ju, Y.S. Zhang, LncRNA ASB16-AS1 promotes proliferation and inhibits apoptosis of non small cell lung cancer cells by activating the Wnt/β catenin signaling pathway, Eur. Rev. Med. Pharmacol. Sci. 25 (4) (2021) 1766, https://doi.org/10.26355/eurrev_202102_25054.
[52] Z.X. Wang, Y. Zhao, Y. Yu, N. Liu, Q.X. Zou, F.H. Liang, K.P. Cheng, F.W. Lin, Effects of lncRNA SNHG20 on proliferation and apoptosis of non-small cell lung cancer cells through Wnt/β-catenin signaling pathway, Eur. Rev. Med. Pharmacol. Sci. 24 (1) (2020) 230–237, https://doi.org/10.26355/eurrev_202001_19915.
[53] Z. Zhang, X. Tan, R. Wu, T. Deng, H. Wang, X. Jiang, P. Zeng, J. Tang, m6A-mediated upregulation of lncRNA-AC026356.1 promotes cancer stem cell maintenance in lung adenocarcinoma via activating Wnt signaling pathway, Aging (Albany NY) 15 (9) (2023) 3538–3548, https://doi.org/10.18632/aging.204689.
[54] J. Zhu, K. Cao, P. Zhang, J. Ma, LINC00669 promotes lung adenocarcinoma growth by stimulating the Wnt/β-catenin signaling pathway, Cancer Med. 12 (7) (2023) 9005–9023, https://doi.org/10.1002/cam4.5604.
[55] Z. Qiu, X.X. Pan, D.Y. You, LncRNA DSCAM-AS1 promotes non-small cell lung cancer progression via regulating miR-577/HMGB1 axis, Neoplasma 67 (4) (2020) 871–879, https://doi.org/10.4149/neo_2020_190826N821.
[56] X. Han, H. Jiang, J. Qi, J. Li, J. Yang, Y. Tian, W. Li, Q. Jing, C. Wang, Novel lncRNA UPLA1 mediates tumorigenesis and prognosis in lung adenocarcinoma, Cell Death Dis. 11 (11) (2020) 999, https://doi.org/10.1038/s41419-020-03198-y.
[57] J. Pan, S. Fang, H. Tian, C. Zhou, X. Zhao, H. Tian, J. He, W. Shen, X. Meng, X. Jin, Z. Gong, lncRNA JPX/miR-33a-5p/Twist1 axis regulates tumorigenesis and metastasis of lung cancer by activating Wnt/β-catenin signaling, Mol. Cancer 19 (1) (2020) 9, https://doi.org/10.1186/s12943-020-1133-9.
[58] Y. Wu, K. Cheng, W. Liang, X. Wang, lncRNA RPPH1 promotes non-small cell lung cancer progression through the miR-326/WNT2B axis, Oncol. Lett. 20 (4) (2020) 105, https://doi.org/10.3892/ol.2020.11966.
[59] F. Li, T. Rong, G. Cao, C. Zhai, Q. Li, R. Gong, G. Li, AOC4P suppresses viability and invasion and induces apoptosis in NSCLC cells by inhibiting the Wnt/β-catenin pathway, Chem. Biol. Interact. 325 (2020) 109110, https://doi.org/10.1016/j.cbi.2020.109110.
[60] G. Cui, X. Fu, W. Wang, X. Chen, S. Liu, P. Cao, S. Zhao, LINC00476 suppresses the progression of non-small cell lung cancer by inducing the ubiquitination of SETDB1, Radiat. Res. 195 (3) (2021) 275–283, https://doi.org/10.1667/RADE-20-00105.1.
[61] X.Y. Shi, X.F. Tao, G.W. Wang, J.F. He, L.F. Wu, Y.Z. Sun, X.J. Sun, LncDBH-AS1 knockdown enhances proliferation of non-small cell lung cancer cells by activating the Wnt signaling pathway via the miR-155/AXIN1 axis, Eur. Rev. Med. Pharmacol. Sci. 25 (1) (2021) 139–144, https://doi.org/10.26355/eurrev_202101_24377.
