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Validation of novel molecular markers for some non-coding RNA genes as regulators of PolyADP-ribose polymerase (PARP1) gene expression in women with breast cancer.

Samaa M. Sayel and Owayes M. Hamed

Hamdaniya University \ Laser and Photonics Center\ Department of Laser and Spectroscopy

Mosul University \ College of Science \ Biology Department

Abstract

DNA damage leads to mutations and epimutations that occur through a process of natural selection. MiR-96-5p was first recognized as a 23-nucleotide miRNA, and substantial evidence has demonstrated its association with cell proliferation and apoptosis. miR-96-5p represents a significant oncogenic miRNA in the context of breast cancer. Poly [ADP-ribose] polymerase (PARP1) serves as a crucial component in the DNA repair process. this study aimed to detect the correlation some types of ncRNA genes with activity of PARP1 gene in women with breast cancer. This study includes 40 women who received confirmed diagnoses of breast cancer, alongside 20 healthy participants who served as controls. And detect the levels of gene expression for PARP1, MiR-96-5p, snoRNA and lncRNA. Our study showed increase in gene expression for MiR-96-5p (5.451) and PARP1 gene (4.791) in blood samples, and snoRNA (5.083), lncRNA (6.110) and PARP1 genes (4.341) in biopsy samples compare with control group 1.0. Also this result showed decrease in gene expression for MiR-96-5p (2.516), snoRNA (0.5329) and lncRNA (0.6432) in biopsy biopsy samples compare with healthy samples. The concussion for this study can depend on MiR-96-5p as molecular marker in breast cancer. Also the miRNA was more abundant in the blood and could be utilized for disease diagnosis, whereas lncRNA levels were elevated in the tissue.

Keywords: breast cancer, DNA repair, MiRNA, SnoRNA, lncRNA, PARP1 gene

Introduction

Breast cancer denotes malignancies that arise from breast tissue, predominantly from the inner lining of the milk ducts or the lobules that produce milk for the ducts. The primary causes of aberrant cellular programming in cancer are genetic and epigenetic alterations. For example, point mutations, deletions, duplications, insertions, translocations, chromosome aberrations, viral infections, and epigenetic inactivation represent various types of potentially cancer-causing events [1]. DNA damage seems to be a critical issue for living organisms. DNA damage leads to mutations and epimutations that, through natural selection, may result in the progression to cancer, [2-3]. DNA double strand break may lead to alterations in chromosome structure, and if such changes are transmitted to subsequent cell generations, they represent a type of

mutation. The mechanisms in question may influence the DNA sequence and/or alter the function and regulation of gene products, potentially resulting in a loss of function [4]. Genetic variation in DNA repair genes may lead to changes in DNA repair function, which can result in the accumulation of DNA damage. This may subsequently trigger programmed cell death (apoptosis) or lead to unregulated cell growth and cancer [5-8]. Base-excision repair (BER) is an important DNA repair pathway responsible for the repair of base damage resulting from X-rays, oxygen radicals, and alkylating agents [9]. MiRNA is a non-coding single RNA strand has a crucial role in the regulation of gene expression by controlling the degradation of proteins as well as RNA stability [10]. MiR-96-5p was initially identified as a 23-nucleotide miRNA and abundant evidence has shown that miR-96 is related to cell proliferation and apoptosis. miR-96-5p is a promising oncogenic miRNA in breast cancer, linked to tumor progression and poor outcomes [11]. Copy number variation (CNV) represents a distinct category of genomic structural variation, with sizes varying from around 50 bp to several Mb, and is primarily defined by deletions and duplications [12]. Small nucleolar RNAs (snoRNAs) range from 60 to 300 nucleotides in length and, as indicated by their name, are primarily located in the nucleolus. They serve as guide RNAs for the post-transcriptional modification of ribosomal RNAs and certain spliceosomal RNAs [13]. LncRNAs are characterized as RNA molecules with a transcript length greater than 200 nucleotides that are not translated into proteins. Transcription is carried out by RNA polymerase II [14]. PolyADP-ribose polymerase (PARP1) serves as a crucial component in the DNA repair process. PARP1 is a prevalent and highly conserved cell signaling protein that specifically catalyzes the poly ADP-ribosylation of DNA-binding proteins, thereby influencing their activity. PARP1 plays a crucial role in the repair of DNA single strand breaks (SSB), which is a sub-pathway associated with base excision repair. The loss of PARP1 is associated with persistent single-strand breaks that are converted to DNA double-strand breaks following the collapse of replication forks [15].

