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Expression Patterns of LncRNA *UCA1* as a Novel Epigenetic Marker in Iraqi CML Patients

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Abstract

Chronic Myeloid Leukemia (CML) is an advanced myeloproliferative neoplasm with a poor prognosis, having an accelerated phase dominated by the uncontrolled expansion of myeloid tissue. In many organs, Specific immature myeloid blasts result from numerous hematopoiesis abnormalities. Using a sample from newly diagnosed chronic myeloid Leukaemia patients, this study aims to evaluate the value of *UCA1* as an oncogene. Thirty Iraqi individuals with CML were gathered for blood samples; 28 cohorts were used as controls. Total RNA was extracted from the samples, and one-step RT-PCR (RT-PCR) was performed using the BRYT Green® Dye technique to measure the *UCA1* expression level. Tested blood samples from both the control and patient groups for TEGT gene expression. Compared with the control group (1.0 ± 0.0; $p < 0.0001$), the patient group showed a significant decrease in *UCA1* expression (0.2742 ± 0.21). Compared to the control group, this study demonstrates notable downregulation of *UCA1* in the patient group. At the same time, the *UCA1* transcript exhibits gene-regulatory potential at multiple levels, including transcription and post-transcriptional regulation. The *UCA1* reduction observed in the patient group may indicate that this lncRNA is involved in disease mechanisms, which warrants further investigation.

Keywords: long non-coding RNA, *urothelial carcinoma associated 1* (*UCA1*), Chronic myeloid leukaemia, QPCR.

اط التعبير لـ

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الخلاصة

يعَدُّ سرطان الدم النخاعي المزمن ((CMLورمًا تكاثريًا نخاعيًا متقدمًا ذو تشخيص ضعيف، يتميز بمرحلة متسارعة يهيمن عليها انتشار غير مسيطر عليه للأنسجة النخاعية. حيث تنشأ أورام نخاعية غير ناضجة في أعضاء متعددة بالجسم، وذلك نتيجةً لعدّة اعتلالات في الخلايا المكونة للدم. يهدف هذا البحث إلى تقييم جين *UCA1* كأحدى الجينات المُسببة للسرطان وذلك باستخدام عينات مأخوذة من مرضى سرطان الدم النخاعي المزمن. حيث تم جمع ثلاثون عينة دم من مرضى عراقيين تم تشخيصهم حديثا بسرطان الدم النخاعي المزمن؛ وكذلك جمعت 28 عينة دم من أشخاص أصحاء كمجموعة سيطرة. تم عزل RNA الكلي من جميع العينات وأجري أختبار RT–PCR أحادي الخطوة (RT–PCR) باستخدام تقنية صبغة BRYT Green® لقياس مستوى التعبيرالجيني *UCA1*. وتمت معايرة التعبير الجيني*UCA1* من خلال قياس مستوى التعبير الجيني *TEGT* لجميع عينات الدم لكل من مجموعتي السيطرة والمرضى. وعند مقارنة نتائج مجموعة السيطرة، التي تم تعيينها عند 1.0 ± 0.0 (p < 0.0001)، يمكن ملاحظة أن مجموعة المرضى شهدت انخفاضًا معنويا كبيرًا في التعبير الجيني *UCA1* (0.2742 ± 0.21). حيث تُظهر هذه الدراسة انخفاضًا ملحوظًا في مستويات التعبيرالجيني *UCA1* لمجموعة المرضى مقارنة بمجموعة السيطرة. في الوقت نفسه، يمتلك *UCA1* إمكانية تنظيم مراحل عملية التعبيرالجيني المختلفة ومنها مرحلتي الترجمة وما بعد الترجمة. وقد أشار هذا الانخفاض الملحوظ في مستوى التعبير الجيني *UCA1* لمجموعة المرضى إلى دور هذا الرنا الطويل غير المشفر (lncRNA) في آليات تطورالمرض، مما يتطلب اجراء المزيد من البحوث والدراسات.

