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The multifaceted role of long non-coding RNA DLEU1 in cancer progression: implications for diagnosis and therapy

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Abstract:
INTRODUCTION AND PURPOSE: Long non-coding RNAs are key regulators of gene expression and tumorigenesis. Deleted in leukemia 1 (DLEU1) is significant in cancer development. This article reviews DLEU1's role across cancers, assessing its diagnostic and therapeutic potential.

METHODS: A review of scientific articles from PubMed and Google Scholar was conducted.

DESCRIPTION OF THE STATE OF KNOWLEDGE: DLEU1 drives tumor growth in glioblastoma, esophageal, colorectal, and oral cancers by interacting with key miRNAs and genes, promoting malignancy. In breast and ovarian cancers, DLEU1 modulates migration and invasion via specific pathways. In cervical cancer, DLEU1 is linked to immune modulation and serves as a prognostic marker.

SUMMARY: DLEU1 is a vital regulator in various cancers, offering potential as a diagnostic and therapeutic target.
Key words: long non-coding RNA, glioblastoma, cancer

INTRODUCTION AND PURPOSE

Long non-coding RNAs (lncRNAs) are a diverse class of RNA molecules that, while not encoding proteins, play pivotal roles in regulating gene expression at various levels, including chromatin modification, transcription, and post-transcriptional processing [1, 2]. Unlike protein-coding RNAs, lncRNAs are typically longer than 200 nucleotides and are involved in a wide range of cellular processes [3].
Their functions are highly context-dependent, with many lncRNAs implicated in regulating key biological pathways, such as those related to development, differentiation, and disease [4].

One particularly intriguing aspect of lncRNAs is their involvement in tumorigenesis—the process by which normal cells become cancerous and divide uncontrollably[5]. Depending on the cellular context, lncRNAs can function as oncogenes or tumor suppressors, influencing the expression of genes that control cell proliferation, apoptosis, and metastasis [6, 7]. Dysregulation of lncRNAs has been strongly linked to the progression of various cancers, making them a critical area of research in cancer biology [8].

Deleted in Lymphocytic Leukemia 1 (DLEU1) is a noteworthy lncRNA that has been implicated in several cancers. Initially identified in a chromosomal region frequently deleted in chronic lymphocytic leukemia, DLEU1 has also been associated with cancers such as breast, gastric, and colorectal cancer. Research suggests that DLEU1 may contribute to tumorigenesis by influencing gene expression and cellular pathways that promote cancer cell proliferation and survival [6, 7]. However, the exact mechanisms by which DLEU1 drives cancer development are still under investigation. Understanding the roles of DLEU1 and other lncRNAs in cancer could lead to the identification of new diagnostic markers and therapeutic targets, offering the potential for more effective cancer treatments in the future [1, 8].

The purpose of this article was to assess the current state of knowledge about role that lncRNAs, especially DLEU1 plays in cancer development and progression and how it may affect diagnostic and therapeutic procedures.

METHODS

An extensive examination of scholarly articles published in scientific journals was carried out through the utilization of online research platforms, specifically the websites PubMed and Google Scholar.

DESCRIPTION OF THE STATE OF KNOWLEDGE

The Role of DLEU1 in Glioblastoma Multiforme Formation

Glioblastoma multiforme (GBM) is the most aggressive and common form of primary brain tumor in adults. Arising from astrocytes, a type of glial cell in the brain, GBM is characterized by its rapid growth, high invasiveness, and poor prognosis. The median survival time for patients diagnosed with GBM is only about 12 to 15 months, even with aggressive treatments including surgery, radiotherapy, and chemotherapy with temozolomide [9,10]. One of the most challenging aspects of GBM is its high recurrence rate after initial treatment, with tumors often returning more resistant to therapies, leading to a poor overall survival outcome [11].

Long non-coding RNAs (lncRNAs) have recently gained significant attention for their roles in GBM, where they can influence gene expression and tumor progression. DLEU1 is one such lncRNA that has been implicated in the pathogenesis of GBM. DLEU1 is known to interact with various microRNAs (miRNAs) and differentially expressed messenger RNAs (DEmRNAs), contributing to the malignant behavior of GBM cells [12].

