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Analysis of miR-155 Expression in Prostate Cancer Patients

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What is already known on this topic?

- *Prostate cancer is the second most common malignancy in men and has multifactorial causes, including genetic, epigenetic, and environmental factors.*
- *miR-155 has been implicated in various cancers and is known to influence critical pathways such as DNA repair (via RAD51), apoptosis, and epithelial–mesenchymal transition (EMT).*
- *In some studies, miR-155 has shown dual roles in cancer, acting either as an oncogene or tumor suppressor depending on the tumor context and microenvironment.*

What this study adds on this topic?

- *This study is one of the first to evaluate miR-155 expression levels in paired tumor and normal prostate tissues using RT-qPCR in a well-defined patient cohort.*
- *It finds no significant difference in miR-155 expression between tumor and normal tissues and no correlation with clinical parameters like PSA level, Gleason score, or PIRADS.*
- *These findings suggest that miR-155 may not serve as a reliable biomarker for prostate cancer in its early stages, contrary to some earlier reports.*

Abstract

Objective: Prostate cancer is a major global health issue and the second most common cancer in men. Its pathogenesis involves genetic and epigenetic changes, hormonal imbalances, and environmental factors, with deficiencies in double-strand break repair mechanisms playing a critical role. microRNAs (miRNAs) are small, non-coding RNA molecules that regulate gene expression post-transcriptionally and are implicated in cancer-related processes.

Methods: microRNA-155 expression was analyzed in paired tumor and normal tissues from 50 patients with pathologically confirmed prostate cancer (PC) using quantitative reverse transcription polymerase chain reaction (qRT-PCR). In addition, clinical parameters such as age, smoking status, diabetes, hypertension, PIRADS scores, and Gleason scores were evaluated for their potential association with miR-155 expression.

Results: No statistically significant difference was observed in miR-155 expression between tumor and normal tissues ($P > .05$). Moreover, miR-155 levels did not show significant correlation with any of the evaluated clinical factors.

Conclusion: These findings suggest that miR-155 may not play a direct role in the development and progression of PC. However, this study contributes to the growing understanding of the relationship between PC, DNA repair mechanisms, and miRNA regulation. Further studies with larger cohorts and standardized methodologies are needed to clarify the potential of miR-155 as a biomarker or therapeutic target in PC.

Keywords: Prostate cancer, microRNA, DNA damage repair, quantitative reverse transcription polymerase chain reaction

Introduction

Prostate cancer (PC) is a disease of epithelial cells that make up the prostate glands and accounts for 14.2% of all newly diagnosed cancers in men worldwide.[1] PC with a worldwide annual mortality of over 200 000[2] is the second most common type of cancer in men globally, following lung cancer.[3] The lifetime risk of developing PC is reported as 1 in 8, and the risk of dying from PC is 1 in 37.[4] PC progresses through a series of genetic and epigenetic changes at the molecular level. During this process, the balance between cellular proliferation and apoptosis (programmed cell death) is disrupted. One of the greatest challenges in understanding the development of PC is identifying the cell of origin that leads to tumor formation. Nearly all PCs (95% adenocarcinomas) arise from glandular tissue.[5] PC is multifocal and genetically heterogeneous, with tumor foci often forming independently in different regions of the gland.[6] The molecular mechanisms responsible for the development of PC are not yet fully understood. However, it is known that changes in the expression of growth factors, particularly transforming growth factor-β (TGFβ), vascular endothelial growth factor (VEGF), and insulin-like growth factors (IGFs), especially IGF-1, can promote the progression of this disease.[7,8] Genes associated with the development of PC are those that are particularly involved in the androgen-receptor signaling pathway and testosterone metabolism.[9] Further investigation into the development and progression of PC is essential for reducing its recurrence and mortality rates. Consequently, identifying novel and reliable biomarkers involved in the pathogenesis of prostate

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neoplasms has become a critical area of research. The identification of alterations in molecules involved in DNA damage repair in PC may lead to the discovery of new biomarkers associated with prostate carcinogenesis.