[62] X. Zhang, M.H. Hamblin, K.-J. Yin, The long noncoding RNA Malat1: its physiological and pathophysiological functions, RNA Biol. 14 (12) (2017) 1705–1714, https://doi.org/10.1080/15476286.2017.1358347.
[63] A. Syllaios, D. Moris, G.S. Karachaliou, S. Sakellariou, I. Karavokyros, M. Gazouli, D. Schizas, Pathways and role of MALAT1 in esophageal and gastric cancer, Oncol. Lett. 21 (5) (2021) 343, https://doi.org/10.3892/ol.2021.12604.
[64] P. Ji, S. Diederichs, W. Wang, S. Böing, R. Metzger, P.M. Schneider, N. Tidow, B. Brandt, H. Buerger, E. Bulk, M. Thomas, W.E. Berdel, H. Serve, C. Müller-Tidow, MALAT-1, a novel noncoding RNA, and thymosin beta4 predict metastasis and survival in early-stage non-small cell lung cancer, Oncogene 22 (39) (2003) 8031–8041.
[65] Z. Fang, W. Chen, Z. Yuan, X. Liu, H. Jiang, LncRNA-MALAT1 contributes to the cisplatin-resistance of lung cancer by upregulating MRP1 and MDR1 via STAT3 activation, Biomed. Pharmacother. 101 (2018) 536–542, https://doi.org/10.1016/j.biopha.2018.02.130.
[66] X. Li, Q. Zhang, Z. Yang, Knockdown of hsa_circ_0058124 inhibits the proliferation of human lung cancer cells by up-regulation of miR-1297, Artif. Cells, Nanomed. Biotechnol. 48 (1) (2020) 584–593, https://doi.org/10.1080/21691401.2020.1725537.
[67] W. Bu, T. Luo, miR-1297 promotes cell proliferation of non-small cell lung cancer cells: involving in PTEN/Akt/Skp2 signaling pathway, DNA Cell Biol. 36 (11) (2017) 976–982, https://doi.org/10.1089/dna.2017.3886.
[68] D.T. Malleske, D. Hayes, S.W. Lallier, C.L. Hill, S.D. Reynolds, Regulation of human airway epithelial tissue stem cell differentiation by β-catenin, P300, and CBP, Stem Cell. 36 (12) (2018) 1905–1916, https://doi.org/10.1002/stem.2906.
[69] M. Gutiérrez-Salmerón, J.M. García-Martínez, J. Martínez-Useros, M. J. Fernández-Aceñero, B. Viollet, S. Olivier, J. Chauhan, S.R. Lucena, A. De la Vieja, C.R. Goding, A. Chocarro-Calvo, C. García-Jiménez, Paradoxical activation of AMPK by glucose drives selective EP300 activity in colorectal cancer, PLoS Biol. 18 (6) (2020) e3000732, https://doi.org/10.1371/journal.pbio.3000732.

[70] M. Kretz, D.E. Webster, R.J. Flockhart, C.S. Lee, A. Zehnder, V. Lopez-Pajares, K. Qu, G.X.Y. Zheng, J. Chow, G.E. Kim, J.L. Rinn, H.Y. Chang, Z. Siprashvili, P. A. Khavari, Suppression of progenitor differentiation requires the long noncoding RNA ANCR, Genes Dev. 26 (4) (2012) 338–343, https://doi.org/10.1101/gad.182121.111.
[71] Y. Bai, G. Zhang, H. Chu, P. Li, J. Li, The positive feedback loop of lncRNA DANCR/miR-138/Sox4 facilitates malignancy in non-small cell lung cancer, Am. J. Cancer Res. 9 (2) (2019) 270–284.
[72] Y.R. Chen, Y.S. Wu, W.S. Wang, J.S. Zhang, Q.G. Wu, Upregulation of lncRNA DANCR functions as an oncogenic role in non-small lung cancer by regulating miR-214-5p/CIZ1 axis, Eur. Rev. Med. Pharmacol. Sci. 24 (5) (2020) 2539–2547, https://doi.org/10.26355/eurrev_202003_20521.