One of the critical interactions involving DLEU1 in GBM is its positive correlation with the expression of TNF receptor-associated factor 4 (TRAF4). TRAF4 is a protein that plays a crucial role in promoting cell proliferation and survival in GBM.

Studies have shown that higher levels of DLEU1 are associated with increased TRAF4 expression, which in turn drives the proliferation of GBM cells, contributing to tumor growth and resistance to apoptosis [13]. This interaction highlights DLEU1 as a significant factor in the aggressiveness of GBM.

DLEU1 also plays a role in the activation of the KPNA3 gene, which is associated with increased proliferation and migration of neoplastic cells. KPNA3 encodes a protein involved in the nuclear import of transcription factors and other proteins that regulate cell cycle progression and cell proliferation. In GBM, the upregulation of KPNA3, facilitated by DLEU1, enhances the proliferative capacity of tumor cells and supports their invasive behavior, further contributing to the malignancy of the tumor [14].

DLEU1 exerts its effects in GBM partly by interacting with specific microRNAs, acting as a competitive endogenous RNA (ceRNA) that sponges miRNAs, thereby preventing them from binding to their target messenger RNAs. For instance, DLEU1 competitively binds to microRNA-421, a miRNA that typically inhibits the expression of myocyte enhancer factor 2D (MEF2D), a transcription factor involved in GBM progression. By sequestering microRNA-421, DLEU1 prevents the suppression of MEF2D, leading to increased MEF2D expression and promoting the proliferation and survival of GBM cells [15].

In another critical interaction, DLEU1 abolishes the tumor suppressor activity of microRNA-4429 on the specificity protein 1 (SP1) gene. SP1 is a transcription factor that, when upregulated, increases the proliferative and anti-apoptotic potential of GBM cells. DLEU1 sequesters microRNA-4429, preventing it from inhibiting SP1 expression. Consequently, this leads to the overexpression of SP1, which not only promotes tumor growth but also activates the transcription of DLEU1 itself, creating a positive feedback loop that further enhances GBM malignancy [16]. This feedback loop is critical for the sustained growth and survival of GBM cells, underscoring the role of DLEU1 in the pathogenesis of GBM.

Given its extensive involvement in promoting GBM cell proliferation, survival, and resistance to apoptosis, DLEU1 is emerging as a potential biomarker for GBM malignancy. Its expression levels could be used to assess tumor aggressiveness and predict patient prognosis. Moreover, DLEU1 represents a promising target for gene therapy in GBM. Strategies that could inhibit DLEU1 expression or disrupt its interactions with key miRNAs and DEmRNAs may offer new therapeutic avenues to combat this deadly disease. By targeting DLEU1, it may be possible to reduce tumor growth, enhance the effectiveness of existing treatments, and ultimately improve the survival outcomes for patients with GBM [17,18].

DLEU1 in digestive tract neoplasms

DLEU1 and gastric cancer

DLEU1 is one of the most overexpressed long non-coding RNAs in esophageal squamous cell carcinoma (ESCC) tissues, with its upregulated expression closely associated with worse patient prognosis. Functional assays have demonstrated that DLEU1 promotes tumor growth in ESCC by inhibiting cell apoptosis. Mechanistically, DLEU1 binds and stabilizes DYNLL1 (Dynein Light Chain LC8-Type 1), a protein involved in various cellular processes, by interfering with RNF114-mediated ubiquitination and subsequent proteasomal degradation of DYNLL1.

The stabilization of DYNLL1 by DLEU1 leads to the upregulation of the anti-apoptotic protein BCL2, thereby promoting cell survival and contributing to the malignancy of ESCC [19]. Furthermore, it has been shown that the upregulation of DLEU1 in ESCC is at least partly due to promoter hypomethylation, which facilitates its increased expression. Importantly, targeting DLEU1 in ESCC cells has been found to sensitize these cells to cisplatin-induced apoptosis, suggesting that DLEU1 could be a potential therapeutic target in ESCC treatment [19].

