

## Review

# Advances in Research on the Role of Long Non-Coding RNAs in Lung Cancer Diagnosis, Treatment, and Drug Resistance

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## Abstract

Lung cancer remains a leading cause of morbidity and mortality worldwide. This review critically synthesizes recent advances on long non-coding RNAs (lncRNAs) in lung cancer. Unlike previous descriptive compilations, we provide an evidence-based analysis, distinguish preclinical from clinically validated findings (e.g., serum exosomal SNHG15, DLX6-AS1), and highlight recurring mechanistic themes (ceRNA, chromatin remodeling, m<sup>6</sup>A). We discuss inconsistencies in the literature, barriers to clinical translation (such as standardization, sample variability, and validation), and propose a roadmap for clinical integration, including how lncRNA panels could complement existing biomarkers like CYFRA21-1. A transparent literature search strategy is included.

**Keywords:** lncRNA; lung cancer; research advances

## 1. Introduction

Globally, cancer incidence and death rates are increasing. In 2020, about 19.3 million new cancer cases were reported (18.1 million excluding non-melanoma skin cancer), with nearly 10 million deaths (9.9 million excluding non-melanoma skin cancer). Breast cancer in women has overtaken lung cancer as the most common, with approximately 2.3 million new cases (11.7%), followed by lung cancer (11.4%). Despite this, lung cancer remains the leading cause of cancer death, causing an estimated 1.8 million deaths (18%) [1]. Lung cancer is mainly classified into small cell lung cancer (SCLC) and non-small cell lung cancer (NSCLC), with NSCLC representing about 85% of cases [2]. Even with aggressive multimodal treatments, lung cancer prognosis stays poor, and its incidence remains high [3]. The molecular mechanisms behind lung cancer development and metastasis are still not fully understood, necessitating further research to identify biomarkers for diagnosis and therapy. ncRNAs are vital components of the genome, and their dysregulation can trigger and promote lung cancer (LC). Detecting ncRNA abnormalities can help predict disease progression, and targeting these ncRNAs might offer new therapeutic options [4]. Long non-coding RNAs (lncRNAs), longer than 200 nucleotides and non-coding, share several features with mRNAs [5]. Abnormal lncRNA expression not only contributes to lung cancer development but also affects patients' responses to conventional and targeted treatments [6]. This review highlights recent insights into the role of lncRNAs in lung cancer pathogenesis, explores their potential in diagnosis and therapy, and reviews their involvement in drug resistance.

Several excellent reviews have summarized lncRNA expression patterns in lung cancer over the past 3–5 years. However, most lack critical evidence grading, do not systematically address contradictory findings, and offer limited practical guidance for clinical implementation. This review distinguishes itself by: (i) stratifying evidence for



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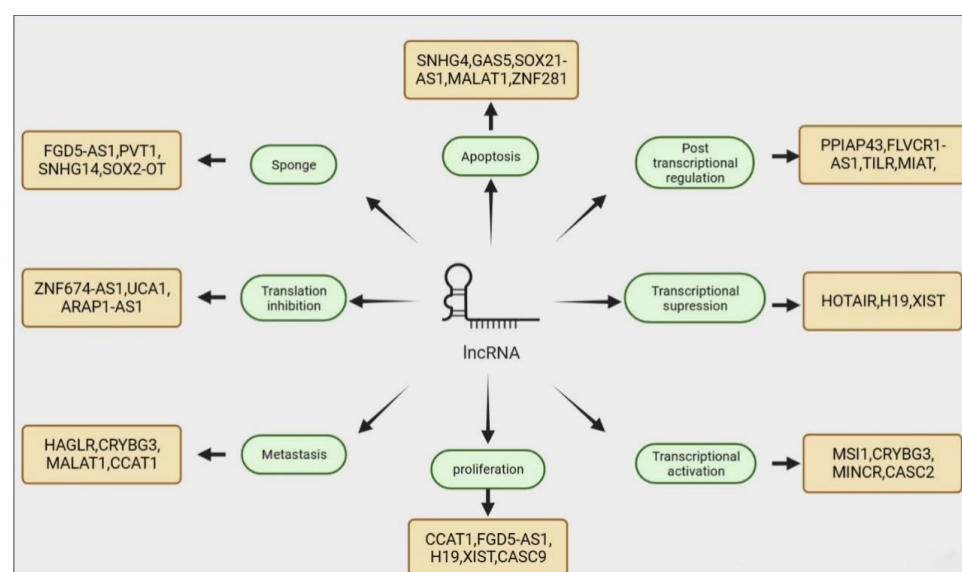
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each lncRNA claim (in vitro, animal, patient cohort, prospective validation); (ii) providing a synthesized framework of recurring mechanisms rather than listing individual axes; (iii) explicitly separating NSCLC vs. SCLC, histologic subtypes (LUAD/LUSC), and driver contexts (EGFR/ALK/KRAS); (iv) discussing sources of inconsistency across studies; and (v) proposing specific steps toward clinical translation (assay standardization, multi-center validation, integration with existing biomarkers).

## 2. Mechanisms and Functions of LncRNAs

Non-coding RNA (ncRNA) genes produce transcripts or functional RNAs that, unlike messenger RNAs, are not translated into proteins. Recently, long non-coding RNAs (lncRNAs), once thought to be non-functional, have attracted significant attention for their crucial role in mediating metastasis in head and neck cancer. Found in both the nucleus and cytoplasm, lncRNAs mainly exert their effects through epigenetic modifications, transcriptional regulation, and translational control [7]. Despite lacking protein-coding ability, lncRNAs regulate immune functions by modulating gene expression or protein activity. They participate in epigenetic regulation, interfere with downstream gene transcription, act as molecular sponges to influence miRNA activity, and bind to proteins to form nucleic acid-protein complexes, affecting protein function or cellular localization to control gene expression and immune responses [8]. These lncRNAs are characterized by unique regulatory mechanisms, diverse biological forms, cis-regulatory activity, and structured RNA domains [9]. A study by Ignacio E. Schor et al. on lncRNA expression during *Drosophila* embryogenesis examined various stages, tissue specificity, nuclear localization, and genetic backgrounds. The findings showed that nearly twice as many lncRNAs are expressed during embryonic stages as previously reported. Generally, lncRNA levels positively correlated with nearby genes, with minimal evidence of transcriptional interference [10]. Research indicates that lncRNA  $\beta$ Faaf promotes insulin synthesis and secretion by upregulating islet-specific genes, such as *Ins2*, *NeuroD1*, and *Creb1*, through miR-138-5p sponging [11]. Conversely, knockdown of *SOX2OT* enhances sepsis-induced hippocampal neurogenesis and cognitive function by downregulating *SOX2* in mice. Inhibiting *SOX2OT*/*SOX2* signaling could be an effective strategy for treating or preventing sepsis-related encephalopathy and neurodegeneration [12]. lncRNAs are classified into oncogenic and tumor-suppressive types (Figure 1).



**Figure 1.** Illustrates that the primary roles of lncRNAs encompass apoptosis, post-transcriptional regulation, transcriptional repression and activation, diffusion, metastasis, translational suppression, and functioning as molecular sponges, with different lncRNAs participating in these processes.