Aim of study: this study aimed to detect the direct effect of miRNA96, snoRNA and lncRNA on the activity of PARP1 gene in women with breast cancer.

Materials and Methods

Case of study

This study includes where 40 women received confirmed diagnoses of breast cancer and 20 healthy participants acted as controls. Based on histopathological assessment, most patients had stage II tumors, while a smaller percentage had stage I or III tumors and Based on clinical staging, the majority of patients were stage II or III. The researchers chose patients through Oncology and Nuclear Medicine Hospital records combined with examination outcomes performed at the hospital. based on ethics of scientific research approval form of a research protocol/ ministry of health and environment (form number 02/2025).

RNA, MiRNA, lncRNA and snoRNA extraction from blood

The extraction of RNA from blood was conducted using the Trizol kit supplied by Transgeneic. We established a method for the simultaneous extraction of mRNA and ncRNA, utilizing the Trizol-phenol-chloroform principle for sample lysis and silica membrane purification for total RNA and ncRNAs, according to the extraction protocol.

Poly A polymerase (Polyadenylation)

To identify ncRNA during real-time PCR analysis, Poly A protocol must be carried out immediately after RNA extraction, utilizing DNase and RNase-free tips at all stages of the process.

Converted to cDNA: After the extraction of mRNA and Non-coding RNA were completed, all molecules converted to cDNA using (RT-PCR technique) using reverse transcriptase enzyme activity.

detection the gene expression levels of mRNA molecules for PARP1 gene by used qPCR technique

Specific primers of PARP1 gene and housekeeping gene used to detect the gene expression levels by q-PCR as shown in the table (1):

Table (1): primers of PARP1 genes and housekeeping gene for mRNA molecules [16]

Primers	Sequence

PARP1-F_RT	5´CCTGATCCCCCACGACTTT´3
PARP1-R_RT	5´GCAGGTTGTCAAGCATTTC´3
H.K_F	5´GACCCAGATCATGTTTGAG ´3
H.K_R	5´CGTACAGGGATAGCACAG ´3

Detection the expression levels for ncRNA gene that affected on BER genes by used qPCR technique

Specific primers designed by primer3 software for this study used to detect the gene expression levels of micRNA and snoRNA and lncRNA molecules (which regulated the PARP1 gene) and U6 gene by q-PCR as shown in the table (2):

Table (2): primers of micRNA and snoRNA molecules and lncRNA and housekeeping gene designed by primer3 software for this study

miRNA	specific gene	Primers Sequence
miR-96-5p-F	PARP1 gene	AACACGCTTTGGCACTAGCAC
miR-96-5p-R	PARP1 gene	CAGTGCAGGGTCCGAGGT
lncRNA	BC069792 F	CCAGCCACGTTCTTCTTGGT
lncRNA	BC069792 R	AGGCCCAGTGCTGTTAAAGA
SnoRNA	SNHG7 F	GGAAGTCCATCACAGGCGAA
SnoRNA	SNHG7 R	GTCAGGATCACGCAGGACAG
U6-F	H.K	5´ GTGCTGCTTGGGCAGCA 3´
U6-R	H.K	5´ GAAATATGGAACGGTTC 3´

qPCR reaction volume

The component of reaction with final reaction volume 20µl, the Ultra syber q-PCR master mix was first component with 10 µl volume, forward primer RT with 0.5 µl volume, Reversed primer RT with 0.5µl volume, the template of cDNA with 4 µl volume and distilled water with 5 µl volume.

qPCR reaction program

The RT-PCR program for mRNA and ncRNA molecules was four stages; Pre denaturation at temperature 95°C for time period 10 min, Denaturation at temperature 95°C for time period 15 sec, Annealing /Extension at temperature 60°C for time period 1 min and Melting curve analysis at temperature 95°C for time period 15 sec, then temperature 60°C for time period 1 min, then temperature 95°C for time period15 sec and temperature 60°C for time period 15 sec.