1.Introduction

Hematopoietic stem cells (HSCs) initiate a carefully managed process called hemopoiesis, which produces various types of blood cells. This process goes through several stages of maturation. There are many types of leukemia, all of which are very dangerous blood cancers caused by the unchecked growth of oligoclonal, differentiated HSCs [1]. Leukemia is caused by a wide range of genetic and epigenetic defects, as well as various risk factors, and the best treatment approach for each case is different. However, in general, high or fatal doses of radiation, long-term exposure to carcinogens like benzene, and work-related accidents or hazards all raise the risk of getting leukemia [2]. Certain genetic and epigenetic issues may arise spontaneously or over time, allowing myeloid progenitor cells to proliferate without halting or differentiating. This can cause blood cancer known as myeloid or myelogenous leukemia [3]. Acute and chronic forms of myeloid leukemia determine the period of illness evolution. As deadly Acute Myeloid Leukemia (AML) progresses rapidly, immature and non-functional blood cells accumulate in the bone marrow. Chronic Myeloid Leukemia (CML), in contrast, slows down growth and results in the accumulation of more developed but still aberrant blood cells [4]. Study showed that, upon diagnosis, around 90% of individuals with CML survive for five years or more, unlike 30% in the case of AML [5]. lncRNAs are non-coding RNAs that have more than 200 nucleotides [6]. lncRNAs have been implicated in various biological processes, including cell growth, oncogenesis, proliferation, apoptosis, and differentiation. They achieve this by altering gene expression at the epigenetic, transcriptional, and post-transcriptional levels [7,8]. Initially detected in bladder cancer, it resurfaced in several malignant tumors. It is fundamental in tumor development, death, invasion, anti-cancer medication resistance, and metabolism. The functional mechanism of *UCA1* in cancer is highly clinically relevant [9]. lncRNAs play a crucial role in various important biological processes, including gene expression regulation, genome editing, and alternative gene splicing [10]. They are also associated with various biological processes such as cancer [11], cell differentiation, organogenesis, and genetic imprinting. There are three groups of lncRNAs based on how they are expressed and what they do in the body: oncogenes, tumor suppressor genes, and duplex lncRNAs.

2.Materials and methods

Bioinformatics method

Online LnRNA target prediction techniques described below were employed in this study to assess the putative LncRNADisease database (http://www.cuilab.cn/lncrnadisease) and the Reactome database (https://reactome.org).

Ethics approval

This study was approved by the Higher Institute of Forensic Sciences Ethics Committee (Date: 20/10/2024**)**

Samples collection

Between March 2024 and December 2024, fifty-eight individuals with CML who were not receiving any treatment were recruited to provide blood samples at the Blood Diseases and Bone Marrow Transplant Center in Medical City. Samples consisted of freshly obtained peripheral blood from various age- and sex-matched healthy individuals and patient participants. Venous blood samples, measuring five milliliters (mL), were collected from both the patient cohort and the healthy control group. All subjects diagnosed with CML were subsequently placed in gel tubes for a 30-minute period at ambient temperature.

The procedure of RNA extraction from blood samples

The sample was processed using TRIzolTM Reagent for RNA extraction, including LncRNA. Then, 0.2 mL of chloroform was added to the water phase, which already had RNA. 0.5 mL of isopropanol was then added to help the RNA form a white, gel-like pellet. The RNA was then washed with 0.5 mL of 70% ethanol. The pellet was then resuspended in 50 µL of nuclease-free water and incubated in a water bath at 55–60°C for 10-15 minutes.

RNA quantitation (Fluorescence method)

A fluorescence method was used to assess the concentration of extracted RNA. 1 µl of RNA was mixed with 200 µl of diluted QuantiFluor Dye, and the RNA concentration was measured in the dark at room temperature for five minutes.

Primer preparation

Primers were supplied by Macrogen company in lyophilized form. The freeze-dried primers were mixed with nuclease-free water to make a stock solution with a final concentration of 100 pmol/l. This basic working solution was created by mixing 10 µl of primer stock solution (kept in the freezer) with 90 µl of nuclease-free water. The combination generates a 10 pmol/µl workable primer solution.

Table 1: Primers used in the PCR.

No.	Primer name		Primer sequence ('5----3')	Reference
1.	*TEGT*	*TEGT*-F	TGCTGGATTTGCATTCCTTACA	[12]
		TEGT-R	ACGGCGCCTGGCATAGA	
2.	*UCA1*	*UCA1*-F	GACCCTACCCGGTCATTTATAG	[12]
		UCA1-R	CTGATGGGCATGGCTTTATTC	

One-step RT-PCR thermal cycling protocol

One-step qPCR enables the measurement of gene expression by reverse-transcribing RNA into cDNA, amplifying it with RT-PCR, and observing the amplified results within the same tube.