Additionally, DLEU1 is involved in the promotion of ESCC development through its interaction with the High-mobility group AT-hook 1 (HMGA1) protein. HMGA1 has been shown to mediate the miR-671-5p/lncRNA DLEU1 axis, which plays a critical role in ESCC pathogenesis. Specifically, HMGA1 promotes the expression of DLEU1 by suppressing miR-671-5p, a microRNA that typically inhibits DLEU1 expression. This HMGA1-mediated upregulation of DLEU1 contributes to the progression of ESCC, further emphasizing the oncogenic role of DLEU1 in this type of cancer [20].

Colorectal cancer development and DLEU1

DLEU1 plays a significant role in the growth, progression, and invasion of colorectal cancer (CRC). DLEU1 has been identified as one of the most upregulated long non-coding RNAs in CRC tissues, with its overexpression closely linked to poor prognosis in patients. DLEU1 promotes CRC development by modulating several molecular pathways that are crucial for cancer cell survival and dissemination.

In one study, DLEU1 was found to interact with microRNA-320b (miR-320b), which normally acts as a tumor suppressor by targeting phosphoribosyl pyrophosphate synthetase 1 (PRPS1), a protein involved in nucleotide biosynthesis that supports rapid cell proliferation. By sponging miR-320b, DLEU1 prevents the inhibition of PRPS1, leading to increased cell proliferation, migration, and invasion, while simultaneously reducing apoptosis in CRC cells. The knockdown of DLEU1 in CRC cells significantly suppressed tumor growth in vivo, highlighting its potential as a therapeutic target [21].

Another study revealed that DLEU1 facilitates the activation of the KPNA3 gene, which is associated with enhanced proliferation and metastatic potential of CRC cells. DLEU1 accomplishes this by recruiting SMARCA1, a key subunit of the NURF chromatin remodeling complex, to the KPNA3 promoter, leading to its transcriptional activation. Elevated levels of DLEU1 and KPNA3 were observed in CRC tissues, and their expression correlated with poorer patient survival. Inhibition of DLEU1 resulted in reduced CRC cell proliferation, migration, and invasion, effects that could be reversed by overexpressing KPNA3, underscoring the importance of the DLEU1/SMARCA1/KPNA3 axis in CRC pathogenesis [22].

DLEU1 in oral squamous cell carcinoma

DLEU1 plays a significant role in the development and progression of oral squamous cell carcinoma (OSCC). Studies have consistently shown that DLEU1 is notably overexpressed in OSCC tissues and cell lines, and its upregulation is associated with poor prognosis and reduced survival rates in patients with OSCC [23].

One key mechanism by which DLEU1 contributes to OSCC is through the regulation of interferon-stimulated genes (ISGs). DLEU1 upregulates ISGs, including IFIT1, IFI6, and OAS1, via activation of the JAK-STAT signaling pathway.

This upregulation is associated with increased histone modifications, specifically H3K4 trimethylation and H3K27 acetylation, which are crucial for gene activation. These ISGs, in turn, enhance OSCC cell proliferation, migration, and invasion [23].

Another important pathway influenced by DLEU1 in OSCC involves its interaction with microRNAs. DLEU1 has been shown to bind to and sequester miR-126-5p, a microRNA that normally acts as a tumor suppressor by targeting GAB1, a gene involved in cell survival and proliferation. By sponging miR-126-5p, DLEU1 prevents the suppression of GAB1, leading to enhanced OSCC cell proliferation, migration, and invasion. Silencing DLEU1 significantly reduces these oncogenic activities, while overexpression of DLEU1 or GAB1 reverses these effects [24].

Furthermore, DLEU1 also regulates the miR-149-5p/CDK6 axis in OSCC. DLEU1 binds to miR-149-5p, a microRNA that targets CDK6, a cyclin-dependent kinase involved in cell cycle regulation. By inhibiting miR-149-5p, DLEU1 prevents the downregulation of CDK6, thereby promoting cell proliferation and contributing to OSCC tumorigenesis. Knockdown of DLEU1 leads to increased apoptosis and reduced proliferation of OSCC cells, effects that can be reversed by inhibiting miR-149-5p [25].

Additionally, DLEU1 influences the expression of several cancer-related genes, including HAS3, CD44, and TP63, which are involved in hyaluronic acid-CD44 signaling, a pathway critical for cell proliferation and migration. DLEU1 knockdown significantly reduces the expression of these genes, leading to decreased OSCC cell proliferation, migration, and invasion, as well as reduced tumor growth in xenograft models [26].