[73] V. Malyla, K.R. Paudel, G. De Rubis, N.G. Hansbro, P.M. Hansbro, K. Dua, Cigarette smoking induces lung cancer tumorigenesis via upregulation of the WNT/β-catenin signaling pathway, Life Sci. 326 (2023) 121787, https://doi.org/10.1016/j.lfs.2023.121787.
[74] K. Liu, X. Fu, Z. Wang, L. Yang, J. Yang, H. Deng, Integrating network pharmacology prediction and experimental investigation to verify ginkgetin anti-invasion and metastasis of human lung adenocarcinoma cells via the Akt/GSK-3β/Snail and Wnt/β-catenin pathway, Front. Pharmacol. 14 (2023) 1135601, https://doi.org/10.3389/fphar.2023.1135601.
[75] L. Pang, Q. Zhang, Y. Wu, Q. Yang, J. Zhang, Y. Liu, R. Li, Long non-coding RNA CCAT1 promotes non-small cell lung cancer progression by regulating the miR-216a-5p/RAP2B axis, Exp Biol Med (Maywood) 246 (2) (2021) 142–152, https://doi.org/10.1177/1535370220961013.
[76] Y. Sun, B. Hu, Y. Wang, Z. Li, J. Wu, Y. Yang, Y. Wei, X. Peng, H. Chen, R. Chen, P. Jiang, S. Fang, Z. Yu, L. Guo, miR-216a-5p inhibits malignant progression in small cell lung cancer: involvement of the Bcl-2 family proteins, Cancer Manag. Res. 10 (2018) 4735–4745, https://doi.org/10.2147/CMAR.S178380.
[77] L. Yuan, J. Li, Y. Yang, Y. Chen, Y. Bu, M. Ye, X. Mao, T. Ma, L. Yu, Y. Nan, LINC00514 promotes gastric cancer cell growth and EMT progression via miR-204-3p/KRAS, Aging (Albany NY) 13 (8) (2021) 12007–12015, https://doi.org/10.18632/aging.202905.
[78] X. Luo, H. Wang, LINC00514 upregulates CCDC71L to promote cell proliferation, migration and invasion in triple-negative breast cancer by sponging miR-6504-5p and miR-3139, Cancer Cell Int. 21 (1) (2021) 180, https://doi.org/10.1186/s12935-021-01875-2.
[79] Q. Han, J. Li, J. Xiong, Z. Song, Long noncoding RNA LINC00514 accelerates pancreatic cancer progression by acting as a ceRNA of miR-28-5p to upregulate Rap1b expression, J. Exp. Clin. Cancer Res. 39 (1) (2020) 151, https://doi.org/10.1186/s13046-020-01660-5.
[80] X. Liu, X. Shen, J. Zhang, Long non-coding RNA LINC00514 promotes the proliferation and invasion through the miR-708-5p/HOXB3 axis in cervical squamous cell carcinoma, Environ. Toxicol. 37 (1) (2022) 161–170, https://doi.org/10.1002/tox.23387.
[81] J.P.H. Chow, R.Y.C. Poon, The CDK1 inhibitory kinase MYT1 in DNA damage checkpoint recovery, Oncogene 32 (40) (2013) 4778–4788, https://doi.org/10.1038/onc.2012.504.
[82] J. Lu, H. Zhong, T. Chu, X. Zhang, R. Li, J. Sun, R. Zhong, Y. Yang, M.S. Alam, Y. Lou, J. Xu, Y. Zhang, J. Wu, X. Li, X. Zhao, K. Li, L. Lu, B. Han, Role of anlotinib-induced CCL2 decrease in anti-angiogenesis and response prediction for nonsmall cell lung cancer therapy, Eur. Respir. J. 53 (3) (2019), https://doi.org/10.1183/13993003.01562-2018.
[83] L. Qi, F. Liu, F. Zhang, S. Zhang, L. Lv, Y. Bi, Y. Yu, lncRNA NEAT1 competes against let-7a to contribute to non-small cell lung cancer proliferation and metastasis, Biomed. Pharmacother. 103 (2018) 1507–1515, https://doi.org/10.1016/j.biopha.2018.04.053.