Changes in the DNA double-strand break (DSB) repair pathways have been associated with the progression of PC and the development of resistance to therapy. In eukaryotic cells, 2 main mechanisms are responsible for DSB repair: homologous recombination (HR) and non-homologous end joining (NHEJ). These pathways are essential for preserving genomic integrity, and malfunctions in either can result in genomic instability and contribute to cancer formation.[10] A variety of proteins are involved in DSB repair, and alterations in these proteins have been identified in cases of PC. microRNAs (miRNAs) are among the molecules involved in regulating DNA repair processes.

microRNAs, a family of small molecules (19-25 ribonucleotides), control gene expression at the post-transcriptional stage by promoting mRNA degradation or inhibiting the translation of specific mRNA targets.[11,12] They play crucial roles in processes such as cell differentiation, cell cycle regulation, and programmed cell death (apoptosis). To date, over 900 miRNAs have been discovered in humans, with each being assigned a unique numerical identifier. Altered expression of miRNAs has been observed in various types of cancer.[13,14] Many miRNAs are located in fragile genomic regions or areas associated with cancer, which could explain the link between tumor development and the abnormal expression of certain miRNAs.[15] Despite the identification of numerous miRNAs, their specific roles in cancer progression and the mechanisms by which they contribute to tumorigenesis remain largely unknown.[16] As the authors' understanding of miRNAs continues to expand, a number of miRNA-based therapeutic strategies are being explored. These approaches are generally designed either to suppress the activity of oncogenic miRNAs that are abnormally upregulated or to restore the function of tumor-suppressive miRNAs whose expression is diminished in cancer.[17,18] Achieving therapeutic efficacy, however, requires a clear understanding of the specific roles individual miRNAs play in tumor development and progression.

microRNA-155 is 22 bp in length,[19] and the gene encoding this miRNA is located on chromosome 21.[20] The sequence encoding miR-155 is also known as the MIR155 host gene (MIR155HG). microRNA-155 is a multifunctional miRNA that significantly influences a range of cellular activities, including proliferation, cellular differentiation, immune regulation, and apoptosis. Due to its extensive involvement in these essential biological pathways, miR-155 is considered a critical factor in both normal cellular homeostasis and the pathogenesis of various diseases.[21] microRNA-155 is also involved in homologous recombination and, in some cases, in NHEJ mechanisms.[22] It is commonly associated with the *RAD51* gene in DNA repair processes. RAD51 plays a critical role in repairing double-strand DNA breaks through homologous recombination. microRNA-155 can influence DNA repair mechanisms by suppressing the expression of RAD51.[22] Recent studies have highlighted the diverse roles of miR-155 in various cancer types.[23,24]

This study aims to explore the clinical significance of miR-155 in PC. For this purpose, changes in miR-155 expression in tumor and normal tissues and their relationships with the clinical findings of patients have been investigated.

Methods

This prospective study included prostate tissue samples obtained from consecutive male patients who underwent Tru-cut prostate biopsies at the Urology Department of İstanbul Education and Research Hospital between 2023 and 2024 due to clinical suspicion of PC. Written informed consent was obtained from all participants. After histopathological verification of tumor and non-tumor (normal) prostate tissues, 50 patients were selected for the final analysis. Pathological evaluations of the tissue samples were conducted at the Pathology Department of İstanbul Education and Research Hospital. Ethical committee approval was received from the Ethics Committee of University of İstanbul University Cerrahpaşa, Cerrahpaşa Faculty of Medicine (Approval no:560526, Date: December 13,2022).

Reverse Transcription-Quantitative Polymerase Chain Reaction

Prostate tissues were initially stored in sterile tubes containing RNA preservative at +4°C for 24 hours. After removing the preservative, the samples were transferred to −80°C for long-term storage. Prior to analysis, the tissues were thawed, lysed with lysis buffer and β-mercaptoethanol, and homogenized for 5 minutes in sterile tubes with ceramic beads using a cooled homogenizer. microRNA was extracted from the homogenized tumor and normal tissue samples using the EXTRACT ME kit (Blirt, Poland) to evaluate miR-155 expression levels. The concentration and purity of the isolated miRNA were measured with a Thermo Fisher Scientific NanoDrop spectrophotometer.

Complementary DNA (cDNA) was synthesized from the isolated miRNA using the ABScript III RT Mix kit (ABclonal) , following the manufacturer's instructions. To ensure consistency, miRNA concentrations were standardized across all samples based on the tissue with the lowest miRNA yield. The resulting cDNA was stored at −20°C for short-term use. Subsequently, miRNA expression levels of miR-155 were analyzed through real-time polymerase chain reaction (PCR), with U6 serving as the reference housekeeping gene. Primer sequences for the genes (Suarge, Türkiye) of miR-155 and U6 were used (Table 1).

Quantitative polymerase chain reactions were conducted using the Abclonal qPCR kit (Applied Biosystems) in a total reaction volume of 20 µL per well. microRNA-155 expression levels were normalized to the expression of the U6 housekeeping gene. The relative miRNA levels of miR-155 in PC and normal tissues were determined using the $2^{-\Delta\Delta Ct}$ method, based on the Ct values of the target and reference genes. The $2^{-\Delta\Delta Ct}$ values were calculated using the following formula.