Table 2: PCR Component Calculation

PCR Component	Calculation
No. of reaction	60 rxn
No. of primers	4
Safety margin	5 %
No. of PCR Cycles	40
Reaction volume /run	10 µl
Annealing temperature of primers	60,65

Real-time polymerase chain reaction quantitatively

Table 3 illustrates the procedure for preparing the PCR master mix. Table 4 lists the *UCA1* thermal cycling settings and the real-time PCR program.

Table 3: Illustrates the procedure for preparing the PCR master mix

No.	Component	Volume
1.	qPCR Master Mix	5
2.	RT mix	0.25
3.	$MgCl_2$	0.25
4.	Forward primer (10 µM)	0.5
5.	Reverse primer (10 µM)	0.5
6.	RNA	1
7.	Nuclease-free Water	2.5
8.	Total volume	10
Aliquot per single rxn		9µl of Master mix per tube, and add 1µl of Template

Table 4: Lists the *UCA1* thermal cycling settings and the real-time PCR program.

Steps	°C	m: s	Cycle
RT. Enzyme Activation	37	15:00	1
Initial Denaturation	95	05:00	
Denaturation	95	00:20	40
Annealing	60 or 65	00:20Acquiring on Green	
Extension	72	00:20	

Statistical analysis

The data were analyzed using an Independent Samples T-Test, and the Livak or CT method was employed to determine the quantitative RT-PCR ratio (Livak and Schmittgen, 2001). If the p-value was less than 0.05, it indicated that both the patient and control groups altered the expression of the *UCA1* gene.

3.Results and discussion

In silico study

Multiple web servers were used to browse, search, and query LncRNA. The LncRNADisease database is a comprehensive resource for studying this exciting area. This database carefully compiles lncRNA-disease connections based on experimental validation, making it a useful resource for researchers. It also incorporates predictive algorithms to identify new lncRNA-disease correlations. Apart from illness connections, LncRNADisease

also records lncRNA interactions at several levels, covering interactions with proteins, other RNAs, microRNAs (miRNAs), and DNA. This multifaceted approach provides a comprehensive picture of lncRNA function, confirming its indispensable role in understanding the intricate interactions between lncRNAs and human disease. These genes are linked to Reactome events, such as activation of MAPK signaling and the formation of the spliceosomal trisnRNP complex.

Demographic distribution of samples

A retrospective study examined 58 blood samples, classified into control (n=30) and patient (n=28) groups. All samples were categorized into age groups, specifically those under 20 years and those over 60 years. In both the control and patient groups, the highest proportions were observed in the 30-39 age range (33.33%) and the 40-49 age range (23.33%). The 20-29 age group had the lowest proportion at 6.67%. Conversely. The findings presented in Table 5 illustrate distinct age distributions between the control group and the patients.

Table 5: Sample research distribution based on age groupings

Age Group (Years)	No. of Control & Patients	Percentage
Under 20	4	13.33%
20-29	2	6.67%
30-39	10	33.33%
40-49	7	23.33%
50-59	3	10.00%
≥60	4	13.33%
total	30	100%

Gene expression

The molecular experiment assessed the expression levels of the IncRNA *UCA1* using the reference gene TEGT. Quantitative PCR was used to measure the threshold cycle (Ct) values for each sample, as illustrated in Figure 1, while Figure 2 displays their melting curves.

