

THE CONTRIBUTORY ROLE OF LONG NON-CODING RNAs TINCR, LUCAT1, AND CASC2 IN HEAD AND NECK SQUAMOUS CELL CARCINOMA

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ABSTRACT

INTRODUCTION

Head and neck squamous cell carcinoma (HNSCC) represent a heterogeneous group of malignancies arising from the mucosal epithelium of the oral cavity, pharynx, and larynx, with high morbidity and mortality. Despite advances in surgery, radiotherapy, and chemotherapy, patient outcomes remain poor due to late diagnosis, therapeutic resistance, and high rates of recurrence.

MATERIALS & METHODS

Extensive literature survey was conducted which highlights the potential role of lncRNAs in the pathogenesis of HNSCC through their regulation of gene expression at different levels.

OBSERVATIONS

The aberrant expression of CASC2, LUCAT1, and TINCR is associated with altered cellular proliferation, invasion, and metastatic potential. These long non-coding RNAs contribute to cell-cycle dysregulation by modulating key oncogenic signalling pathways, including Wnt/ β -catenin, JAK2/STAT3, and MAPK/ERK pathway.

Their dysregulation often leads to altered tumour growth and metastatic behaviour.

CONCLUSION

Dysregulated expression of CASC2, LUCAT1, and TINCR ultimately impacts patient prognosis and survival outcomes, highlighting their relevance in the pathogenesis and progression of HNSCC.

KEYWORDS

CASC2, HNSCC, lncRNA, LUCAT1, TINCR

INTRODUCTION

Squamous cell carcinomas make up the majority of head and neck tumours, with head and neck squamous cell carcinoma (HNSCC) as the most frequent cancer globally. Established risk factors include tobacco use, alcohol consumption, and betel nut chewing, while human papillomavirus (HPV) infection has emerged as a significant etiological contributor (1). The prognosis for patients with HNSCC is still poor, with a 25–60% 5-year survival rate, despite significant advancements in clinical practice (2). The

majority of HNSCC patients are detected at advanced stages due to the lack of predictive biomarkers, which increases the likelihood of distant metastases, recurrence, and poor prognosis (3,4). Therefore, there is an urgent need to identify potential biomarkers for early prognostic evaluation and to explore novel therapeutic strategies for patients with HNSCC.

Transcripts longer than 200 nucleotides are known as long non-coding RNAs (lncRNAs), and they are essential to the molecular pathophysiology of head and neck cancer. They regulate tumorigenic processes either by promoting the degradation of particular messenger RNAs (mRNAs) or suppressing their translation, thereby impacting key pathways linked to cell division, apoptosis, and metastasis (5,6). Additionally, an increasing amount of data suggests that many lncRNAs have distinct expression patterns in HNSCC, with a number of these RNAs being linked to the modulation of tumour initiation and development (7). In order to study their possible roles in tumour biology, the current study focuses on the abnormal expression and dysregulation of the lncRNAs CASC2, LUCAT1, and TINCR in HNSCC. Given their involvement in key cellular processes such as cell proliferation, apoptosis, invasion, and metastasis, these lncRNAs may serve as promising molecular biomarkers in disease diagnosis, prognosis and act as potential therapeutic targets.

2. Long Non-Coding RNAs (lncRNAs) in HNSCC

2.1. Tissue differentiation-induced non-coding RNA (TINCR)

It is located on human chromosome 19p13.3 and, sometimes referred to as placenta-specific protein 2 (PLAC2). It has been demonstrated that TINCR overexpression in oral squamous cell carcinoma (OSCC) stimulates the Wnt/ β -catenin

signalling pathway, which in turn promotes cell proliferation and metastasis (8). Once this pathway is triggered, the β -catenin destruction complex is disrupted, leading to stabilization and nuclear translocation of β -catenin. Once it reaches the nucleus, β -catenin forms a complex with T-cell factor-4 (TCF-4) to drive transcription of downstream target genes such as Cyclin D1, Matrix Metalloproteinase -9 (MMP-9), and Matrix Metalloproteinase -7 (MMP-7) (8–10). This process contributes to poor patient outcomes by enhancing OSCC cell proliferation and metastasis through TINCR-mediated stimulation of Wnt/ β -catenin signalling (11). Furthermore, the absence or downregulation of TINCR was linked to lymph node metastasis, and pathological differentiation. The JAK2/STAT3 pathway was activated by TINCR silencing, which is in line with previous findings and further suggests that TINCR may function as a tumour suppressor in this particular cancer type (12).

Similar oncogenic effects of TINCR have been observed in esophageal squamous cell carcinoma (ESCC) (13). High TINCR expression promotes cell-cycle dysregulation, proliferation, invasion, migration, and resistance to apoptosis. Mechanistically, TINCR regulates the expression of cancer-associated molecules including claudin-7 (CLDN7) and Annexin-1 (ANXA1), both of which show a positive correlation with TINCR expression in ESCC tissues. While ANXA1 binds P65 and suppresses NF- κ B activation, which promotes tumour growth and shields cells from apoptosis (14), CLDN7 maintains the integrity of epithelial tight junctions (15). It's interesting to note that TINCR downregulation in OSCC is connected to JAK2/STAT3 signalling activation, indicating that TINCR may function as a tumour suppressor in some situations by promoting cell differentiation through this pathway's control (12).