### 3. Applications of lncRNAs in Lung Cancer Diagnosis and as Biomarkers

The use of lncRNAs in diagnosing lung cancer and as biomarkers has gained widespread interest. Research indicates that, compared to normal tissues, specific lncRNAs are significantly different in lung cancer tissues, making them promising diagnostic markers. Most lncRNAs are located in the nucleus and are transcribed from intergenic regions, exons, or distal protein-coding regions of the genome by RNA polymerase II. Some lncRNAs are also found in the cytoplasm, though they are not translated [13]. lncRNAs can be readily detected in bodily fluids using common molecular techniques such as qRT-PCR, microarray hybridization, and sequencing. Circulating lncRNAs in bodily fluids can help distinguish early-stage tumor patients from healthy individuals, offering potential prognostic value. Many studies have shown that combining lncRNAs with traditional cancer biomarkers improves diagnostic accuracy [14]. Although their role in lung cancer diagnosis and treatment remains under investigation, lncRNAs hold significant promise. As our understanding of their functions and mechanisms grows, more lncRNAs related to lung cancer are likely to be identified and utilized in cancer care detection.

#### 3.1. lncRNAs as Biomarkers for Lung Cancer Detection

Abnormal expression of lncRNAs has been recognized as a common biomarker for lung cancer detection, offering several advantages. First, lncRNAs can enable earlier lung cancer diagnosis because their expression changes during the early stages of tumor formation. Second, the detection methods for lncRNAs are relatively simple and can be analyzed using routine laboratory techniques. For example, lncRNA MIR22HG in small cell lung cancer (SCLC) can regulate miR-9-3p levels and may serve as a potential biomarker for SCLC treatment [15]. lncRNA LINC01635 may play an important role in the progression of non-small cell lung cancer (NSCLC) by targeting miR-455-5p and could serve as a biomarker and therapeutic target for lung cancer [16]. Important lncRNAs are also found in serum exosomes, which can serve as biomarkers for lung cancer. For instance, increased expression of serum exosomal lncRNA SNHG15 predicts poor prognosis in NSCLC and may be a potential biomarker for early diagnosis and prognostic prediction of NSCLC [17]. Exosomal lncRNA DLX6-AS1 is significantly expressed in NSCLC patients, showing higher sensitivity and specificity than CYFRA21-1, suggesting that it could be a potential diagnostic marker for NSCLC [18]. Studies have shown that plasma exosomal lncRNA HAGLR is significantly reduced in NSCLC, suggesting that the disease may be at a later tumor stage and has a poor prognosis. lncRNA HAGLR and circulating tumor cells (CTCs) may serve as potential biomarkers for predicting the risk of NSCLC metastasis [19]. Additionally, upregulation of RFPL3S expression has been significantly correlated with poor prognosis in lung cancer, suggesting it may be a potential prognostic biomarker for lung cancer [20].

lncRNAs can regulate gene expression in lung cancer cells, influencing cellular activities such as proliferation, migration, invasion, and apoptosis. Studies have shown that PPIAP43 RNA transcription could serve as a potential biomarker for radiation sensitivity in small-cell lung cancer (SCLC) [21]. LINC02535 interacts with miR-30a-5p as a competitive endogenous RNA (ceRNA), thereby upregulating the expression of GALNT3, enhancing the function of MUC1, and activating the NF- $\kappa$ B signaling pathway, promoting the malignant progression of LUAD cells. [22]. The lncRNA LINC00662, driven by copy number amplification, promotes tumorigenesis through the EZH2/BIK axis, suggesting it as a potential molecular target for NSCLC [23–25].

### 3.2. Applications of LncRNAs in Lung Cancer Diagnosis

LncRNAs have already had a significant impact on the diagnosis of lung cancer. Studies have shown that the expression patterns of certain lncRNAs in lung cancer are closely associated with tumor development, metastasis, and prognosis. Moreover, research by Chhavi Gupta et al. has demonstrated that analyzing lncRNAs in nasal passages can serve as a non-invasive method for diagnosing lung cancer [26]. Therefore, lncRNAs can be used for lung cancer diagnosis and prognosis assessment. For example, in the lncRNA-associated ferroptosis model, the expression levels of AC125807.2, AL365181.3, AL606489.1, LINC02320, and AC099850.3 were upregulated, while SALRNA1, AC026355.1, and AP002360.1 were downregulated in NSCLC cell lines, with significant expression differences validated by RT-PCR, effectively predicting the prognosis of NSCLC [27]. In a study by Osama Sweef et al., total RNA from human bronchial epithelial BEAS-2B cells exposed to chronic Cr(VI) was analyzed using lncRNA, mRNA, and miRNA microarray techniques. By using lncRNA-miRNA interaction and miRNA target prediction algorithms, three carcinogenic pathways were identified (HOTAIRM1/miR-182-5p/ERO1A, GOLGA8B/miR-30d-5p/RUNX2, and PDCD6IPP2/miR-23a-3p/HOXA1), which have significant diagnostic and prognostic value [28]. After transfection with SNHG4-silencing plasmids, the survival rate of A549 cells was significantly suppressed, while apoptosis was promoted. lncRNA SNHG4 may have diagnostic and prognostic significance for non-small cell lung cancer (NSCLC) [29,30]. In a study by Chunlin Ke et al., the lncRNA HOTAIR rs1899663 C > A polymorphism was identified as a risk factor for lung cancer. lncRNA HOTAIR may have prognostic value in lung cancer screening [31]. Increased expression of lncRNA SNHG16 in cancers is associated with tumor progression and poor prognosis, suggesting that SNHG16 may be helpful in lung cancer diagnosis [32]. Research indicates that GAS5 inhibits cell growth and induces apoptosis. Downregulation of GAS5 expression in NSCLC tissues and plasma, followed by its increase after surgery, suggests that GAS5 downregulation is associated with a poor prognosis in NSCLC patients [33].

LncRNAs can be extracted and detected from the serum samples of lung cancer patients using routine laboratory techniques. For example, the long non-coding RNA LINC-PINT, which is downregulated in non-small cell lung cancer (NSCLC) patients, can be detected as a diagnostic and prognostic biomarker using reverse transcription quantitative PCR [34].

Certain lncRNAs can participate in signaling pathways in lung cancer, interacting with key factors to influence the biological activities of lung cancer cells. For example, PGM5P4-AS1 contains binding sites for miR-1275 and can positively regulate LZTS3 expression via miR-1275 sponging, making it a potential candidate for the precise diagnosis of lung cancer [35]. Research using Gene Set Enrichment Analysis (GSEA) revealed that LINC01614 is the most aberrantly expressed lncRNA in NSCLC tissues and may be involved in TGF- $\beta$ -, P53-, IGF-IR-, Wnt-, and RTK/Ras/MAPK-mediated signaling pathways, potentially assisting in the diagnosis of NSCLC [36].