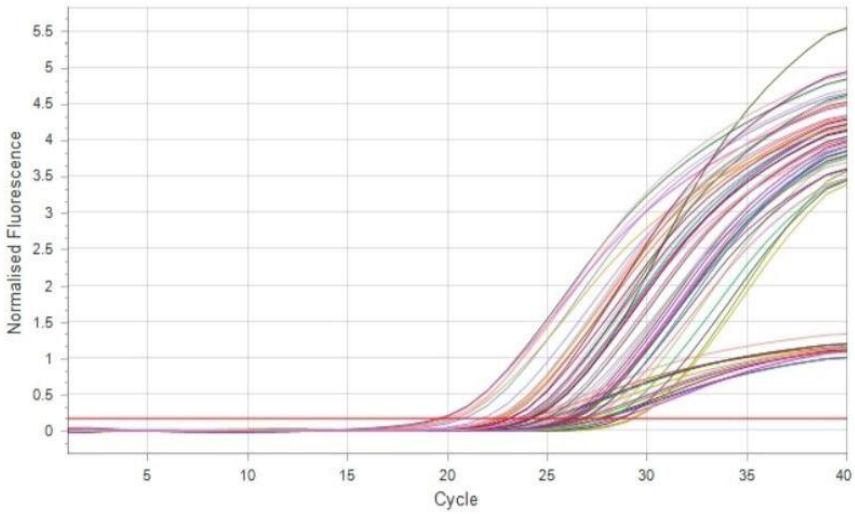

Figure1: Amplification plots for lncRNA *UCA1* and *TEGT* expression obtained by real-time PCR

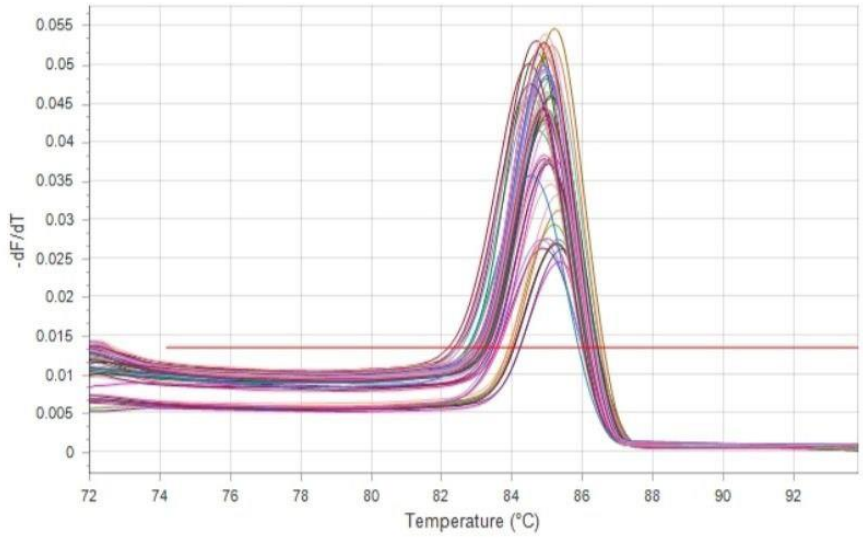

Figure 2: The lncRNA *UCA1* and *TEGT* expression melting curve

We examined *UCA1*'s expression using patient sample fold changes against those of control samples. The patient group had considerably lower *UCA1* expression (0.2742 ± 0.21) than the control group, with a difference of 1.0 ± 0.0 ($p < 0.0001$). Table 6 and Figure 3 illustrate these findings.

Table 6: Fold of lncRNA *UCA1* gene expression

Mean ± SD *UCA1* Fold of Change		**Sig.**	**P-Value**
Control	**Patient**		
1.0 ± 0.0	0.2742 ± 0.21	**	<0.0001

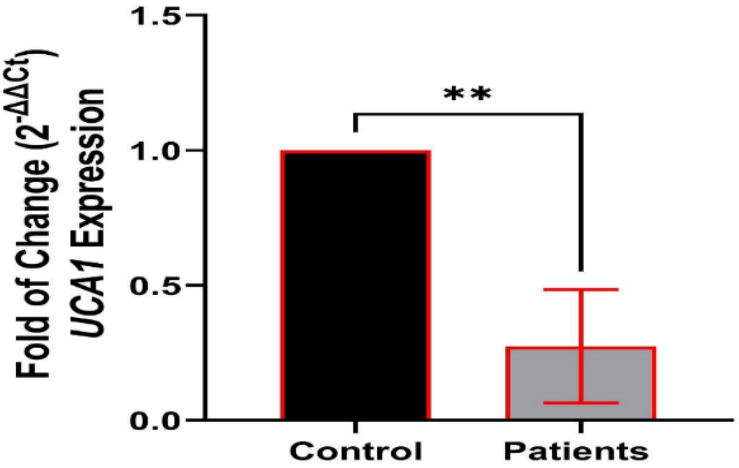

Figure 3: Fold of change lncRNA *UCA1* gene expression

Over the past 15 years, the human long non-coding RNA (lncRNA) transcriptome has experienced rapid growth. This has revealed a complex regulatory layer in cell biology, particularly in the context of oncogenesis. Many lncRNAs have been linked to leukemic cancers, but little is known about their exact functions or how they are constructed at the molecular level. This study examines the expression profile of *UCA1*, an lncRNA associated