[84] Y. Wang, D. Zhang, Y. Li, Y. Wu, H. Ma, X. Jiang, L. Fu, G. Zhang, H. Wang, X. Liu, H. Cai, Constructing a novel signature and predicting the immune landscape of colon cancer using N6-methylandenosine-related lncRNAs, Front. Genet. 14 (2023) 906346, https://doi.org/10.3389/fgene.2023.906346.
[85] N. Jiang, Q. Guo, Q. Luo, Inhibition of ITGB1-DT expression delays the growth and migration of stomach adenocarcinoma and improves the prognosis of cancer patients using the bioinformatics and cell model analysis, J. Gastrointest. Oncol. 13 (2) (2022) 615–629, https://doi.org/10.21037/jgo-22-233.
[86] Z. Yousefian Naeini, N. Esfandiari, M. Hashemi, K. Hushmandi, S. Arbabian, M. Entezari, Introduced the ITGB1-DT as a novel biomarker associated with five potential drugs using bioinformatics analysis of breast cancer proteomics data and RT-PCR, Mol. Cell. Probes 71 (2023) 101930, https://doi.org/10.1016/j.mcp.2023.101930.
[87] B.-Q. Qiu, X.-H. Lin, S.-Q. Lai, F. Lu, K. Lin, X. Long, S.-Q. Zhu, H.-X. Zou, J.-J. Xu, J.-C. Liu, Y.-B. Wu, ITGB1-DT/ARNTL2 axis may be a novel biomarker in lung adenocarcinoma: a bioinformatics analysis and experimental validation, Cancer Cell Int. 21 (1) (2021) 665, https://doi.org/10.1186/s12935-021-02380-2.
[88] D. Li, X. Liu, N. Jiang, D. Ke, Q. Guo, K. Zhai, H. Han, X. Xiao, T. Fan, Interfering with ITGB1-DT expression delays cancer progression and promotes cell sensitivity of NSCLC to cisplatin by inhibiting the MAPK/ERK pathway, Am. J. Cancer Res. 12 (7) (2022) 2966–2988.
[89] X.-X. Zuo, Y. Yang, Y. Zhang, Z.-G. Zhang, X.-F. Wang, Y.-G. Shi, Platelets promote breast cancer cell MCF-7 metastasis by direct interaction: surface integrin α2β1-contacting-mediated activation of Wnt-β-catenin pathway, Cell Commun. Signal. 17 (1) (2019) 142, https://doi.org/10.1186/s12964-019-0464-x.
[90] J. Wang, P. Zhou, X. Wang, Y. Yu, G. Zhu, L. Zheng, Z. Xu, F. Li, Q. You, Q. Yang, W. Zhuo, J. Sun, Z. Chen, Rab25 promotes erlotinib resistance by activating the β1 integrin/AKT/β-catenin pathway in NSCLC, Cell Prolif. 52 (3) (2019) e12592, https://doi.org/10.1111/cpr.12592.
[91] Q. Wu, J. Ma, J. Wei, W. Meng, Y. Wang, M. Shi, lncRNA SNHG11 promotes gastric cancer progression by activating the wnt/β-catenin pathway and oncogenic autophagy, Mol. Ther. 29 (3) (2021) 1258–1278, https://doi.org/10.1016/j.ymthe.2020.10.011.
[92] W. Huang, F. Huang, Z. Lei, H. Luo, LncRNA SNHG11 promotes proliferation, migration, apoptosis, and autophagy by regulating hsa-miR-184/AGO2 in HCC, OncoTargets Ther. 13 (2020) 413–421, https://doi.org/10.2147/OTT.S237161.
[93] Z. Yang, Y. Ming, T. Wang, T. Zhi, Y. Zhou, M. Tian, lncRNA SNHG11 promotes the development of colorectal cancer by mediating miR-339-3p/SHOX2, Minerva Med. 113 (5) (2022) 898–899, https://doi.org/10.23736/S0026-4806.20.06619-7.