Statistical analysis

Statistical analysis was performed using Graph Pad Prism software. The statistical relationships among the clinic pathological variables associated with breast cancer were assessed using a t-test and Anova one way to analyze quantitative data. To establish a threshold for evaluating breast cancer risk using a Receiver Operating Characteristic (ROC) curve, you would analyze the curve's area under the curve (AUC) and specific sensitivity and specificity values at different threshold points. The correlation between the variables was examined through Pearson correlation analysis, which provides a correlation coefficient (r) that quantifies the interrelations between two continuous variables. In all statistical analyses, a p-value of less than 0.05 is considered statistically significant.

Result and discussion

Analysis of PARP1 gene expression

The expression levels of the PARP1 gene were measured in both blood and biopsy samples from patients and compared to healthy controls. The mean expression in blood samples was **4.791**, while in biopsy samples it was **4.341**, both markedly higher than in control samples, which showed a mean expression level of only **1.028**. This suggests a significant upregulation of PARP1 gene expression in patient tissues compared to normal controls. **Also** the relative value between PARP1 gene expression in blood sample and control is 0.34 and between biopsy samples and control is 0.45 and there are no relative value between levels in biopsy and blood samples**.** That is mean there is a high genetic variation between the blood and the control, and between the tissue and the control.

Table (3): Mean and Standard error of study groups for PARP1 gene

PARP1 GENE	Blood	Biopsy	Control
Mean	4.791	4.341	1.028
Std. Error of Mean	1.063	0.7704	0.1154

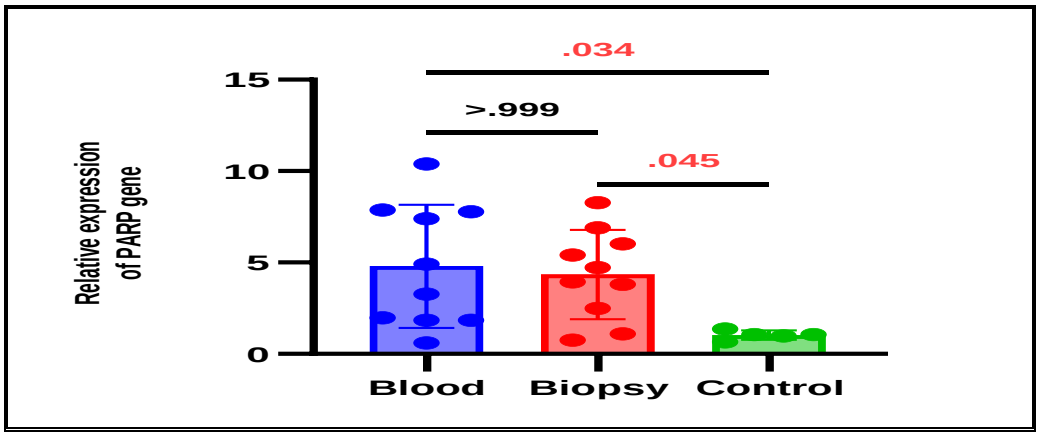

Figure1: Relative PARP1 gene expression comparison between patients and controls

PARP1 (Poly ADP-ribose polymerase) is a key component of the cellular response to DNA damage, especially in processes like base excision repair and chromatin remodeling. Elevation in PARP1 expression in both blood and biopsy samples indicates an increased demand for DNA repair activity in the diseased state. Such upregulation may support tumor cell survival by enhancing their ability to repair endogenous or therapy-induced DNA damage [17]. Elevated PARP1 expression may also contribute to cancer progression and therapy resistance. By facilitating continuous DNA repair, tumor cells may evade apoptosis and develop resilience to genotoxic treatments [18]. Inhibition of PARP1 activity has therefore become a therapeutic strategy, especially in tumors deficient in homologous recombination repair mechanisms, as blocking PARP1 can push cells into synthetic lethality [19].