with several cancer-causing pathways, in a cohort of Iraqi patients with CML categorized by age and gender. The annual Iraqi cancer registry for 2019 reports 339 chronic myeloproliferative diseases overall. With a greater incidence rate in men than in women (1.12 in men and 0.60 in women), the ratios and rates of incidence were 0.86% and 0.94%, respectively. Through a male-to-female ratio of 1:1.16, the average age at detection was 40.39 ± 14.30 years [13]. Among all the cases, the pediatric age group accounted for 3.7%; the alive and dead groups accounted for 1.8% and 9.4%, respectively. Although it was the lowest in adults aged 60 years and above, at 21.1% (40/189). The juvenile age group had a higher death percentage 62.5% (5/8) than the patient group who was ≥60 years, 42.1% (8/19). Previous research has demonstrated that lncRNAs exert a wide range of effects on cellular physiology. These effects include altering cell lineage, disrupting metabolic balance, promoting uncontrolled cell proliferation, and developing treatment resistance. The goal of this study is to add to what is known about *UCA1*'s role in CML by examining its expression levels and the effects they may have on disease progression. This is especially important for studying Iraqi patients. In previous research [14], using RT-PCR and other techniques, researchers examined the expression of lncRNAs in multiple cancer cell types, including *UCA1* [15]. *UCA1*'s overexpression accelerates bladder cell growth and the spread of metastases. *UCA1* first appeared in bladder cancer and is located on human chromosome 19 (p. 13.12) [16]. According to a recent study, *UCA1* regulates oncogene activity in several kinds of cancers, including colorectal cancer, ovarian cancer, blood cell carcinoma, and breast cancer [17]. To determine the role of *UCA1* expression in predicting survival, patients in a separate study were divided into two groups based on their median *UCA1* level: high expression and low expression. Thus, supporting *UCA1* as a potential prognostic biomarker in CML, high *UCA1* expression was significantly associated with both disease-free survival ($P < .01$) and lower overall survival [18]. *UCA1* has been shown to activate the PI3K/AKT/mTOR signaling pathway [19]. Another study shows that *UCA1* downregulation sensitizes DNR therapy to target acute Leukemia cells. Direct transcriptional repression of BCL2, independent of microRNA interactions, achieves this increased sensitivity, thereby undermining mitochondrial death evasion and fostering cell death [20]. Studies show that *UCA1* may help inhibit PI3K/AKT, which is an important signaling pathway involved in many physiological processes, such as cell growth, invasion, and metastasis. There is a strong link between PD, DN, SLE, and cancer. This process involves vital physiological mechanisms, including cell proliferation, invasion, and metastases. LncRNAs in the PI3K/AKT signaling pathway have recently attracted significant interest due to their role in regulating various diseases [21]. Many investigations have revealed that UCA1 knockdown downregulates multiple miRNAs [22], thereby triggering the AMPK signaling pathway. It also alters the signalling pathways by which miR-200c regulates AMPK and prevents hemangioma cells from growing, migrating, and invading [23]. *UCA1* changes the mTOR signaling pathway that is linked to heart muscle cells and lowers the amounts of miR 122 and miR 143 in those cells. This prevents the cells from running out of oxygen and glucose. Damage and death in cardiomyocytes come from hypoxia/reoxygenation (H/R) or oxygen glucose deprivation (OGD) [23]. In hemangiomas, *UCA1* regulates the Wnt/β-catenin signaling pathway and alters how cells grow and change shape. In breast cancer, Cyclin-dependent kinase inhibitor 1B (CDKN1B) inhibits cell cycle progression and halts the G1 phase. Estrogen Receptor (ER) and Progesterone Receptor (PR) negative breast cancer usually corresponds with a low p27 level, so it is more likely to have a high-grade tumor [24]. Part of the Cip/Kip family, p27, this protein blocks cyclin-dependent kinase (CDK) from functioning. It can attach to cyclin D either alone or in concert with CDK4, its active form. This action stops cyclin D–CDK4 from activating, which controls the G1 stage of the cell cycle. Both wild-type C/EBPα and C/EBPα-p30 isoforms can bind to the *UCA1* promoter,

although they have distinct effects on *UCA1* output. Whereas wild-type C/EBPα reduces *UCA1* transcription, C/EBPα-p30 can boost it. In particular, the expression of *UCA1* increases in AML cases that are cytogenetically normal but have two distinct types of CEBPA mutations. Additionally, *UCA*1 regulates the activity of p27kip1, a protein that controls the cell cycle, thereby allowing AML cells to continue growing [25].