[94] R. Cheng, X. Lu, C. Xu, F. Zhang, G. Zhang, SNHG11 contributes to NSCLC cell growth and migration by targeting miR-485-5p/BSG axis, Biomed. Pharmacother. 128 (2020) 110324, https://doi.org/10.1016/j.biopha.2020.110324.
[95] J. Liang, D. Fang, F. Yao, L. Chen, Z. Zou, X. Tang, L. Feng, Y. Zhuang, T. Xie, P. Wei, P. Li, H. Zheng, S. Zhang, Analysis of shared ceRNA networks and related-hub genes in rats with primary and secondary photoreceptor degeneration, Front. Neurosci. 17 (2023) 1259622, https://doi.org/10.3389/fnins.2023.1259622.
[96] M. Yu, C. Shi, D. Xu, X. Lin, T. Ji, Z. Shi, X. Zhuge, S. Zhuo, Q. Yang, LncRNA ASB16-AS1 drives proliferation, migration, and invasion of colorectal cancer cells through regulating miR-185-5p/TEAD1 axis, Cell Cycle 21 (1) (2022), https://doi.org/10.1080/15384101.2021.1973700.
[97] W. Liu, R. Zhuang, S. Feng, X. Bai, Z. Jia, E. Kapora, W. Tan, Long non-coding RNA ASB16-AS1 enhances cell proliferation, migration and invasion via functioning as a ceRNA through miR-1305/Wnt/β-catenin axis in cervical cancer, Biomed. Pharmacother. 125 (2020) 109965, https://doi.org/10.1016/j.biopha.2020.109965.
[98] H. Chen, X. Tan, Y. Ding, Knockdown SNHG20 suppresses nonsmall cell lung cancer development by repressing proliferation, migration and invasion, and inducing apoptosis by regulating miR-2467-3p/E2F3, Cancer Biother. Radiopharm. 36 (4) (2021) 360–370, https://doi.org/10.1089/cbr.2019.3430.
[99] X. Lin, X. Tang, T. Zheng, J. Qiu, K. Hua, Long non-coding RNA AOC4P suppresses epithelial ovarian cancer metastasis by regulating epithelial-mesenchymal transition, J. Ovarian Res. 13 (1) (2020) 45, https://doi.org/10.1186/s13048-020-00644-5.
[100] T.-H. Wang, Y.-S. Lin, Y. Chen, C.-T. Yeh, Y.-L. Huang, T.-H. Hsieh, T.-M. Shieh, C. Hsueh, T.-C. Chen, Long non-coding RNA AOC4P suppresses hepatocellular carcinoma metastasis by enhancing vimentin degradation and inhibiting epithelial-mesenchymal transition, Oncotarget 6 (27) (2015) 23342–23357.
[101] L. Ye, T. Xiang, J. Zhu, D. Li, Q. Shao, W. Peng, J. Tang, L. Li, G. Ren, Interferon consensus sequence-binding protein 8, a tumor suppressor, suppresses tumor growth and invasion of non-small cell lung cancer by interacting with the Wnt/β-catenin pathway, Cell. Physiol. Biochem. 51 (2) (2018) 961–978, https://doi.org/10.1159/000495399.
[102] Y.-T. Li, X.-Y. Tan, L.-X. Ma, H.-H. Li, S.-H. Zhang, C.-M. Zeng, L.-N. Huang, J.-X. Xiong, L. Fu, Targeting LGSN restores sensitivity to chemotherapy in gastric cancer stem cells by triggering pyroptosis, Cell Death Dis. 14 (8) (2023) 545, https://doi.org/10.1038/s41419-023-06081-8.
[103] X.-Y. Tan, Y.-T. Li, H.-H. Li, L.-X. Ma, C.-M. Zeng, T.-T. Zhang, T.-X. Huang, X.-D. Zhao, L. Fu, WNT2-SOX4 positive feedback loop promotes chemoresistance and tumorigenesis by inducing stem-cell like properties in gastric cancer, Oncogene 42 (41) (2023) 3062–3074, https://doi.org/10.1038/s41388-023-02816-1.