The ROC analysis explains the groups of research male and female groups against the controls. In male vs control the AUC (Area Under the Curve) quantifies the diagnostic accuracy the finding of AUC = **0.9** that's Indicates excellent discriminatory power. While in female vs controls the AUC= **0.9** and leading to Indicates excellent discriminatory power also.

Table (4): ROC analysis of relative PARP1 gene expression comparison between patients and controls

Groups of PARP1 gene	AUC	95% confidence interval	Sensitivity%	Specificity%	Cut off	P value
Blood vs control	0.9	0.7141 to 1.000	90%	100%	> 1.612	0.0143
Biopsy vs control	0.9	0.7342 to 1.000	80%	100%	> 1.933	0.0143

Analysis of miRNA96 gene expression

The quantitative expression analysis of miRNA-96 revealed notable differences between patients and controls. In blood samples from patients, the mean expression level was **5.451**, whereas in biopsies it dropped to **2.516**. In contrast, healthy controls exhibited a lower expression mean of **2.288**. The elevation in blood suggests an upregulation of miRNA-96 systemically, while the expression within tissue samples is comparatively moderate. **Also** the relative value between PARP1 gene expression in blood sample and control is **.208** and between biopsy samples and control is **.995** and there is no relative value between levels in biopsy and blood samples**.** The miRNA is showed the highest value in the blood, meaning it can be used as a marker to diagnose a disease.

Table (5): Mean and Standard error of study groups for MiRNA 96.

MIRNA 96	Blood	Biopsy	Control
Mean	5.451	2.516	2.288
Std. Error of Mean	1.294	0.4565	1.000

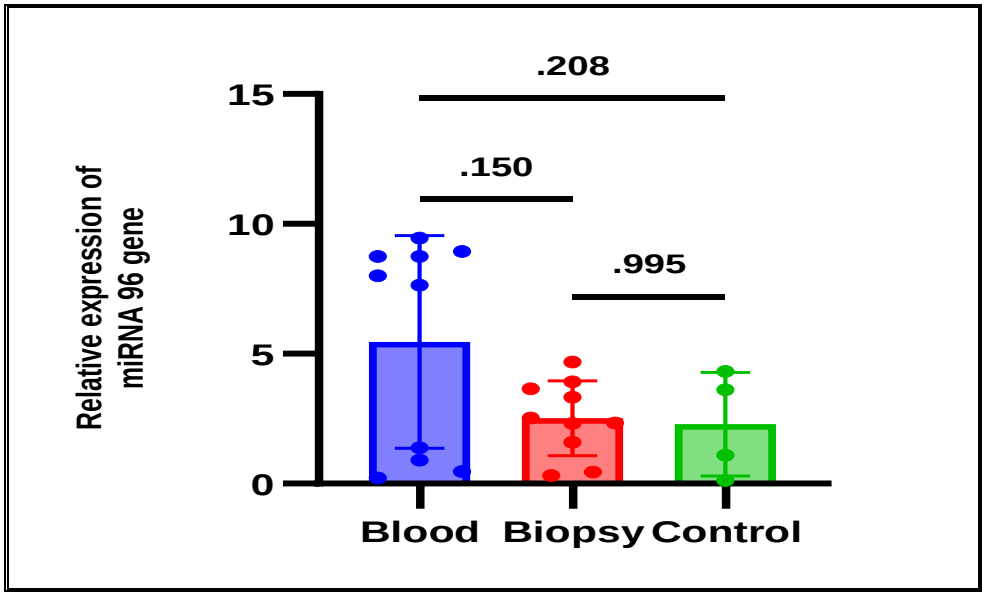

Figure2: Relative miRNA 96 gene expression comparison between patients and controls

