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RESEARCH ARTICLE

The Impact of miRNA-34a expression, RETN Genetic Polymorphism and Protein Level on Breast Cancer Progression

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Abstract

Introduction: Obesity, inflammation, and a variety of malignancies, including breast cancer, are all linked to the adipokine resistin. The goal of this study was to determine if specific polymorphism in the RETN (rs3219175) gene in addition to its level enhance the chance of breast cancer susceptibility and to determine if miRNA -34a expression affect the resisten protein levels and its role in breast cancer progression. **Materials and Methods:** This study included 100 participants (50 patients and 50 control groups) ranging in age from 35-70 years. Blood samples were collected from Al-Eluia Hospital for Women's Care, an Oncology Teaching Hospital, between January and July 2021. For the molecular experiment, DNA was extracted from the whole blood that stored in EDTA tube and genetic analysis for the studied SNP was by taq man method with special prop while for RETN protein levels were quantified by ELISA. While for miRNA -34a expression, the total RNA was extracted from the whole blood for both the patients and control groups with the using of miRNA specific primers and by using specific extraction kit with U6 that were used as internal housekeeping gene. **Results:** The results showed that the GG genotype was more common in the control group than in the patients (8.9% vs. 5.3%), and that this relationship is significantly associated with the disease (OR= 0.21, p=0.002), while genotype GA was more common in the control group than in the patients (33.9 percent vs. 32.1 percent, p= 0.922). The genotype AA was found to be more common in patients than in controls (65.5 percent vs. 57.1 percent, respectively) with an etiological factor of (OR= 2.63, p=0.012), indicating a significant departure from Hardy Weinberg suggestion for control and patient groups ($\chi 2$= 2.301, 4.103) respectively. Serum RETN levels were significantly higher in patients compared to controls (126.52 ±2.36 vs 62.55 ±0.97, p =0.001). **Conclusion:** At the same time, the results showed that expression of miRNA34a gene in patients was downregulated as compared with controls (39.04 vs 37.73) and its reversibly correlate to RETN levels.

Keywords: Breast Cancer- RETN gene- miRNA34-a- Single Nucleotide Polymorphism

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Introduction

Breast cancer is the most often diagnosed cancer in women, accounting for 11.6 percent of all cancer cases worldwide, according to Globocan cancer incidence and mortality. Age, reproductive and gynecological variables, physical activity, consumption of alcohol and nicotine, as well as family history [1, 2] and gynecological illnesses such as adenomyosis and polycystic ovarian syndrome all influence the chance of developing breast cancer [3].

For determining an individual's level of breast cancer risk, genetic testing and mammography screening have low specificity and sensitivity [4]. Single nucleotide polymorphism (SNP) genotyping may help forecast an individual's risk of breast cancer and guide disease therapy, according to researches [5, 6].

Resistin (RETN) is a cysteine-rich protein with 12.5-kDa released by adipose tissue on a continuous

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basis [7]. RETN is thought to play a role in inflammation and immunological responses, as well as acting as a pro-inflammatory mediator [8]. The RETN gene, which codes for RETN, is found on chromosome 9 and contains numerous single nucleotide polymorphisms (SNPs) in the promoter and 3′-untranslated regions [9]. RETN gene expression has been found to be increased in human breast cancer tissue [10] and polycystic ovary syndrome [11]. MicroRNA (miRNA) is defined as a single-stranded RNAs with a18-25 nucleotides length. Accumulative evidences from numerous biological experiments demonstrates that miRNAs play an essential and significant role in many biological processes such as gene expression regulation by post transcriptionally binding to 5'untranslated regions (UTR), coding sequences, or 3′UTR of target messenger RNAs (mRNAs) [12]. The miR-34 family members, miR-34a is more prevalent than miR-34b/c in cancer research. the detailed downstream and upstream mechanisms of miR-34a remain ambiguous [10]. Among the miRNAs, miR-34a is a pivotal anti-oncogene miRNA [13].

Although RETN genetic polymorphisms and altered miRNA-34a expression have each been independently associated with breast cancer, no previous study has simultaneously investigated the interaction between RETN rs3219175 polymorphism, circulating resistin protein levels, and miRNA-34a expression within a single patient cohort. This integrated genetic and epigenetic approach represents a critical gap in understanding inflammation-driven breast cancer susceptibility, particularly in Middle Eastern populations. The aim of the study to evaluated a functional RETN gene polymorphism (rs3219175) with its protein level and miRNA34-a expression in the susceptibility to breast cancer among a sample of Iraqi women patients.

Materials and Methods

This study included 50 patients (women) suffering from breast cancer and 50 age-matched healthy women subjects. Participants were recruited from an Oncology Teaching Hospital, which may overrepresent patients with advanced or clinically severe disease. Therefore, the findings may not fully reflect the broader population of breast cancer patients. The sample size (n = 100) was determined based on patient availability during the study period. While adequate to detect moderate genetic associations, the authors acknowledge that larger cohorts are required to confirm genotype-specific risks and improve statistical power.

This study was approved by the Ethics Committee of the Institute of Genetic Engineering and Biotechnology for Postgraduate Studies, University of Baghdad, and the Iraqi Ministry of Health and Environment (Approval No.: IOGEAB-A00401). All procedures were conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrollment.