Statistical Analysis

Gene expression values were expressed as mean ± standard error (SE), whereas age-related data were shown as mean ± SD. The Mann–Whitney *U*-test was used for statistical comparisons between the 2 groups. Pearson's correlation test was employed to assess the relationships between gene expression levels and other variables. All statistical analyses were carried out using IBM SPSS Statistics software version 21.0 (IBM SPSS Corp.; Armonk, NY, USA). A *P*-value of less than .05 was considered statistically significant.

Result

A total of 50 PC patients who had not received any prior treatment were enrolled in the study. Clinical data of the patients were obtained from their medical records. The demographic and clinical data of the patients can be seen in Table 2.

The CT values for miR-155 and U6 (the reference gene) were measured in tumor and normal tissue samples from 50 patients. The ΔCT values were derived by subtracting the CT value of the reference gene from that of the target gene for both tissue types.

Table 1. Identification Numbers for microRNA Assays Used in Quantitative Polymerase Chain Reaction

Gene Code	Accession No	Assay Name and Product No	Amplicon Length (bp)
hsa-miR-155-5p	MIMAT0000646	miRNA qPCR SL Assay for hsa-miR-155-5p, MIREXs-H155-5	64-68
RNU6-6P	Entrez Gene ID: 26826	miRNA qPCR U6 Control Assay for Human RNU6-6P, MIREXs-U6	89

miRNA, microRNA; qPCR, quantitative polymerase chain reaction.

Gene expression was then determined for each patient using these ΔCT values. According to the $2^{-\Delta CT}$ analysis, the gene expression levels of miR-155 in both tumor and normal tissue samples are presented in Table 3. The $2^{-\Delta CT}$ analysis showed no statistically significant difference in miR-155 gene expression between tumor and normal tissues ($P > .05$).

To calculate the ΔΔCT values, the difference between the ΔCT values of tumor and normal tissues was computed. Upon converting the ΔCT values to $2^{-\Delta\Delta CT}$ values to assess the fold change in gene expression, it was found that the expression of miR-155 in tumor tissues was 0.55 times that in normal tissue samples (Table 4).

The expression levels of miR-155 in tumor and normal tissue samples were examined in relation to various clinical factors, including smoking, age, Prostate-specific antigen (PSA) levels, diabetes, hypertension, the presence of nodules, Gleason score, and PIRADS status. However, no significant correlation was observed between miR-155 expression and any of these clinical parameters.

Discussion

PC is the second most common cancer in men and ranks fourth in cancer-related deaths.[25] The incidence of PC increases after the age of 40,[26] reaching its highest levels in the 80s. The development of PC is commonly associated with risk factors such as a family history of PC, obesity, hypertension, lack of physical activity, and high testosterone levels.[27]

Table 2. Demographic and Clinical Data of Prostate Cancer Patients

Age (Years)	66.3 ± 7.1		
PSA	14.6 ± 14.3		
Smoking	Non-Smoker (-)	39 patients	78%
	Smoker (+)	11 patients	22%
Diabetes	–	38 patients	76%
	+	12 patients	24%
Hypertension	–	29 patients	58%
	+	21 patients	42%
Nodule	–	36 patients	72%
	+	14 patients	28%
Gleason Score	<7	29 patients	58%
	>7	21 patients	42%
PIRADS(Prostate Imaging Reporting and Data System)	3	14 patients	28%
	4	17 patients	34%
	5	19 patients	38%

Changes in biomolecules like DNA can trigger the development of cancer. Therefore, the efficiency of DNA repair mechanisms and the functions of molecules involved in regulating this activity are crucial in the carcinogenesis process. The role of miRNAs, which are key modulators of DNA repair mechanisms, and their disease-specific expression patterns are being extensively studied for their potential in the diagnosis and treatment of cancer.[28] microRNA-155 has been shown to target molecules associated with DNA repair mechanisms, particularly the mismatch repair (MMR) and HR systems, in PC. This miRNA regulates the expression of genes such as BRCA1 and RAD51,[29] which play key roles in HR repair, and downregulates the expression of core proteins of the MMR system, including hMSH2, hMSH6, and hMLH1, leading to a mutator phenotype and microsatellite instability (MSI).[30]