miRNA-96 is recognized as a crucial regulator of post-transcriptional gene expression, particularly in the context of cancer progression. Its upregulation in patient blood samples points toward a systemic molecular dysregulation potentially driven by tumor-derived exosomes or circulating tumor cells that secrete or release specific miRNAs into the bloodstream. This elevated circulating level of miRNA-96 may function as a tumor-associated signal, participating in the inhibition of apoptosis, enhancement of cell proliferation, and modulation of oncogenic signaling pathways such as PI3K/AKT and FOXO. This differential pattern between blood and tissue samples supports the idea that miRNA-96 could serve as a dual biomarker: indicating systemic disease activity and potentially reflecting tumor burden[20]. Overexpression of this MiRNA may contribute to tumor invasiveness, epithelial-to-mesenchymal transition (EMT), and resistance to apoptosis—all key hallmarks of malignancy. The strong expression level in blood samples emphasizes the importance of considering miRNA-96 not only as a diagnostic marker but also as a potential therapeutic target, particularly in strategies aiming to restore tumor suppressor activity or normalize deregulated pathways. The observed pattern in this study aligns with the general understanding of miRNA-96 as an oncomiR—a MiRNA that promotes oncogenic behavior [21].

Analysis of lncRNA gene expression

The expression of lncRNA BCO was analyzed across blood and biopsy samples in patients, in comparison to healthy controls. The mean expression level in blood samples was **0.6432**, which is notably lower than in controls (**1.146**). However, biopsy samples showed a significant upregulation, with a mean expression of **6.110**. This dramatic elevation within tissue samples, coupled with the downregulation in circulation, suggests a compartmentalized role of lncRNA BCO in the disease process. **Also,** the relative value between PARP1 gene expression in blood sample and biopsy is < **0.01** and between biopsy samples and control is **<0.01** and there is no relative value between levels in biopsy and blood samples**.** It was shown that the only affects the tissue, meaning that the tissue is the target tissue affected.

Table (6): Mean and Standard error of study groups for lncRNA BCO

LncRNA BCO	Blood	Biopsy	Control
Mean	0.6432	6.110	1.146
Std. Error of Mean	0.1278	0.6875	0.4375

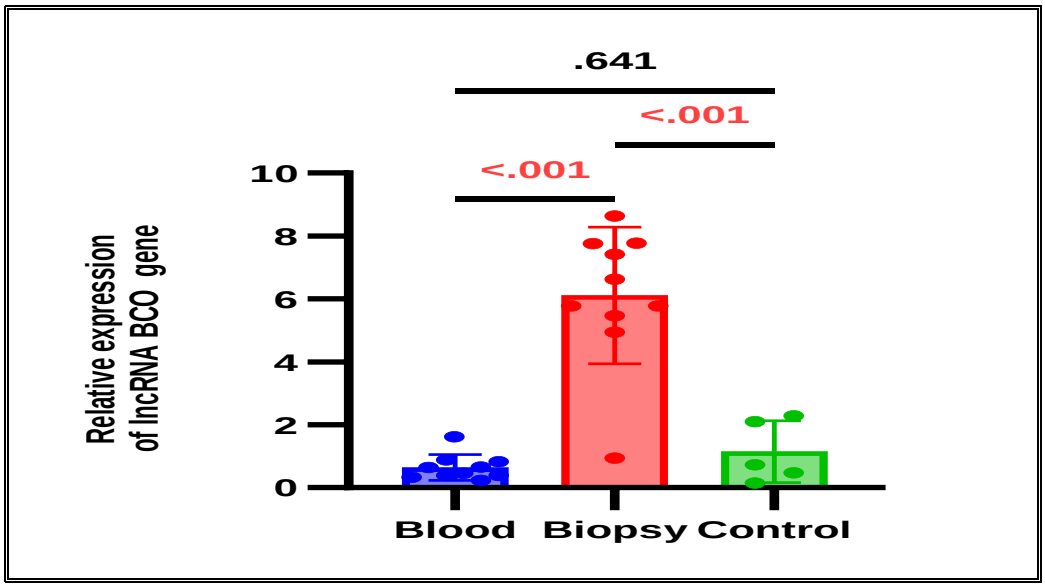

Figure3: Relative lncRNA BCO gene expression comparison between patients and controls