4.Conclusion

This investigation revealed significantly reduced *UCA1* expression in the patient group (0.2742 ± 0.21) compared to the control group (1.0 ± 0.0; $p = 0.0001$). It is known that *UCA1* can regulate genes at multiple levels, but this decrease suggests that lncRNAs are involved in the disease processes under study.

Conflict of interest

The authors declare that they have no pertinent ties or financial participation that could create a conflict of interest for this article.

Reference

[1] R. Elsaid, F. Soares-da-Silva, M. Peixoto, D. Amiri, N. Mackowski, P. Pereira, A. Bandeira, and A.Cumano, "Hematopoiesis: a layered organization across chordate species,"*Frontiers in Cell and Developmental Biology*, vol. 8, p. 606642, 16, 2020.

[2] A. A. Bhat, S. N. Younes, S. S. Raza, L. Zarif, S. Nisar, I. Ahmed, R. Mir, S. Kumar ,SK. Sharawat, S. Hashem, and I.Elfaki, "Role of non-coding RNA networks in leukemia progression, metastasis and drug resistance," *Molecular Cancer*, vol. 19, pp. 1-21, 2020.

[3] D. Pulte, L. Jansen, and H. Brenner, "Changes in long term survival after diagnosis with common hematologic malignancies in the early 21st century,"*Blood Cancer Journal*, vol. 10, no. 5, p. 56, 2020.

[4] Y. Dong, O. Shi, Q. Zeng, X. Lu, W. Wang, Y. Li, and Q. Wang, "Leukemia incidence trends at the global, regional, and national level between 1990 and 2017,"*Experimental Hematology & Oncology*, vol. 9, pp. 1-1, 2020.

[5] H. Kantarjian, E. Jabbour, and S. O'Brien, "Chronic myelogenous leukemia," *Molecular Hematology*, vol. 83-97, 2024.

[6] Y. Liang, E. Li, H. Zhang, L. Zhang, Y. Tang, and Y. Wanyan, "Silencing of lncRNA *UCA1* curbs proliferation and accelerates apoptosis by repressing SIRT1 signals by targeting miR-204 in pediatric AML,"*Journal of Biochemical and Molecular Toxicology*, vol. 34, no. 3, p. e22435, 2020.

[7] C. Dai, H. Qianjiang, R. Fu, H. Yang, A. Shi, and H. Luo, "Epigenetic and epitranscriptomic role of lncRNA in carcinogenesis," *International Journal of Oncology*, vol. 66, no. 4, p. 29, 2025.

[8] A. M. Salih, I. H. Aziz, and F. Y. Mohsin, “Estimation of miRNA 208 effects in inducing epithelial-mesenchymal transition by targeting *CDH2* in breast cancer patients of Iraqi population,” *Iraqi Journal of Science*, vol. 66, no. 1, pp. 103–113, 2025.

[9] J. Li, Y. Li, L. Fu, H. Chen, F. Du, Z. Wang, Y. Zhang, Y. Huang, J. Miao, and Y. Xiao, "Targeting ncRNAs to overcome metabolic reprogramming mediated drug resistance in cancer," *International Journal of Oncology*, vol. 66, no. 5, pp. 1-29, 2025.

[10] J. K. Lone and R. Bhardwaj, "Post-transcriptional Gene Regulation: The Role of Noncoding RNAs, in Non-coding RNAs for Crop Improvement: Concepts and Applications," *Singapore: Springer Nature Singapore*, pp. 41-56, 2025

[11] K. Taniue and N. Akimitsu, "The functions and unique features of LncRNAs in cancer development and tumorigenesis," *International journal of molecular sciences*, vol. 22, no. 2, p. 632, 2021.