## 4. LncRNAs Associated with Lung Cancer Development and Metastasis

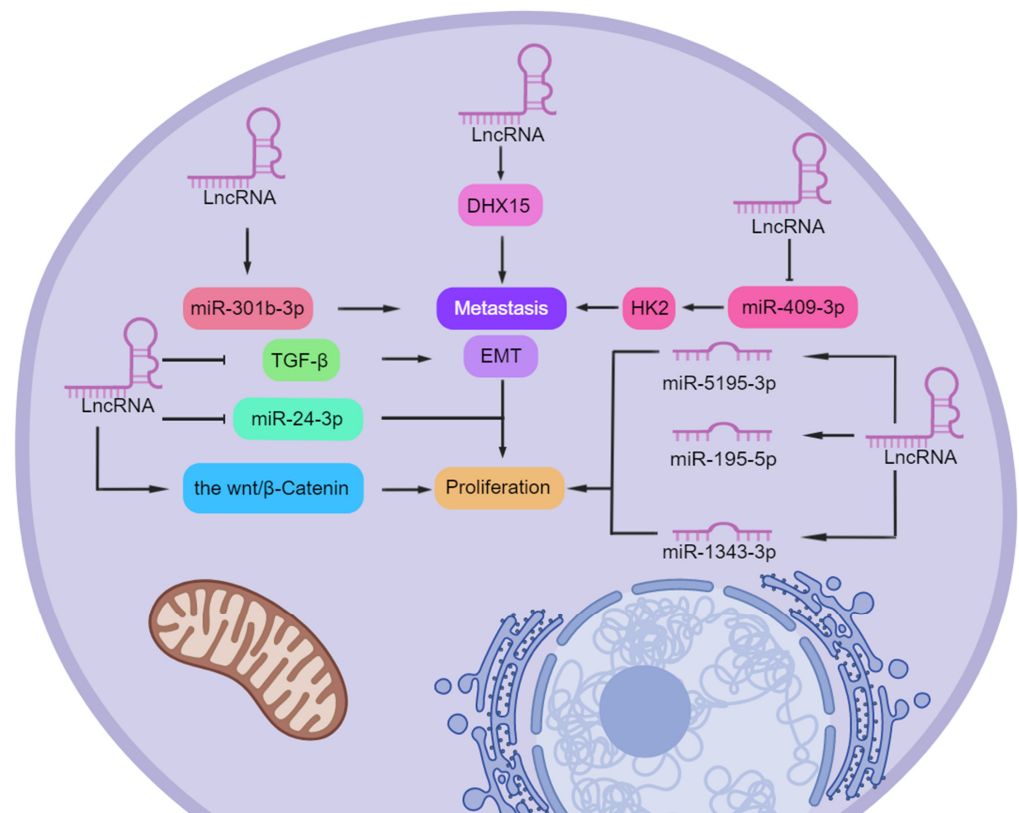
During lung cancer development, ncRNAs can participate in tumor cell transformation and proliferation by regulating gene expression, cell proliferation, apoptosis, and other pathways. Specific lncRNAs and miRNAs have been shown to play regulatory roles in lung cancer development and metastasis, including regulating tumor-associated signaling pathways, promoting cell proliferation and invasiveness, and inhibiting apoptosis.

Moreover, ncRNAs can also participate in the metastatic process of lung cancer by regulating the migratory and invasive capabilities of lung cancer cells. Metastasis is one of the major challenges in lung cancer treatment, as metastatic lung cancer is often resistant to therapy. Several studies have shown that certain lncRNAs and miRNAs play important

roles in lung cancer metastasis, such as regulating epithelial–mesenchymal transition, cell migration, and invasiveness. Aberrant expression of these ncRNAs may enhance the metastatic and invasive capabilities of lung cancer cells, thereby promoting metastasis.

#### 4.1. Regulatory Mechanisms of LncRNAs in Lung Cancer Development and Metastasis

LncRNAs regulate the expression of lung cancer-related genes by interacting with transcription factors and chromatin-modifying enzymes, and by modulating miRNA function, thereby influencing the biological characteristics of lung cancer cells through multiple molecular mechanisms. For example, M2 macrophage-derived exosomal lncRNA AGAP2-AS1 enhances the radiosensitivity of lung cancer by reducing microRNA-296 and increasing NOTCH2 [37]. LncRNA CCAT1 regulates tumor cell proliferation and invasion in NSCLC by inhibiting miR-152 [38]. Long non-coding RNA SMASR suppresses lung cancer EMT by negatively regulating the TGF- $\beta$ /Smad signaling pathway [39] (Figure 2).



**Figure 2.** Mechanisms of LncRNAs Associated with Lung Cancer Development and Metastasis. LncRNAs primarily induce tumor metastasis through the DHX15, miR-409-3p to HK2, and miR-301b-3p pathways. They can also influence TGF- $\beta$ -induced EMT mechanisms, triggering tumor metastasis. Additionally, oncogenic pathways, such as Wnt/ $\beta$ -Catenin signaling, can induce lncRNA upregulation, promoting cancer dissemination.

#### 4.2. Molecular Pathways of LncRNAs Associated with Metastasis in Lung Cancer

In this process, some lncRNAs regulate the expression of transcription factors and cell adhesion proteins, thereby affecting cell morphology and cell–cell interactions and promoting the metastasis of lung cancer cells.

Certain lncRNAs promote the proliferation and metastasis of lung cancer cells by activating and controlling specific proteins and interacting with them. For example, the exogenous overexpression of DLX6-AS1-encoded peptides promotes NSCLC cell growth by activating the Wnt/ $\beta$ -catenin pathway [40,41]. Long non-coding RNA LALTOP stabilizes topoisomerase II $\alpha$  mRNA to promote NSCLC [42], while FGD5-AS1 enhances NSCLC cell

proliferation and migration by upregulating DDX5 [43]. lncRNA JHDM1D-AS1 interacts with DHX15 protein to promote NSCLC growth and metastasis [44]. Studies have shown that lncRNA H19 mediates lung cancer cell proliferation and invasion by upregulating N-cadherin and vimentin, while downregulating E-cadherin [45]. lncRNAs CASC9 and HIF-1 $\alpha$  form a positive feedback loop to promote cancer cell proliferation and metastasis [46].

Some lncRNAs can regulate cytokine and transcription factor expression, thereby promoting the growth of lung cancer cells. For example, lnc-RAB1A-2, activated by RAB1A, can influence the expression of fibroblast growth factor 1 (FGF1), a gene involved in the PI3K/AKT/mTOR pathway, thereby exacerbating lung cancer prognosis by upregulating FGF1 [47,48]. lncRNA FLVCR1-AS1 promotes cancer proliferation and invasion by absorbing miR-573 and upregulating the expression of E2F transcription factor 3 [49].

Additionally, some lncRNAs can regulate signaling pathways during lung cancer cell invasion and metastasis, which are crucial to the metastatic process. lncRNAs can regulate the activity and effects of these signaling pathways by interacting with key molecules.

Some upregulated lncRNAs influence lung cancer cell signaling pathways, thereby affecting cell proliferation and migration. For example, the lncRNA-ATB/miR-200b axis accelerates NSCLC cell proliferation, migration, and invasion by upregulating FN1 [50]. IL-1 $\beta$ -induced upregulation of lncRNA-CHRF worsens NSCLC by regulating the miR-489/Myd88 axis [51]. The JMJD2C-mediated MALAT1/miR-503-5p/SEPT2 axis is involved in NSCLC progression, contributing to disease worsening [52]. LINC01748 acts as a ceRNA by sponging miR-520a-5p, leading to HMGA1 overexpression and increasing NSCLC cell invasiveness [53]. OIP5-AS1 enhances PD-L1 expression and promotes cell proliferation in NSCLC by sponging miR-34a [54]. LNC11649 interacts with MSI1 to promote its cytoplasmic distribution and subsequently activates the Akt signaling pathway, regulating NSCLC cell proliferation and migration [55]. lncRNA ZEB2-AS1 is upregulated in NSCLC, which elevates the viability and malignant degree of NSCLC cells by downregulating PTEN, thus aggravating the progression of NSCLC [56]. lncRNA CRYBG3 promotes lung cancer metastasis by activating the eEF1A1/MDM2/MTBP axis [57]. lncRNA CCAT1 sponges miR-490 to enhance cell proliferation and migration in NSCLC [58]. The lncRNA JPX/miR-33a-5p/Twist1 axis regulates tumorigenesis and metastasis in lung cancer by activating Wnt/ $\beta$ -catenin signaling. The JPX/miR-33a-5p/Twist1 axis may serve as a novel ceRNA regulatory network, participating in the EMT process and accelerating lung cancer malignancy by activating Wnt/ $\beta$ -catenin signaling [59].