TINCR expression was discovered to be much lower in laryngeal squamous cell carcinoma (LSCC) than in nearby normal tissues. According to functional investigations, LSCC cell invasion and proliferation were markedly suppressed by TINCR overexpression. In LSCC tissues and cell lines, miR-210 was increased at the molecular level, negating the benefits of TINCR. As a result, scientists hypothesized that TINCR acts as a miR-210 sponge, stopping the growth of LSCC tumours (16).

In contrast, Nasopharyngeal carcinoma (NPC) showed significant TINCR upregulation, strongly associated with poor survival and chemoresistance. Silencing TINCR not only impaired NPC growth but also prevented cisplatin resistance (17). According to mechanistic research, TINCR maintains acetyl-CoA synthesis and lipid biosynthesis by protecting ATP citrate lyase (ACLY) from ubiquitin-mediated destruction. The PAD1–MAPK–MMP2/9 axis facilitated this metabolic reprogramming, which increased drug resistance, metastasis, and NPC proliferation (18).

When combined, these results demonstrate the role of TINCR in head and neck malignancies. It appears to have tumour-suppressive qualities in LSCC and, in some cases, in OSCC, but it acts as an oncogene in OSCC, ESCC, and NPC. This duality highlights the intricacy of lncRNA-mediated control in cancer and raises the possibility that TINCR could be used as a therapeutic target as well as a predictive biomarker in head and neck cancers (12).

2.2. Lung cancer-associated transcript 1 (LUCAT1)

LUCAT1, sometimes referred to as smoke-and-cancer-associated lncRNA-1 (SCAL1), is found on

chromosome 5q14.3. It was discovered to be raised in lung cancer cells after initially being discovered to be upregulated in healthy bronchial epithelial cells exposed to cigarette smoke (19). Since then, LUCAT1 has been linked to the development of multiple cancers in humans. Yoon et al. showed that LUCAT1 is markedly increased in patient tissues and cell lines of ESCC, and its higher expression is associated with poor prognosis and a shorter overall survival (20).

Similar oncogenic activity has been reported in OSCC with the findings of Kong et. al indicating towards increased LUCAT1 expression in OSCC cell lines and tissues, while its downregulation significantly reduced migration, proliferation, and invasion (21). Downregulation of LUCAT1 also resulted in reduced expression of proliferating cell nuclear antigen (PCNA), a prognostic, tumour staging, and differentiation marker. Research findings show that PCNA has a strong positive correlation with LUCAT1 expression (22–25). These results highlight its part in the carcinogenesis of OSCC. LUCAT1's subcellular location is directly related to its oncogenic activities in the cytoplasm, it operates as a competing endogenous RNA (ceRNA) to modify mRNA stability and metabolism, while in the nucleus, it controls DNA methylation and interacts with nuclear proteins for gene regulation. Furthermore, LUCAT1 participates in several signalling pathways, such as p53, Wnt/ β -catenin, and AKT/GSK-3 β , highlighting its diverse function in cancer biology (19).

Evidence also supports a role for LUCAT1 in TSCC. The research findings of K Zhang et. al. showed that tongue squamous cell carcinoma (TSCC) tissues and cell lines exhibit downregulated miR-375, with lower levels associated with poor overall survival. Crucially, LUCAT1 levels and miR-375 expression were

negatively connected, and LUCAT1 knockdown inhibited migration, proliferation, and cell-cycle progression in part by reviving miR-375 activity (26). Moreover, it has been demonstrated that reduced LUCAT1 expression inhibits the MAPK/ERK pathway in OSCC, which lowers cell proliferation (27). This altogether suggests that LUCAT1 plays a role in carcinogenesis by influencing key oncogenic signalling cascades in addition to epigenetic and post-transcriptional control.

Evidence suggests LUCAT1 as a crucial oncogenic lncRNA in a variety of head and neck malignancies. Its potential as a biomarker and therapeutic target is highlighted by its steady overexpression, capacity to block tumour-suppressive miRNAs, control of DNA methylation, and involvement in key signalling pathways (19,28). Even though the data is strong, functional validation in larger population groups is necessary to validate its therapeutic applicability in precision oncology.

2.3.Cancer susceptibility candidate 2 (CASC2)

CASC2, a tumour-suppressive lncRNA, found on chromosome 10q26 has been linked to several cancers. Its downregulation has been connected to chemoresistance and aggressive phenotypes in a number of cancers, including cervical cancer, glioma, and hepatocellular carcinoma (29). In OSCC, reduced CASC2 expression promotes tumour progression, partly by acting as a ceRNA for oncogenic microRNAs such as miR-21 (30). Another CASC2 target that has been discovered in more recent research is miR-31-5p, which has been demonstrated to increase the tumorigenicity of OSCC (31).