[12] J. M. Hughes, I. Legnini, B. Salvatori, S. Masciarelli, M. Marchioni, F. Fazi, M. Morlando, I. Bozzoni, and A. Fatica, "C/EBPα-p30 protein induces expression of the oncogenic long non-coding RNA *UCA1* in acute myeloid leukemia," *Oncotarget*, vol. 6, no. 21, p. 18534, 2015.

[13] A. A. Ahmed, K. J. Khaleel, A. A. Fadhel, and B. A. Al-Rubaii, "Chronic Myeloid Leukemia: A retrospective study of clinical and pathological features," *Bionatura*, vol. 7, no. 3, p. 41, 2022.

[14] H. Choudhry, "*UCA1* Overexpression Promotes Hypoxic Breast Cancer Cell Proliferation and Inhibits Apoptosis via HIF-1α Activation," *Journal of Oncology*, vol. 2021, no. 1, p. 5512156, 2021.

[15] G. Yang, R. Shi, and Q. Zhang, "Hypoxia and oxygen-sensing signaling in gene regulation and cancer progression," *International Journal of Molecular Sciences*, vol. 21, no. 21, p. 8162, 2020.

[16] J. Wang, X. Zhang, W. Chen, X. Hu, J. Li, and C. Liu, "Regulatory roles of long noncoding RNAs implicated in cancer hallmarks,"*International Journal of Cancer*, vol. 146, no. 4, pp. 906-916, 2020.

[17] N. F. Hosseini, H. Manoochehri, S. G. Khoei, and M. Sheykhhasan, "The functional role of long non-coding RNA *UCA1* in human multiple cancers: a review study,"*Current Molecular Medicine*, vol. 21, no. 2, pp. 96-110, 2021.

[18] Z. G. Li, W. C. Xiang, S. F. Shui, X. W. Han, D. Guo, and L. Yan, "Long noncoding RNA *UCA1* functions as miR-135a sponge to promote the epithelial to mesenchymal transition in glioma,"*Journal of Cellular Biochemistry*, vol. 121, no. 3, pp. 2447-2457, 2020.

[19] M. Suicmez, G. Namalir, M. Konus, and H. Ozdil, "Silencing of lncRNA *UCA1* Reverses Doxorubicin Resistance of Breast Cancer Through Inhibiting PI3K/AKT/mTOR Signaling Pathway," *Chemistry Select*, vol. 9, no. 20, p. e202400819, 2024.

[20] Q. Yao, L. Zhang, Y. Wang, J. Liu, L. Yang, and Y. Wang, "LncRNA *UCA1* elevates the resistance of human leukemia cells to daunorubicin by the PI3K/AKT pathway via sponging miR-613," *Bioscience Reports*, vol. 41, no. 6, p. BSR20201389, 2021.

[21] S. Ghafouri-Fard, A. Abak, F. T. Anamag, H. Shoorei, J. Majidpoor, and M. Taheri, "The emerging role of non-coding RNAs in the regulation of PI3K/AKT pathway in the carcinogenesis process," *Biomedicine & Pharmacotherapy*, vol. 137, p. 111279, 2021.

[22] M. M. Al-Attar, S. J. Al-Awadi, and S. Y. Abdulfattah, "Role of miRNA-27a and miRNA-224 in post-transcriptional regulation of PCSK9 gene in Iraqi patient with coronary artery disease," *Human Gene*, vol. 34, pp. 201121, 2022.

[23] Z. Liu, Y. Wang, S. Yuan, F. Wen, J. Liu, L. Zou, and J. Zhang, "Regulatory role of long non-coding RNA *UCA1* in signaling pathways and its clinical applications," *Oncology Letters*, vol. 21, no. 5, p. 404, 2021.

[24] Q. S. Wang, J. Zhou, and X. Li, "LncRNA *UCA1* protects cardiomyocytes against hypoxia/reoxygenation induced apoptosis through inhibiting miR-143/MDM2/p53 axis," *Genomics*, vol. 112, no. 1, pp. 574-580, 2020.

[25] P. Moezi, M. Kargar, A. Doosti, and M. Khoshneviszadeh, "Multiplex touchdown PCR assay to enhance specificity and sensitivity for concurrent detection of four foodborne pathogens in raw milk," *Journal of Applied Microbiology*, vol. 127, no. 1, pp. 262-267, 2019.