ROLE OF DLEU1 IN BREAST, OVARIAN AND CERVICAL CANCER

DLEU1 in breast cancer

The long non-coding RNA Deleted in Lymphocytic Leukemia 1 has emerged as a significant contributor to the development and progression of breast cancer, particularly through its involvement in key cellular processes such as migration, invasion, and epithelial-mesenchymal transition (EMT). Studies have consistently shown that DLEU1 is highly expressed in breast cancer tissues compared to normal adjacent tissues, suggesting its potential as a diagnostic biomarker [27].

DLEU1 promotes breast cancer malignancy by modulating various molecular pathways. DLEU1 regulates the migration and invasion of breast cancer cells through the DLEU1/microRNA-300/RAB22A axis. Specifically, DLEU1 overexpression enhances the migratory and invasive capacities of breast cancer cells by downregulating microRNA-300, which in turn leads to the upregulation of RAB22A, a protein involved in EMT. The study further demonstrated that knocking down DLEU1 results in the opposite effect, reducing the invasiveness of breast cancer cells [28].

In another study, DLEU1 was found to act as a coactivator for Hypoxia-Inducible Factor 1-alpha (HIF-1α), a key transcription factor that responds to low oxygen conditions within tumors. DLEU1 interacts with HIF-1α to enhance the transcription of CKAP2, a gene implicated in cell division and tumor growth. The upregulation of CKAP2 by DLEU1 activation promotes the malignancy of breast cancer by triggering the ERK and STAT3 signaling pathways, which are known to facilitate cancer cell proliferation and survival. The knockdown of DLEU1 in vivo significantly inhibited the growth and metastasis of breast cancer cells [29].

Bioinformatics analysis has revealed its involvement in estrogen receptor (ER)-related pathways in breast cancer. DLEU1 was found to be highly expressed in ER-positive breast cancer cells, where it is believed to co-regulate the expression of ESR1 (the gene encoding ER) alongside miR-19a, another molecule located on the same chromosome as DLEU1. This co-regulation may influence the expression profiles of ER-related genes, thereby affecting the behavior of breast cancer cells [30].

DLEU1 in premature ovarian failure and ovarian cancer

DLEU1 has been linked to both premature ovarian failure (POF) and ovarian cancer, performing unique yet crucial functions in these diseases. In the setting of POF, there is notable upregulation of DLEU1, which interacts with miR-146b-5p to regulate granulosa cell apoptosis, a pivotal aspect of ovarian dysfunction. More precisely, DLEU1 functions as a miR-146b-5p sponge, decreasing its levels and resulting in higher apoptosis of granulosa cells. This process plays a role in the development of POF by disturbing the equilibrium of cell survival in the ovaries, crucial for the upkeep of normal ovarian function [31].

In ovarian cancer, DLEU1 is also involved in promoting tumor growth and metastasis through affecting multiple molecular pathways. A study by Xu at al. showed that DLEU1 increases the expression of the transcription factor TFAP2A by acting as a sponge for miR-429, a microRNA with tumor-suppressive properties. This communication helps ovarian cancer cells to multiply, move, and spread, showing that DLEU1 promotes cancer development via the miR-429/TFAP2A pathway. Alternatively, inhibition of DLEU1 results in a notable decrease in these harmful actions, indicating its promise as a treatment target for ovarian cancer [32].

A different study on epithelial ovarian carcinoma (EOC) Wang et al. revealed that DLEU1 has a connection with miR-490-3p, a microRNA that possesses tumor-suppressing properties. Through the suppression of miR-490-3p, DLEU1 enhances the levels of CDK1, cyclin D1, and other proteins that promote cell cycle advancement and prevent apoptosis. DLEU1 overexpression in ovarian cancer cells boosts their proliferation, migration, and invasiveness, and in vivo experiments indicate that DLEU1 facilitates tumor growth in xenograft models. These results emphasize the importance of DLEU1 in advancing ovarian cancer by controlling critical molecular targets related to cell cycle and survival pathways [33].