[104] A. Dongre, R.A. Weinberg, New insights into the mechanisms of epithelial-mesenchymal transition and implications for cancer, Nat. Rev. Mol. Cell Biol. 20 (2) (2019) 69–84, https://doi.org/10.1038/s41580-018-0080-4.
[105] Y. Zhang, J. Tang, C. Wang, Q. Zhang, A. Zeng, L. Song, Autophagy-related lncRNAs in tumor progression and drug resistance: a double-edged sword, Genes Dis 11 (1) (2024) 367–381, https://doi.org/10.1016/j.gendis.2023.04.015.
[106] K. Chen, C. Ye, Z. Gao, J. Hu, C. Chen, R. Xiao, F. Lu, K. Wei, Immune infiltration patterns and identification of new diagnostic biomarkers GDF10, NCKAP5, and RTKN2 in non-small cell lung cancer, Transl Oncol 29 (2023) 101618, https://doi.org/10.1016/j.tranon.2023.101618.
[107] J. Li, J. Li, H. Hao, F. Lu, J. Wang, M. Ma, B. Jia, M. Zhuo, J. Wang, Y. Chi, X. Zhai, Y. Wang, M. Wu, T. An, J. Zhao, F. Yang, Z. Wang, Secreted proteins MDK, WFDC2, and CXCL14 as candidate biomarkers for early diagnosis of lung adenocarcinoma, BMC Cancer 23 (1) (2023) 110, https://doi.org/10.1186/s12885-023-10523-z.
[108] K. Yang, W. Zhang, L. Zhong, Y. Xiao, S. Sahoo, M. Fassan, K. Zeng, P. Magee, M. Garofalo, L. Shi, Long non-coding RNA HIF1A-As2 and MYC form a double-positive feedback loop to promote cell proliferation and metastasis in KRAS-driven non-small cell lung cancer, Cell Death Differ. 30 (6) (2023) 1533–1549, https://doi.org/10.1038/s41418-023-01160-x.
[109] T. Zhan, N. Rindtorff, M. Boutros, Wnt signaling in cancer, Oncogene 36 (11) (2017) 1461–1473, https://doi.org/10.1038/onc.2016.304.
[110] R. Nusse, H. Clevers, Wnt/β-Catenin signaling, disease, and emerging therapeutic modalities, Cell 169 (6) (2017) 985–999, https://doi.org/10.1016/j.cell.2017.05.016.
[111] X. Chen, T. Zhang, X. Ren, Y. Wei, X. Zhang, X. Zang, X. Ju, C. Qin, D. Xu, CHKB-AS1 enhances proliferation and resistance to NVP-BEZ235 of renal cancer cells via regulating the phosphorylation of MAP4 and PI3K/AKT/mTOR signaling, Eur. J. Med. Res. 28 (1) (2023) 588, https://doi.org/10.1186/s40001-023-01558-w.
[112] O. Rahbar Farzam, S. Najafi, M. Amini, Z. Rahimi, R. Dabbaghipour, O. Zohdi, G. Asemani Shahgoli, B. Baradaran, B. Akbari, Interplay of miRNAs and lncRNAs

in STAT3 signaling pathway in colorectal cancer progression, Cancer Cell Int. 24 (1) (2024) 16, https://doi.org/10.1186/s12935-023-03202-3.

[113] Y. Xu, Z. Qiu, J. Chen, L. Huang, J. Zhang, J. Lin, LINC00460 Promotes Neuroblastoma Tumorigenesis and Cisplatin Resistance by Targeting miR-149-5p/DLL1 axis and Activating Notch Pathway in Vitro and in Vivo, Drug Deliv Transl Res, 2023, https://doi.org/10.1007/s13346-023-01505-6.

[114] Y. Tang, W. Tian, S. Zheng, Y. Zou, J. Xie, J. Zhang, X. Li, Y. Sun, J. Lan, N. Li, X. Xie, H. Tang, Dissection of FOXO1-induced LYPLAL1-DT impeding triple-negative breast cancer progression via mediating hnRNPK/β-Catenin complex, Research 6 (2023) 289, https://doi.org/10.34133/research.0289.