microRNA-155 expression is associated with various physiological and pathological processes, including immune response, inflammation, tumor development, and treatment resistance. Chronic inflammation caused by infections, metabolic changes, inflammatory disorders, or other environmental factors is a significant contributor to the onset of various cancers. In the case of PC, prostatic inflammation is considered a potential risk factor. Moreover, the presence of inflammatory cells in PC is often linked to a worse prognosis and increased resistance to treatment.[31] Some studies highlight its oncogenic properties, while others emphasize its anti-oncogenic or pro-immunological characteristics.[32] These features are noted to differ in lymphatic cancers compared to solid tumors. In particular, miR-155 has been found to be generally highly expressed in lymphatic cancers and conditions such as liposarcoma. microRNA-155 levels can vary in an anti-inflammatory environment, and it has been suggested that the change might be minimal depending on its role in specific microenvironments or immune cells.[33] A meta-analysis indicated that high levels of circulating miR-155 in lung cancer patients were detected, suggesting its potential as a non-invasive biomarker, although it was concluded that it does not hold prognostic significance.[32] Changes in miR-155 expression have particularly been observed in urological malignancies. The upregulation of miR-155 expression is frequently seen in urological cancers, where it contributes to tumor progression by targeting specific proteins and signaling pathways.[34]

In the authors' study, we did not find any difference in miR-155 expression levels between tumor and normal tissue, and no relationship was found between these expression levels

Table 3. Expression Level of microRNA-155 in Tumor and Normal Tissue Samples

	Tumor Tissue	Normal Tissue	*P**
miR-155	0.0037 ± 0.0015	0.0136 ± 0.0059	.27

The data are presented as mean ± SE. miR, microRNA. *Mann–Whitney *U*-test.

Table 4. microRNA-155 Expression Levels in Tumor and Normal Tissue Samples

	ΔC_T (Ort)	$\Delta\Delta C_T$(Ort)	$2-\Delta\Delta^{CT}$
Tumor tissue	11.36	0.87	0.55
Normal tissue	10.49	1	1

The data are presented as mean ± SE.
*Mann–Whitney *U*-test.

and the clinical data of the patients. The exact role of miR-155 in PC remains unclear. Studies suggest that miR-155 may play a dual role in PC. It could act as an oncogene[34] or a tumor suppressor,[35] depending on the genes it targets and the tumor microenvironment. In 2015, findings related to the significance of miR-155 in PC were first reported.[36] It has been shown that miR-155 expression is upregulated in PC tissues and cells.[37] Overexpression of miR-155 significantly promotes PC cell proliferation, while downregulation of miR-155 induces cell cycle arrest and promotes apoptosis in PC cells.[38] On the other hand, a study by Ji et al[39] demonstrated that miR-155 inhibits the secretion of VEGF by suppressing the TGF-β/SMAD2 signaling pathway in PC, weakening angiogenic potential. This suggests that miR-155 could also be used as an inhibitor in PC. Abnormalities in epigenetic regulation are common findings in carcinogenesis. In PC, increased promoter methylation of miR-155 has been observed compared to normal tissue, leading to the downregulation of miR-155 gene expression.[40] In a study by Guo et al,[37] the expression levels of miR-155 in PC were positively correlated with tumor-node-metastasis (TNM) stage, tumor volume, and lymph node metastasis, and negatively correlated with tumor grade. However, in the authors' study, no significant correlation was found between miR-155 expression and the clinical variables used. The difference in findings may be attributed to the smaller sample size of 86 patients in this study, which could have caused numerical differences between subgroups of clinical variables. Although miR-155 has emerged as a promising therapeutic target, discrepancies in clinical data, including the authors' own findings, highlight the complexity of its role in PC progression. Since miR-155 plays a dual role in PC, modulating its expression through antagomiRs,[41] mimics, or exosome-based delivery systems[42] has shown promise in regulating oncogenic pathways and enhancing therapeutic efficacy. Combining these RNA-based approaches with natural compounds may offer a multifaceted and targeted strategy for PC treatment.

Conclusion

In this study, aimed at exploring the potential role of miR-155 in PC development and progression, no significant difference was found in miR-155 expression levels between cancerous and normal tissues. Additionally, no correlations were observed between miR-155 expression and clinical parameters such as PSA levels, tumor grade, or other patient characteristics. These findings suggest that miR-155 may not have a major role in the pathogenesis of PC, at least within the context of the studied cohort. However, given the complex and context-dependent functions of miR-155 reported in various cancers, its role in PC cannot be entirely ruled out. Further studies with larger sample sizes, diverse patient populations, and comprehensive clinical data are needed to better understand the regulatory mechanisms of miR-155 and to assess its potential as a diagnostic or prognostic biomarker. A deeper understanding of miR-155's function could ultimately contribute to the development of more precise diagnostic tools and targeted therapies for PC.

Data Availability Statement: The data that support the findings of this study are available on request from the corresponding author.

Ethics Committee Approval: Ethical committee approval was received from the Ethics Committee of University of İstanbul University Cerrahpaşa, Cerrahpaşa Faculty of Medicine (Approval no: 560526, Date: December 13, 2022).

Informed Consent: Written informed consent was obtained from the patients who agreed to take part in the study.

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