Some downregulated lncRNAs influence lung cancer cell signaling pathways, thereby promoting cell proliferation and migration. Studies have shown that overexpression of miR-24-3p inhibits lung cancer cell proliferation and promotes apoptosis. SOX21-AS1 negatively regulates the miR-24-3p/PIM2 axis to promote cell proliferation, migration, and invasion [60]. MCM3AP-AS1 acts as a competitive endogenous RNA (ceRNA) regulating the miR-195-5p/E2F3 axis, promoting NSCLC [61]. Long non-coding RNA SMASR inhibits lung cancer EMT by negatively regulating the miR-24-3p/Smad signaling pathway [62]. Overexpression of Linc00485 downregulates miR-298, leading to the upregulation of c-Myc, thereby promoting lung cancer [63].

lncRNAs can also affect the metastatic ability of lung cancer by regulating processes such as cell cycle, apoptosis, and angiogenesis. They can interact with multiple regulatory factors and effector molecules to regulate these biological processes. For example, lncRNA CYB561-5 promotes aerobic glycolysis and tumorigenesis in non-small cell lung cancer (NSCLC) by interacting with basigin [64]. However, lncRNA DUXAP8 regulates HK2 and LDHA by inhibiting miR-409-3p, promoting cell viability, migration, and glycolysis in NSCLC [65]. Studies have shown that lncRNA MCF2L-AS1, as an oncogene in NSCLC, controls CCDN1 via ELAVL1, driving NSCLC cell growth [66]. Long non-coding RNA

AFAP1-AS1 accelerates lung cancer cell migration and invasion by interacting with SNIP1 and upregulating c-Myc [67]. lncRNA SNHG4 enhances KDM3A and p21 expression, ultimately promoting the tumorigenicity of NSCLC cells in vivo [68]. LCAT3 is a novel m6A-regulated lncRNA that exerts oncogenic effects in lung cancer by binding with FUBP1 to activate c-MYC [69]. lnc-THOR promotes the growth ability of NSCLC cells by interacting with IGF2BP1 [70]. lncRNA snaR promotes the proliferation of EGFR wild-type NSCLC cells [71]. TP73-AS1 contributes to proliferation, migration, and cisplatin resistance, but inhibits apoptosis in NSCLC cells by upregulating TRIM29 and absorbing miR-34a-5p [72]. Research shows that LINC01224 promotes tumor progression and cisplatin resistance in NSCLC by sponging miR-2467 [73]. lncRNA-CCAT1 enhances tumor growth in NSCLC by directly inhibiting miR-218 and indirectly increasing BMI-1 expression [74]. Studies have shown that lncRNA MIAT regulates NSCLC migration and invasion by modulating miR-139-5p and MMP2 [75]. lncRNA LNBC3 promotes NSCLC by stabilizing BCL6 [76]. lncRNA PVT1 promotes cell growth and metastasis in NSCLC by regulating the miR-361-3p/SOX9 axis and activating the Wnt/ $\beta$ -catenin signaling pathway [77]. Long non-coding RNA TILR constitutively inhibits TP53 and apoptosis in lung cancer, collaborating with PCBP2 to constitutively suppress p53 expression, thereby maintaining p53 transcriptional activity at sufficiently low levels to avoid false apoptosis induction [78]. IGF2BP3 promotes lung cancer cell development by binding with the CERS6-AS1 promoter and downregulating miR-1202, which may be associated with the upregulation of GPD5 [79]. Table 1 summarizes the lncRNAs that affect the occurrence and metastasis of lung cancer through expression dysregulation.

**Table 1.** Association of lncRNA with metastasis of lung cancer cells.

| Function                                                  | lncRNA Name | Signaling Pathways/Axis    | Potential Targets          | References |
|-----------------------------------------------------------|-------------|----------------------------|----------------------------|------------|
| Promoted proliferation and migration of lung cancer cells | AFAP1-AS1   | Not reported               | AFAP1 protein              | [80]       |
|                                                           | SLNCR1      | Not reported               | secretory phospholipase A2 | [81]       |
|                                                           | TCL6        | PI3K/AKT signaling         | PDK1                       | [82]       |
|                                                           | FGD5-AS1    | hsa-miR-107                | FGFRL1                     | [83]       |
|                                                           | LINC00662   | miR-320d/E2F1              | E2F1                       |            |
|                                                           | LINC00847   | miR-147a/IFITM1            | IFITM1                     | [84]       |
|                                                           | JPX         | Not reported               | miR-145-5p                 | [85]       |
|                                                           | DNACR       | miR-1225-3p/ErbB2          | ErbB2                      | [86]       |
|                                                           | HAR1A       | ANXA2/p65                  | ANXA2                      | [87]       |
|                                                           | LINC00511   | miR-625-5p/GSPT1           | GSPT1                      | [88]       |
|                                                           | LINC00839   | miR-17-5p/NF- $\kappa$ B   | NF- $\kappa$ B             | [89]       |
|                                                           | PVT1        | miR-17-5p, miR-361-3p/SOX9 | BAMBI, SOX9                | [90,91]    |
|                                                           | CASC19      | Wnt/ $\beta$ -catenin      |                            |            |
|                                                           | MALAT1      | miR-301b-3p/LDLR           | LDLR                       | [92]       |
|                                                           | FEZF1-AS1   | miR-374b-5p/SRSF7          | SRSF7                      | [93]       |
|                                                           | ATB         | ITGA11/miR-516b-5p         | miR-516b-5p                | [94]       |
|                                                           | B4GALT1-AS1 | Not reported               | miR-141-3p                 | [95]       |
|                                                           | SLCO4A1-AS1 | miR-30e/SOX9               | SOX9                       | [96]       |
|                                                           | SNHG6       | Not reported               | IKK $\alpha$               | [97]       |
|                                                           | HOTAIR      | Not reported               | miR-101-3p                 | [98]       |
|                                                           | MIAT        | Not reported               | HNRNPA1                    | [99]       |
|                                                           | CHRF        | miR-128-3p/PELI3           | PELI3,                     | [100]      |
|                                                           | TP73-AS1    | microRNA-489/Myd88         | Myd88                      | [101]      |
|                                                           | DDX11-AS1   | miR-27b-3p/LAPTM4B         | LAPTM4B                    | [102]      |
|                                                           | OECC        | PI3K/AKT                   | AKT                        | [103]      |
|                                                           | MRPL39      | PI3K/Akt/mTOR              | mTOR                       | [104]      |
|                                                           | LUCAT1      | miR-130/TSC1               | TSC1                       | [105]      |
|                                                           | NORAD       | miR-301b/STAT3             | STAT3                      | [106]      |
|                                                           | HOXA11-AS   | Not reported               | miR-136-5p                 | [107]      |
|                                                           | 1308        | RNA-148a-3p/DNMT1          | DNMT1                      | [108]      |
|                                                           | DLGAP1-AS1  | miR-124/ADAM 15            | ADAM 15                    | [109]      |
|                                                           | AL513318.2  | miR-193a-5p/DTL            | DTL                        | [110]      |
|                                                           | TRG-AS1     | Not reported               | SLC6A8                     | [111]      |
|                                                           |             | miR-224-5p/SMAD4           | SMAD4                      | [112]      |