Long non-coding RNAs such as BCO are increasingly recognized for their diverse roles in gene regulation, chromatin remodeling, and cellular signaling, particularly in malignancies. The significantly elevated levels of lncRNA BCO in biopsy samples indicate a likely involvement in tumor growth or maintenance at the tissue level. Such overexpression may contribute to oncogenic processes through epigenetic regulation, modulation of transcription factors, or by acting as competitive endogenous RNAs (ceRNAs) that sequester tumor-suppressive miRNAs. The observed suppression of lncRNA BCO in the bloodstream may reflect one of two possibilities. First, the lncRNA may be predominantly retained within tumor cells due to its intracellular function, resulting in minimal secretion or release into circulation. Alternatively, the downregulation in blood might suggest systemic regulatory mechanisms attempting to counteract its pathological over activity within tumor tissues. Either way, this sharp contrast between compartments underscores the localized nature of its function and raises the possibility of using biopsy-based BCO expression as a diagnostic or prognostic marker. lncRNAs are also known to interact with pathways involved in immune evasion, angiogenesis, and metastasis. The highly upregulated expression in tumor tissue could point to such roles, especially in promoting invasion or modulating the tumor microenvironment. The strong expression of lncRNA BCO in tissue may have clinical relevance not only for diagnosis but also for therapeutic response prediction [25-27].

Analysis of snoRNA gene expression

The analysis of small nucleolar RNA (snoRNA) expression revealed marked variations between patient samples and controls. In blood samples, the mean snoRNA expression was **0.5329**, which is significantly reduced compared to control levels (**1.432**). In contrast, biopsy samples showed a substantial increase, with a mean expression level of **5.083**, indicating a strong upregulation in tumor tissue relative to both circulation and healthy tissue. **Also,** the relative value between PARP1 gene expression in blood sample and biobsy is **< 0.01** and between biopsy samples and control is **<0.04** and there is no relative value between levels in biopsy and blood samples**.** It was shown that the snoRNA affects the tissue, meaning that the tissue is the target tissue affected.

Table (7): Mean and Standard error of study groups for snoRNA.

snoRNA	Blood	Biopsy	Control
Mean	0.5329	5.083	1.432
Std. Error of Mean	0.1123	0.5735	0.5666

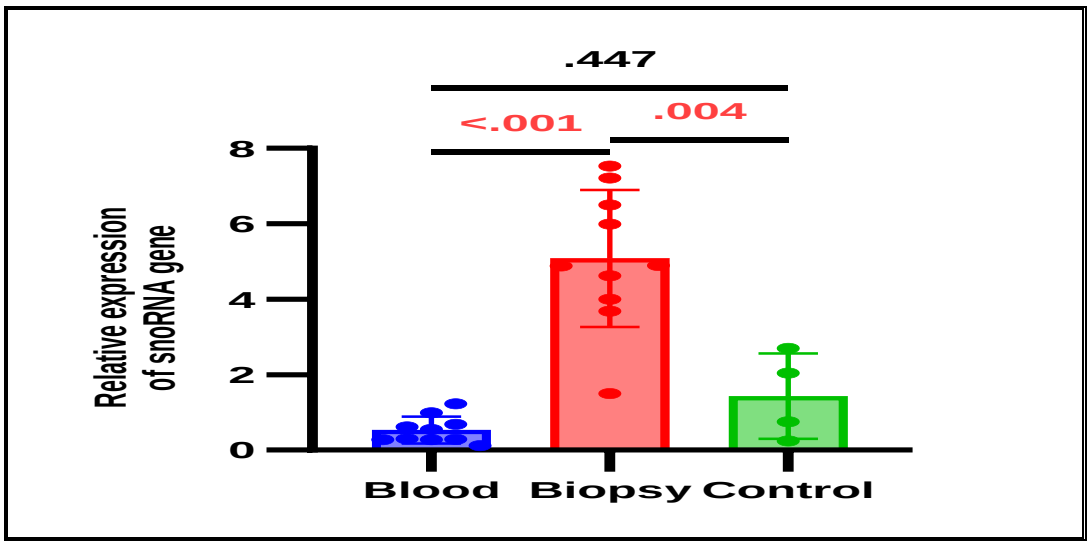

Figure4: Relative snoRNA gene expression comparison between patients and controls