Mechanistically, CASC2 exhibits tumour suppressive effects through diverse pathways. Sponging oncogenic miRNAs, such as miR-21, miR-18a, and miR-181a, aids in restoring the

equilibrium of gene regulation that sustains healthy cellular homeostasis. Furthermore, CASC2 has been shown to suppress Wnt/ β -catenin and MAPK signalling, both of which are central to cancer cell survival, growth, and metastasis. Despite these insights, the precise mechanisms underlying CASC2 downregulation remain poorly understood (32).

It was demonstrated that CASC2 downregulation in NPC increased proliferation and inhibited apoptosis, while its overexpression had an opposing effect. Experimental evidence confirmed that CASC2 directly binds to miR-18a-5p, reducing its expression and restoring apoptosis. On the contrary, increased levels of miR-18a-5p inhibited CASC2-mediated tumour inhibition, partly through the downregulation of the DNA repair protein RBBP8. These results were further supported by animal studies, which revealed that CASC2 downregulation decreased RBBP8 levels, promoted tumour development, and enhanced miR-18a-5p expression (33).

CASC2 also influences other signalling axes in different cancer types. For instance, it suppresses EMT and metastasis in lung cancer by targeting SOX4 (34), and acts as a tumour suppressor in gliomas by blocking Wnt/ β -catenin signalling, thereby serving as a prognostic marker (35). CASC2 has been found to be negatively correlated with cyclin-dependent kinase 1 (CDK1) in OSCC. Since CDK1 is a key regulator of apoptosis and cell cycle progression and is frequently elevated in malignancies like colorectal carcinoma, CASC2 may partially limit tumour growth by regulating CDK1 (36,37).

All things considered, CASC2 is a crucial tumour suppressor lncRNA that has a wide range of effects on signalling pathways, microRNA

networks, and cell cycle regulators (38). Its potential as a therapeutic target and biomarker is highlighted by its link with a poor prognosis and its consistent downregulation in a variety of malignancies. To completely characterize its regulatory networks and convert these discoveries into practical uses, however, more mechanistic research is necessary (39).

CONCLUSION

The growing body of research outlined in this review highlights the crucial role that lncRNAs TINCR, LUCAT1, and CASC2 play in the development of HNSCC. These lncRNAs mainly affect significant oncogenic and tumour-suppressive signalling pathways, which in turn affect key cancer hallmarks such as cell proliferation, invasion, metastasis, apoptosis, chemoresistance, and patient survival.

TINCR exhibits dual roles in head and neck malignancies. It primarily acts as an oncogene in OSCC and ESCC, by activating Wnt/ β -catenin signalling, thereby promoting proliferation, metastasis, and resistance to apoptosis. Conversely, in LSCC, it exerts tumour-suppressive effects by inhibiting invasion and proliferation through miR-210 sponging and modulation of JAK2/STAT3 signalling. Furthermore, TINCR overexpression in NPC highlights its function in cancer metabolism and therapy resistance by driving aggressive tumour behaviour and chemoresistance through metabolic reprogramming mediated by the PAD1–MAPK–MMP2/9 axis. This functional heterogeneity highlights the tissue-specific regulation of TINCR in head and neck cancers. LUCAT1 consistently acts as an oncogenic lncRNA across ESCC, OSCC, and TSCC, with overexpression correlating with poor prognosis, increased proliferation, migration, invasion, and cell-cycle dysregulation. Mechanistically, LUCAT1 acts as a ceRNA for tumour-suppressive

miRNAs, regulates DNA methylation, and modifies key pathways involved in tumour formation including p53, Wnt/ β -catenin, AKT/GSK-3 β , and MAPK/ERK. In contrast, CASC2 is consistently downregulated in OSCC and NPC and largely acts as a tumour-suppressive lncRNA. Loss of CASC2 promotes tumour growth, invasion, and chemoresistance through dysregulation of oncogenic miRNAs, including miR-21, miR-31-5p, and miR-18a-5p, along with impaired apoptosis and DNA repair. Collectively, these findings indicate that lncRNAs play a key role in regulation of oncogenic networks in head and neck malignancies. Their dysregulation contributes to tumour heterogeneity, disease progression and therapy resistance, supporting their potential as future biomarkers and therapeutic targets.

FUTURE PROSPECTS

The future of cancer treatment is increasingly being shaped by biomarker-driven strategies. lncRNAs CASC2, LUCAT1, and TINCR have emerged as potential molecular biomarkers in the management of HNSCC. Their consistent dysregulation across HNSCC and its subtypes indicates considerable diagnostic and prognostic potential. However, there is relatively limited understanding about their role in tumorigenesis and clinical applicability. Additional in-vitro and in-vivo investigations are necessary to elucidate the exact roles of these lncRNAs in the pathophysiology of HNSCC. A deeper understanding of the key signalling pathways and miRNA networks associated with these lncRNAs is required. Furthermore, large-scale clinical studies are essential to validate their clinical relevance and potential utility.

AUTHORS DECLARATIONS

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