Table 1. Cont.

| Function                                        | LncRNA Name | Signaling Pathways/Axis | Potential Targets                      | References |
|-------------------------------------------------|-------------|-------------------------|----------------------------------------|------------|
| Contributes to cancer stem cell-like phenotypes | CAR10       | miR-892a/GJB2           | GJB2                                   | [113]      |
|                                                 | LBX2-AS1    | Notch signaling         | Notch1, p21, Hes1                      | [114]      |
|                                                 | UCA1        | Not reported            | MAPK1                                  | [115]      |
|                                                 | NCK1-AS1    | Not reported            | miR-137                                | [116]      |
|                                                 | TRPM2-AS    | Not reported            | EGFR                                   | [117]      |
|                                                 | SNHG14      | miR-382-5p              | SPIN1, miR-340                         | [118]      |
|                                                 | FAM83A-AS1  | Not reported            | HIF-1 $\alpha$                         | [119]      |
|                                                 | DUXAP8      | Not reported            | A549 cell                              | [120]      |
|                                                 | AWPPH       | Not reported            | TGF- $\beta$ 1                         | [121]      |
|                                                 | SNHG15      | miR-211-3p              | ZNF217                                 | [122]      |
|                                                 | DANCR       | Not reported            | p21                                    | [123]      |
|                                                 | LINC01288   | Not reported            | IL-6                                   | [124]      |
| Enhances radiotherapy immunity in lung cancer   | Linc00662   | Not reported            | Lin28                                  | [125]      |
|                                                 | TDRG1       | Not reported            | Sox2 mRNA                              | [126]      |
| Promote glycolysis of lung cancer cells.        | AGAP2-AS1   | Not reported            | microRNA-296, NOTCH2                   | [127]      |
| Inhibition of lung cancer cell apoptosis        | HOTTIP      | miR-615-3p/HMGB3        | HMGB3                                  | [128]      |
|                                                 | LINC01123   | miR-199a-5p/c-Myc       | c-Myc                                  | [129]      |
|                                                 | LINC00665   | let-7c-5p/HMMR          | HMMR                                   | [130]      |
|                                                 | LINC00243   | Not reported            | PDK4                                   | [131]      |
|                                                 | DLEU1       | Not reported            | CDK1, SRC, P70 (S6K), MMP2, E-cadherin | [132]      |
|                                                 | KDM5BAS1    | Not reported            | H1838, H1299 cell                      | [133]      |
|                                                 | LNC511      | miR-625/LOXL4           | LOXL4                                  | [134]      |

Footnote: “Not reported” indicates that the information was not available in the original study; ceRNA, competing endogenous RNA; EMT, epithelial–mesenchymal transition.

#### 4.3. Key Recurring Mechanisms and Prioritized LncRNAs

From the extensive list of lncRNAs described above, several mechanistic themes consistently emerge across independent studies. The most robust and reproducible include: (i) ceRNA networks involving PVT1, JPX, SNHG families, and MALAT1, which sponge multiple miRNAs to derepress oncogenes; (ii) chromatin remodeling via EZH2 recruitment by LINC00662, HOTAIR, and CASC9; (iii) RNA-binding protein stabilization as seen with AFAP1-AS1/SNIP1/c-Myc and THOR/IGF2BP1; (iv) exosome-mediated intercellular communication exemplified by AGAP2-AS1 and SNHG15; and (v) m<sup>6</sup>A-dependent regulation typified by LCAT3.

Among the dozens of lncRNAs reported, only a subset have been validated in independent patient cohorts or functional animal models with consistent results. These include: PVT1 (proliferation, metastasis, and resistance; validated in  $\geq 3$  cohorts), MALAT1 (metastasis and prognosis; meta-analysis available), HOTAIR (prognostic polymorphism; replicated), SNHG15 (serum exosomal diagnostic biomarker; AUC > 0.8 in two cohorts), and DLX6-AS1 (diagnostic performance superior to CYFRA21-1). In contrast, many other lncRNAs (e.g., LINC00847, TRG-AS1, AL513318.2) are supported only by single studies or in vitro data and should be considered preliminary.

### 5. LncRNA-Mediated Response to Treatment and Chemoresistance in Lung Cancer

While the previous section focused on lncRNAs driving metastasis, here we examine their equally critical roles in therapeutic response and acquired resistance.

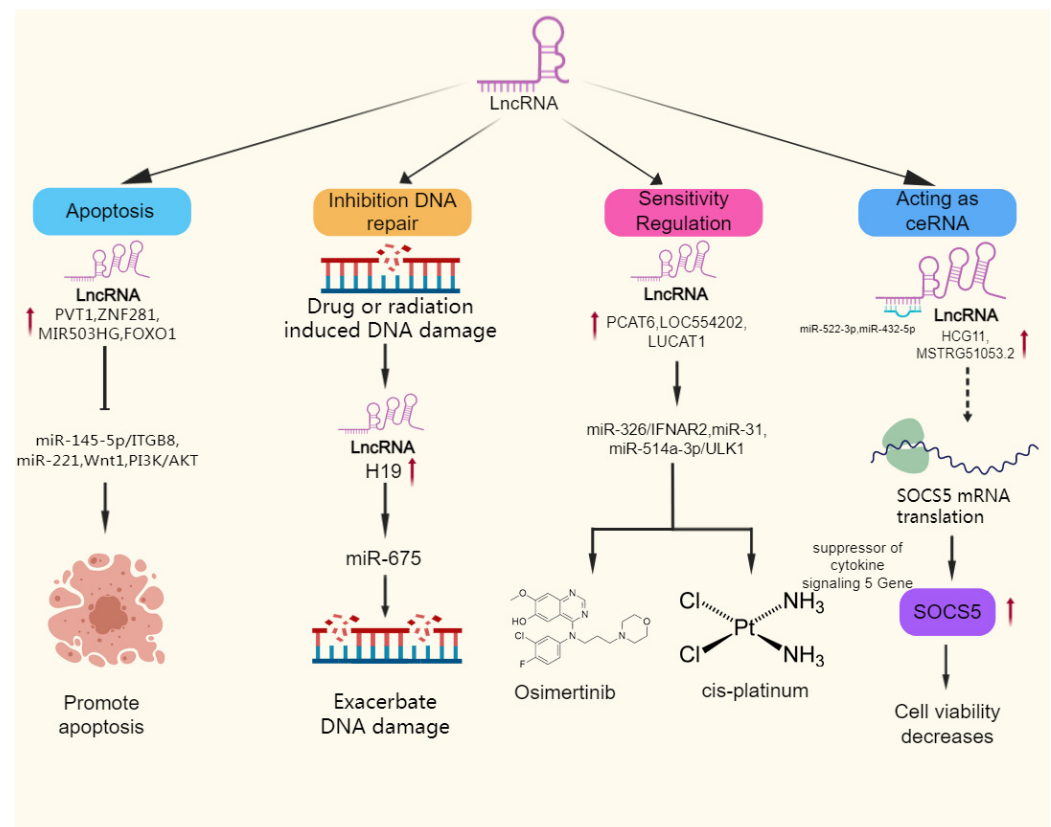
Lung cancer treatment involves various strategies such as chemotherapy, radiotherapy, targeted therapy, and immunotherapy to inhibit the growth and spread of lung cancer cells.