snoRNAs have traditionally been associated with ribosomal RNA modification and processing within the nucleolus. The current findings suggest a dual regulatory pattern, with suppression in circulation and activation within tumor tissue, reflecting a possible shift in snoRNA function during tumorigenesis. The high expression of snoRNA in biopsy samples suggests that these molecules may be coopted by tumor cells to support rapid proliferation and metabolic adaptation. Tumor environments often require enhanced ribosomal biogenesis and protein synthesis, both of which are supported by snoRNA-mediated modifications. Additionally, snoRNAs can serve as precursors for smaller regulatory RNAs with miRNA-like activity, further contributing to cancer progression through post-transcriptional regulation of gene expression [23]. Conversely, the downregulation observed in blood may be attributed to reduced release of these RNAs into circulation or increased degradation in the extracellular space. This could also reflect a mechanism by which tumor cells sequester snoRNAs for internal use, thereby minimizing their presence systemically.

Such patterns indicate that snoRNAs, particularly when evaluated in tissue samples, could serve as effective biomarkers for tumor detection and classification [24].

Correlation Between miRNA-96 and PARP1 Gene Expression

Correlation analysis between miRNA-96 and PARP1 gene expression was conducted separately for both blood and biopsy samples. The statistical correlation in blood samples showed a Person's r value of **–0.4684** with a p-value of **0.1722**, while the biopsy samples revealed a correlation of **–0.3526** with a p-value of **0.3177**. These values indicate a negative trend in both cases, though neither reached statistical significance ($p > 0.05$). meaning there is a reflection in the results if the MiRNA is raised, the gene expression decreases. It has been shown that the PARP1 gene and MiRNA can be relied upon in the follow-up of the case in diagnosing the disease.

Table (8): Correlation between miRNA and PARP1 gene expression levels between study groups

Correlations	R	95% confidence interval	P value
miRNA 96 vs. PARP1 (blood)	**-0.4684**	**-0.8479 to 0.2287**	**0.1722**
miRNA 96 vs. PARP1 (biopsy)	**-0.3526**	**-0.8038 to 0.3561**	**0.3177**

Although a negative correlation was observed between miRNA-96 and PARP1 gene expression in both blood and biopsy samples, the lack of statistical significance suggests that the interaction between these two molecules may not be strongly linear or consistent across patients. The inverse trend implies that higher levels of miRNA-96 might be associated with reduced PARP1 expression, potentially through direct or indirect regulatory mechanisms, as miRNAs typically downregulate their target genes by binding to complementary sequences in the mRNA [22]. In a biological context, such a relationship would be plausible since miRNA-96 has been implicated in targeting tumor suppressor or DNA repair-related genes in several cancer models. If PARP1 were among its downstream targets, overexpression of miRNA-96 could

feasibly suppress PARP1-mediated DNA repair activity, potentially sensitizing tumor cells to genotoxic stress. Another possibility is that PARP1 expression is influenced by multiple overlapping regulatory inputs, including other miRNAs, transcription factors, and epigenetic modifiers, which may dilute the apparent correlation with any single miRNA. Similarly, miRNA-96 itself may act on a broader network of targets, and its influence on PARP1 expression may vary depending on cellular context, tumor stage, or external stressors such as therapy. The modest negative correlation remains biologically suggestive but highlights the need for mechanistic studies and larger sample sizes to confirm functional interactions. Moreover, post-transcriptional regulation is often nonlinear and context-dependent, which may explain the observed variability in the dataset[23].

Conclusion

revealed the negative correlation was observed between miRNA-96 and PARP1 gene expression in both blood and biopsy samples, The inverse trend implies that higher levels of miRNA-96 might be associated with reduced PARP1 expression, potentially through direct or indirect regulatory mechanisms, as miRNAs typically downregulate their target genes by binding to complementary sequences in the mRNA**.**

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