Cervical cancer

One of the significant findings is the inclusion of DLEU1 in immune-related prognostic models. For instance, in a study that constructed a six-lncRNA immune prognostic signature for cervical cancer, DLEU1 was identified as a key component. This signature was effective in predicting patient outcomes, with higher DLEU1 expression correlating with poorer prognosis. The study linked this immune prognostic signature with pathways such as Wnt and TGF-beta signaling, which are crucial for immune responses and tumorigenesis in CC [34].

Further exploration into DLEU1's role in immune modulation was conducted through the development of a novel signature based on immune-related lncRNAs, where DLEU1 was again identified as a critical player. This study showed that DLEU1 expression was negatively correlated with the infiltration of immune cells such as CD8+ T cells and macrophages M1/M2, which are vital for anti-tumor immunity.

The high-risk group, characterized by elevated DLEU1 levels, also exhibited higher expression of immune checkpoint proteins, suggesting that these patients might benefit from checkpoint inhibitor therapies like PD-1/PD-L1 inhibitors [35].

The role of DLEU1 extends to necroptosis, a form of programmed cell death that has been implicated in cancer progression and treatment response. A study focusing on necroptosis-related lncRNAs in CC identified DLEU1 as part of a prognostic signature that could predict overall survival (OS) and response to treatments. The signature, which included DLEU1, showed that lower risk scores (indicating lower DLEU1 expression) were associated with better prognosis and enhanced sensitivity to treatments like Rucaparib and Navitoclax [36].

Another dimension of DLEU1's involvement in CC is its association with m6A RNA methylation, an epigenetic modification that regulates gene expression and has been linked to cancer. A study analyzing m6A-modification-associated lncRNAs identified DLEU1 as a significant prognostic marker. The study's risk model, which included DLEU1, effectively predicted patient outcomes and indicated that high DLEU1 expression was associated with poorer survival. Moreover, the model suggested that patients with high DLEU1 expression might benefit more from immunotherapy, particularly targeting the PD1 immune checkpoint [37].

DLEU1 has also been identified as a hub node in ceRNA (competing endogenous RNA) regulatory networks. This network analysis revealed that DLEU1 interacts with multiple miRNAs and mRNAs, influencing genes associated with cancer recurrence. These interactions suggest that DLEU1 not only promotes initial tumor growth but may also contribute to the recurrence of CC by regulating critical pathways involved in cell proliferation and survival [38].

The relationship between DLEU1 and human papillomavirus (HPV) infection, a major risk factor for CC, further emphasizes its role in cervical carcinogenesis. Research has shown that DLEU1 is significantly upregulated in HPV-positive CC tissues and that its expression correlates with poorer prognosis in these patients. DLEU1's ability to differentiate between HPV-positive and HPV-negative CC cases suggests its potential as a diagnostic biomarker. Additionally, the interaction between DLEU1 and various miRNAs, as predicted by the RNA Interactome Database, points to complex regulatory mechanisms through which DLEU1 may influence HPV-driven carcinogenesis [39].

The functional role of DLEU1 in promoting cervical cancer proliferation and invasion has been elucidated through its interaction with miR-381 and HOXA13. DLEU1 has been shown to inhibit miR-381, leading to the upregulation of HOXA13, a gene associated with cell proliferation and invasion. This axis of DLEU1/miR-381/HOXA13 is a critical pathway through which DLEU1 exerts its oncogenic effects in CC [40].

SUMMARY

Our aim was to explore the role of long non-coding RNA DLEU1 in the development and progression of various cancers, focusing on its potential as a diagnostic and therapeutic target. lncRNAs, including DLEU1, are crucial in regulating gene expression and are involved in numerous cellular processes. DLEU1, initially associated with chronic lymphocytic leukemia, has been found to play significant roles in other cancers, such as glioblastoma multiforme, gastric cancer, and colorectal cancer.

DLEU1 holds significant potential as both a diagnostic marker and a therapeutic target in various cancers. Its overexpression in several cancer types suggests it could be used to identify aggressive tumors and predict patient outcomes. Therapeutically, targeting DLEU1 to disrupt its interactions with key oncogenic pathways could offer new avenues for cancer treatment, potentially reducing tumor growth and improving the effectiveness of existing therapies.
However, further research is needed to fully understand the mechanisms by which DLEU1 contributes to tumorigenesis and to explore its clinical applications in cancer diagnosis and treatment.

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