Chemically modified oligonucleotides and nuclear targeting offer potential ncRNA-based therapies [135]. However, the response of lung cancer cells to treatment and their associated resistance remain significant clinical challenges. lncRNAs play an essential regulatory role in these processes.

Some lncRNAs regulate the sensitivity of lung cancer cells to chemotherapy drugs. They can influence the response of lung cancer cells to chemotherapeutic agents by modulating the expression of drug targets, regulating cell apoptosis, and controlling key biological processes such as the cell cycle. Additionally, certain lncRNAs can affect the DNA damage repair capacity of lung cancer cells, influencing their sensitivity to radiotherapy.

### 5.1. The Mechanisms of lncRNA Mediating the Response to Lung Cancer Treatment

lncRNAs can influence treatment response by regulating gene expression in lung cancer cells. lncRNAs act as regulators of transcription factors, chromatin-modifying factors, or RNA-binding proteins, interacting with DNA, RNA, or proteins to regulate the expression of specific genes. By modulating the expression levels of these genes, lncRNAs can affect lung cancer cells' response to treatment. lncRNAs can also regulate the activation or inhibition of signaling pathways by interacting with proteins associated with these pathways, thereby influencing the response of lung cancer cells to treatment (Figure 3).



**Figure 3.** Mechanisms of lncRNA in mediating the treatment response in lung cancer. lncRNAs mediate various molecular mechanisms, such as regulating drug sensitivity, promoting tumor cell apoptosis, inhibiting tumor DNA damage repair, and reducing tumor cell viability. Flat arrow: Inhibition; Red arrow: Upregulation; Black arrow: Direct effect; Dashed arrow: Indirect effect (via pathways/signaling axes).

### 5.2. Application of lncRNAs in Lung Cancer Treatment

Long non-coding RNAs (lncRNAs) have gradually become a research hotspot in lung cancer treatment. Certain lncRNAs can influence key processes such as proliferation, differentiation, and apoptosis of lung cancer cells through regulating gene expression

and cell signaling pathways, thereby affecting treatment outcomes. lncRNAs can also regulate signaling pathways in lung cancer cells, thereby inhibiting their growth and migration. For example, knockdown of Circ\_0072088 can partially suppress cell growth, migration, invasion, and glycolysis by regulating the miR-1225-5p/WT1 axis [136]. The GGGT haplotype in lncRNA MALAT1 suppresses brain metastasis and lung cancer lymph node metastasis through the MALAT1/miR-328/KATNB1 pathway [137]. INC00174 binds to miR-584-3p targeting RPS24, and inhibition of LINC00174 can alleviate the malignant phenotype of LC cells by regulating the miR-584-3p/RPS24 axis [138]. Studies have shown that MEG3, as a tumor suppressor, inhibits NSCLC cell migration and invasion by sponging miR-21-5p and partially enhancing PTEN expression through the PI3K/AKT signaling pathway [139]. Knockdown of PVT1 inhibits proliferation, migration, and invasion by regulating the miR-145-5p/ITGB8 axis and suppressing the MEK/ERK signaling pathway, but induces NSCLC cell apoptosis [140]. ZNF674-AS1 inhibits NSCLC cell migration and invasion by regulating the miR-23a/E-cadherin axis [141].

Certain lncRNAs influence lung cancer cells and affect lung cancer treatment by regulating their own expression and that of other genes. For example, chemoresensitization experiments show that overexpression of Lnc-LSAMP-1 forms a complex that protects the LSAMP gene from degradation, thereby enhancing the inhibitory effect of Tyrosine kinase inhibitors (TKIs) on cell proliferation [142]. The single-nucleotide polymorphism rs4142441 and MYC co-regulated lncRNA OSER1-AS1 suppresses non-small cell lung cancer (NSCLC) by isolating ELAVL1 [143]. Studies have shown that knockdown of PCAT1 reduces cell proliferation and stem cell characteristics (Sox2 and Nanog expression) in H226 and A549 cells in vitro, and inhibits tumor development in vivo [144]. ZNF281 may upregulate PTEN by downregulating miR-221 in NSCLC, thereby inhibiting cancer cell proliferation and promoting apoptosis [145]. Overexpression of MIR503HG inhibits cell proliferation and promotes apoptosis in vitro, and suppresses tumorigenesis in vivo. MIR503HG suppresses NSCLC by downregulating Wnt1 expression [146]. Inhibition of lncRNA UCA1 significantly suppresses the migration and invasion of NCI-H23 lung cancer through the inhibition of mitogen-activated protein kinase 1 (MAPK1) [147]. lncRNA PGM5P4-AS1 inhibits lung cancer by sponging microRNA miR-1275 and upregulating leucine zipper tumor suppressor 3 (LZTS3) [148]. BCYRN1 is upregulated in NSCLC, and its downregulation can inhibit NSCLC cell proliferation and cell cycle progression by suppressing the Wnt/ $\beta$ -catenin pathway [149] (Table 2).

**Table 2.** Summarizes the lncRNAs that influence lung cancer treatment.

| Function                                     | lncRNA Name | Dysregulation-On | Signaling Pathways/Axis | Potential Targets | References |
|----------------------------------------------|-------------|------------------|-------------------------|-------------------|------------|
| Inhibition of lung cancer cell proliferation | UC.77       | ↓                | Not reported            | p21               | [150]      |
|                                              | LINRIS      | ↓                | Not reported            | MiR-10a           | [151]      |
|                                              | ARAP1-AS1   | ↓                | Not reported            | Cyclin D1         | [152]      |
|                                              | LOC285194   | ↓                | KRAS/BRAF/SMEK          | p53               | [153]      |
|                                              | GATA2-AS1   | ↑                | Not reported            | GATA2             | [154]      |
|                                              | DUXAP8      | ↓                | Not reported            | EZH2, LSD1        | [155]      |
|                                              | NEF         | ↑                | Not reported            | GLUT1             | [156]      |
|                                              | FER1L4      | ↑                | PI3K/Akt                | Akt               | [157]      |
|                                              | BRE-AS1     | ↑                | Not reported            | NR4A3             | [158]      |
|                                              | SNHG9       | ↓                | Not reported            | YAP               | [159]      |
| Effects on the cell cycle                    | HCG11       | ↑                | miR-875/SATB2           | MiR-875           | [160]      |
|                                              | CCAT1       | ↓                | miR-216a-5p/RAP2B       | RAP2B             | [161]      |
|                                              | ARAP1-AS1   | ↓                | Not reported            | Cyclin D1         | [152]      |
|                                              | SFTA1P      | ↑                | PI3K-AKT                | AKT               | [162]      |
|                                              | DLX6-AS1    | ↓                | miR-16-5p/BMI1          | BMI1              | [163,164]  |

Table 2. Cont.

| Function                                                   | LncRNA Name | Dysregulation-On | Signaling Pathways/Axis          | Potential Targets | References |
|------------------------------------------------------------|-------------|------------------|----------------------------------|-------------------|------------|
| Promote cell apoptosis                                     | CASC7       | ↑                | Not reported                     | miR-92a           | [165]      |
|                                                            | FER1L4      | ↑                | PTEN/AKT/p53                     | p53               | [166]      |
|                                                            | MT1JP       | ↑                | miRNA-423-3p/Bim                 | Bim               | [167]      |
|                                                            | FOXO1       | ↑                | PI3K/AKT                         | AKT               | [168]      |
|                                                            | MINCR       | ↓                | Not reported                     | c-Myc             | [169]      |
| Inhibition of lung cancer cell invasion                    | GATA6-AS1   | ↑                | Not reported                     | miR-324-5p        | [170]      |
|                                                            | HCG11       | ↑                | Not reported                     | SOCS5             | [171]      |
|                                                            | BX357664    | ↑                | Not reported                     | N/A               | [172]      |
|                                                            | LINC01124   | ↑                | Not reported                     | N/A               | [173]      |
|                                                            | NKILA       | ↑                | IL-11/STAT3                      | IL-11             | [174]      |
|                                                            | BZRAP1-AS1  | ↑                | Not reported                     | N/A               | [175]      |
|                                                            | MEG3        | ↑                | miR-650/SLC34A2                  | SLC34A2           | [176]      |
|                                                            | PITPNA-AS1  | ↓                | Not reported                     | MiR-32-5p         | [177]      |
| Promoting apoptosis or inhibiting EMT in lung cancer cells | linc-ITGB1  | ↓                | Not reported                     | Snail             | [178]      |
|                                                            | FENDRR      | ↑                | HuR/MDR1                         | MDR1              | [179]      |
|                                                            | SMASR       | ↑                | TGF-β/Smad                       |                   | [180]      |
|                                                            | PRNCR1      | ↓                | PRNCR1/miR-126-5p/MTDH           | MTDH              | [181]      |
|                                                            | LINC00641   | ↑                | miR-424-5p/PLSCR4                | PLSCR4            | [182]      |
|                                                            | HHIP-AS1    | ↑                | miR-153-3p/PCDHGA9               | PCDHGA9           | [183]      |
|                                                            | XIST        | ↓                | miR-212-3p/CBLL1                 | CBLL1             | [184]      |
|                                                            | DUXAP8      | ↓                | AKT/mTOR                         | miR-498           | [185]      |
|                                                            |             |                  |                                  |                   |            |
| Promotes ferroptosis of lung cancer cells                  | SDCBP2-AS1  | ↓                | SDCBP2-AS1/microRNA-656-3p/CRIM1 | CRIM1             | [186]      |
|                                                            | OGFRP1      | ↑                | miR-299-3p/SLC38A1               | SLC38A1           | [187]      |
|                                                            | NEAT1       | ↑                | Not reported                     | ACSL4             | [188]      |

Footnote: ↑, upregulation; ↓, downregulation; “Not reported” indicates that the information was not available in the original study; EMT, epithelial–mesenchymal transition.

### 5.3. Association and Regulation of LncRNAs with Lung Cancer-Related Drug Resistance

The association of lncRNAs with lung cancer-related drug resistance can be achieved through various mechanisms. Differentially expressed lncRNAs regulate drug resistance and radiosensitivity and can be used to predict patient sensitivity to chemotherapy and targeted therapies [189]. lncRNAs can also regulate drug transport and metabolism in lung cancer cells, thereby altering intracellular drug concentrations and activity and influencing lung cancer cell resistance to chemotherapy agents.

Studies have shown that upregulation of LINC00460 is associated with osimertinib resistance, and EGFR mutations in the primary tumor and plasma of EGFR-mutated lung cancer patients may be associated with poor prognosis in patients treated with osimertinib [190]. Knocking down lncRNA MSTRG.292666.16 reduces osimertinib resistance in H1975R cells [191].

PCAT6 enhances gefitinib resistance in NSCLC through the miR-326/IFNAR2 axis [192]. lncRNA MCF2L-AS1, as an oncogene in NSCLC, drives gefitinib resistance in NSCLC cells by regulating CCDN1 through ELAVL1 [193]. Knockdown of PCAT-1 increases sensitivity to gefitinib in NSCLC by inhibiting phosphorylation of AKT and GSK3 [194]. Additionally, lncRNA LOC554202 promotes acquired gefitinib resistance in non-small cell lung cancer by upregulating miR-31 expression [195]. The mechanism by which lncRNA mediates changes in the efficacy of cisplatin treatment involves regulating several phenotypes, including drug efflux, apoptosis, autophagy, and cancer stemness,

through miRNA sponging effects and gene expression regulation. Additionally, lncRNAs may regulate cisplatin resistance or sensitivity in NSCLC by modulating Wnt and MAPK/Slug signaling pathways, which are closely related to cancer progression [196]. Studies have shown that lncRNA MSTRG51053.2 can function as a ceRNA for miR-432-5p to regulate DDP resistance in NSCLC. MGST1, MGST3, GST- $\omega$ 1, and ABCG2 mRNAs, along with miR-432-5p and miR-665 miRNAs, as well as MSTRG51053.2 and PPAN lncRNAs, have been identified as potential drug targets to reverse DDP resistance in NSCLC [197]. lncRNA CASC2, activated by ELF1, suppresses cisplatin resistance in NSCLC through the miR-18a/IRF-2 signaling pathway [198]. lncRNA NNT-AS1 participates in cisplatin resistance in cancer cells via the miR-1236-3p/ATG7 axis [199]. Knockdown of SNHG1 improves cisplatin resistance in NSCLC by regulating the miR-140-5p/Wnt/ $\beta$ -catenin pathway [200]. LUCAT1 promotes cisplatin resistance in non-small cell lung cancer (NSCLC) cells by regulating the miR-514a-3p/ULK1 axis [201]. LINC01559 is a novel ceRNA that enhances IGF2BP3 expression through sponging miR-320a, thereby driving osimertinib resistance in lung adenocarcinoma [202]. Silencing long intergenic non-coding RNA 00707 enhances sensitivity to cisplatin-resistant NSCLC cells by sponging miR-145 [203]. CASC2c is expressed at low levels in NSCLC tissues and cells; it suppresses NSCLC cell proliferation and migration by inhibiting p-ERK1/2 and  $\beta$ -catenin expression, thereby reversing cisplatin resistance in NSCLC cells [204].

LNA GapmeR antisense oligonucleotides (ASOs) targeting HIF1A-As2 significantly improve sensitivity to 10058-F4 (a MYC-specific inhibitor) and cisplatin treatment in PDX and KRAS (LSLG12D)-driven lung tumors [205]. Nedaplatin reduces multidrug resistance in NSCLC by downregulating the lncRNA MVIH, which is upregulated in resistant NSCLC cells. Nedaplatin can reverse the EMT process by reducing MVIH expression, thereby overcoming cisplatin resistance in NSCLC cells [206].

lncRNAs can also regulate the radiosensitivity of lung cancer cells. By modulating radiation therapy-related signaling pathways and regulatory factors, they influence the response of lung cancer cells to radiotherapy. Certain lncRNAs can regulate the DNA damage repair capacity of lung cancer cells, thereby affecting their radiation sensitivity. For example, the knockdown of H19 suppresses the response of lung cancer cells to infrared-induced DNA damage but promotes its repair. H19 may interact with miR-675, exacerbating infrared-induced methylation damage. H19 acts as a regulator of the DNA damage response in lung cancer cells [207].

lncRNA GLIDR regulates cisplatin resistance through the miR-342-5p/PPARGC1A axis [208]. CRNDE induces resistance to EGFR-TKI by downregulating eIF4A-MUC1 signaling [209]. LSINCT5 is highly expressed in erlotinib-resistant cells, and its interference can inhibit Akt expression and activity, thereby enhancing cellular sensitivity to erlotinib [210]. lncRNA MBNL1-AS1 suppresses NSCLC stem cell resistance by upregulating miR-301b-3p, which targets TGFBR2 [211]. lncRNA TUG1 enhances chemosensitivity in cancer cells by attenuating miR-221-dependent PTEN suppression. lncRNA FENDRR, which is downregulated in NSCLC, is a potential tumor suppressor gene that inhibits NSCLC cell growth and chemoresistance [212] (Table 3).

**Table 3.** Summarizes the research progress on the mechanisms, regulation, and targets of lncRNAs affecting drug resistance in lung cancer.

| Anti-Lung Cancer Drugs | lncRNA Name     | Dysregulation-On | Effect on Resistance | Potential Targets | Reference |
|------------------------|-----------------|------------------|----------------------|-------------------|-----------|
| Osimertinib            | LINC00460       | ↑                | promoting inhibition | N/A<br>c-Kit      | [190]     |
|                        | MSTRG.292666.16 | ↓                |                      |                   | [191]     |

Table 3. Cont.

| Anti-Lung Cancer Drugs | LncRNA Name  | Dysregulation-On | Effect on Resistance | Potential Targets        | Reference |
|------------------------|--------------|------------------|----------------------|--------------------------|-----------|
| Gefitinib              | PCAT6        | ↑                | promoting            | miR-326/IFNAR2           | [192]     |
|                        | MCF2L-AS1    | ↑                | promoting            | ELAVL1                   | [193]     |
|                        | PCAT-1       | ↓                | inhibition           | AKT, GSK3                | [194]     |
|                        | LOC554202    | ↑                | promoting            | miR-31                   | [195]     |
| Cisplatin              | MSTRG51053.2 | ↑                | promoting            | miR-432-5p               | [197]     |
|                        | CASC2        | ↑                | inhibition           | miR-18a/IRF-2            | [198]     |
|                        | NNT-AS1      | ↓                | inhibition           | miR-1236-3p/ATG7         | [199]     |
|                        | SNHG1        | ↓                | inhibition           | miR-140-5p/Wnt/β-catenin | [200]     |
|                        | LUCAT1       | ↑                | promoting            | miR-514a-3p/ULK1         | [201]     |
|                        | ACTA2-AS1    | ↑                | inhibition           | TSC2                     | [202]     |
|                        | 00707        | ↑                | inhibition           | miR-145                  | [203]     |
|                        | CASC2c       | ↑                | inhibition           | p-ERK1/2, β-catenin      | [204]     |
|                        | HIF1A-As2    | ↑                | promoting            | DHX9                     | [205]     |
|                        | MVIH         | ↓                | inhibition           | E-cadherin               | [206]     |
|                        | LSINCT5      | ↑                | promoting            | Akt                      | [210]     |

Footnote: ↑, upregulation; ↓, downregulation; “Not reported” indicates that the information was not available in the original study.

## 6. Conclusions and Future Prospects

lncRNAs play a crucial role in the future of tumor diagnosis and treatment. In recent years, lncRNAs have provided new research perspectives and diagnostic and therapeutic approaches for complex tumor biology. This review summarizes the latest findings on the role of lncRNAs in lung cancer pathogenesis, provides comprehensive data on novel lncRNAs related to lung cancer diagnosis, biomarkers, and the occurrence and metastasis of lung cancer, and elaborates on the potential applications of lncRNAs in lung cancer treatment. Additionally, we discuss a series of studies on lncRNA-mediated resistance in lung cancer, summarizing their roles in resistance to conventional anticancer chemotherapeutic agents, including platinum-based drugs, osimertinib, and gefitinib.

lncRNAs play an essential role in regulating multiple molecular pathways. Investigating the mechanisms of lncRNAs and their impact on genes and proteins not only enhances our understanding of disease mechanisms but also explores the potential of lncRNAs as diagnostic biomarkers and therapeutic targets in lung cancer progression. Numerous studies suggest that lncRNA expression levels can serve as biomarkers for early lung cancer diagnosis and for predicting prognosis. Targeting lncRNAs could also be a therapeutic strategy for lung cancer. The role of lncRNAs in mediating resistance in lung cancer is also a significant research direction. lncRNAs can influence drug sensitivity by regulating metabolic pathways and the cell cycle in lung cancer cells. Therefore, studying lncRNA-mediated resistance mechanisms in lung cancer will aid in the development of new treatment strategies.

With advancements in technology, techniques such as gene knockouts, insertions, and single mutations have expanded the biological functions of lncRNAs, and research on lncRNAs in lung cancer has become more in-depth. However, further research is needed to explore the mechanisms of lncRNAs in clinical applications, investigate their potential for treatment and prognosis, and examine the mechanisms of lncRNA-mediated drug resistance. This will provide more insights and methods for disease treatment and prevention.

lncRNAs play critical roles in lung cancer pathogenesis, diagnosis, and drug resistance. However, translation into clinical practice remains limited by assay heterogeneity, lack of

prospective validation, and inconsistent findings across studies. Based on the evidence reviewed, we propose that future research should prioritize: (i) standardization of detection protocols (e.g., qRT-PCR normalization, sample types); (ii) multi-center prospective cohorts with predefined cutoffs; (iii) integration of lncRNA panels with current biomarkers (CEA, CYFRA21-1, ctDNA); (iv) functional validation in PDX/organoid models; (v) clear criteria for therapeutic targeting (conserved structure, druggable domains, ASO/CRISPRi feasibility); and (vi) transparent reporting of raw data and clinical confounders to resolve contradictory results. Addressing these issues will help move lncRNAs from discovery to clinical application in lung cancer.

**Realistic timelines and barriers for clinical implementation:** Even for the most promising lncRNAs (e.g., SNHG15, DLX6-AS1), clinical adoption remains 5–10 years away. Major barriers include: (i) lack of standardized pre-analytical protocols—exosome isolation methods vary widely, affecting reproducibility; (ii) normalization issues—no consensus on reference genes (U6, GAPDH, or spike-in controls); (iii) regulatory requirements—FDA/EMA would require locked-down assays with lot-to-lot consistency, which most research-grade qRT-PCR kits do not provide; (iv) cost-effectiveness—adding a lncRNA panel must improve diagnostic accuracy sufficiently to justify reimbursement. A pragmatic pathway would be to first integrate one or two tier-1 lncRNAs into existing multiplex panels (e.g., alongside CEA and CYFRA21-1) in retrospective biobank studies, then move to prospective observational studies, and finally to randomized trials if clinical utility is demonstrated.

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