About 6 ml of blood sample was withdrawen from both patients and control and divided in to three parts, the first 2 ml was used for RETN serum level evaluation using an Enzyme-Linked Immunosorbent Assay (ELISA) kit from Innova Biotech CO.LTD Ltd., China, with cat. no. In.hu2287. This technique involves coating a microplate with antibodies specific to RETN. The serum sample or a standard containing a known amount of RETN is then added to the wells. A second antibody, conjugated to horseradish peroxidase (HRP), binds to the captured RETN. After washing away unbound components, a substrate (TMB) is added, which is catalyzed by HRP to produce a blue color. The intensity of this color, measured at 450 nm, is directly proportional to the amount of RETN in the sample. By comparing the sample's absorbance to a standard curve, the concentration of RETN can be accurately determined [22].

Patient and healthy subjects blood sample have been collected in EDTA tube and kept under -30 C until the time of work. By using commercial kit (mini-prep ZYMO research, USA), genomic DNA was isolated from the blood samples. The electrophoresis has been done to detect the isolation process by running DNA (5 µl) through agaros gel (2%). Stained with RedSafe (Intron, Korea), the gel have been subjected to 90 voltage for 45 minutes. A specific region of the RETN gene that contain the desired SNP have been amplified by using RT –PCR and two kinds of probe in order to detect the RETN genotype (rs3219175) SNP. The sequence of sense primer; GGGCCCAGGGACTTATTAGC. And the anti-sense primer; CGACCTCCTGGATCCTCTCA. The sequence of probe that align to the sequence of G allele; FAM-CTGTCTGCTCAGCGGCTTCCTCTT-BHQ, while the probe that align to the A allele was; HEX-CTGTCTGCTCAGTGGCTTCCTCTT-BHQ. Genotype frequencies were analyzed using odds ratios with 95% confidence intervals; however, subgroup analyses were interpreted cautiously due to limited sample size per genotype. The RT-PCR reaction mixture components were KAPA SYBR FAST qPCR Master Mix (2X) Universal (10 µL) with final concentration 2x, And each of Forward, Reverse primers, Probe 1, 2 (0.4 µL) with final concentration 0.2Mm with Up to 10 µL Nuclease-free water. And template DNA Sample Volume with final concentration1pg-100ng.

The cycling condition and temperatures for the amplification and detection of RETN gene.

of the RT-PCR are 95 °C to activated enzyme for 05:00 min.at Hold cycle, and at the denaturation, Annealing and Extension cycle (95 °C for 30 sec., 60 °C for 30 sec. and 72 °C for 30 sec., respectively). with total cycles (40), Once the genotypes of all the samples have been revealed the frequency of each genotype have calculated.

The rest 2 ml of blood was used for estimation of miRNA34a gene expression levels, which were evaluated using the qRT-PCR SYBR Green assay on the QIAGEN Rotor-Gene Q Real-Time PCR System (Germany) to quantify target gene expression. The primers are listed in Table 1.

The reaction was performed in a final volume of 20 µL. According to the manufacturer's protocol, it was determined that 10 µL of TransStart® Top Green

Table 1. The Primer Sequence for miRNA 34-a and U6 Gene

Primer	Sequence (5′→3′ direction)	primer size bp	Ta °C	Reference
U6	F 5-CTCGCTTCGGCAGCACA-3	24	59	2
	R 5-AACGCTTCGGCAGCACA-3	22		
miRNA 34-a gene	F 5 - GGTTTTTTTCAATCAGCAAGTATAC -3	22	60	Designed
MiRNA 34-a gene	R 5 -GTTGGCTCTGGTGCAGGGTCCGAGGTATT -3	26		

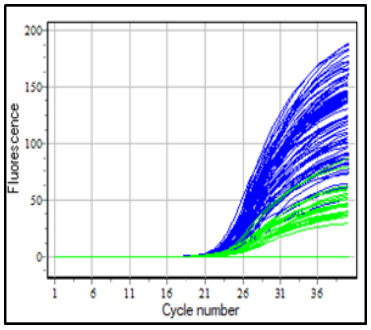

Figure 1. Amplification Curves of RT-PCR Results

qPCR Super Mix (Cat no. AQ131-01, TransGen Biotech Company, Beijing, China) would be required to prepare the required number of reactions. Three microliters of cDNA were used as the template, one microliter of each of the forward and reverse primers, and five microliters of nuclease-free water were added to finish the reaction's final volume. Thermal Cycling conditions for qRT PCR of U6 and miRNA34a genes expression are 94 °C to for 60 sec. at initial denaturation for 1 cycle, and at the denaturation, Annealing and Extension cycle (94 °C for 60 sec., 60 °C for 15 sec. and 72 °C for 20 sec., respectively). with total cycles (35). The Dissociation was 65-95 °C for 1 sec. and 1 cycle. U6 functions as a housekeeping gene and is a short nuclear RNA (snRNA). Quantitative real-time PCR (qRT-PCR) was used to measure gene expression levels in both the patient and control groups. Cycle threshold (Ct) values were established for a housekeeping gene and each target gene. Two well-known techniques were utilized for data analysis: the ΔCt method and the ΔΔCt method for fold-change computation [15, 16].

The Statistical Analysis System (SAS, 2018) software was employed to evaluate the effects of different groups (patients and controls) in the study parameters [17].

Results

After the completion of RT-PCR, each sample showed a curve of amplification the G allele represented by Fam channel and the A allele represented by the Hex channel. The results are shown in Figure 1.

The results of genotypes frequencies have been calculated and summarized in Table 2 for the SNP rs3219175. The results showed higher frequency of GG genotype in control than patients (8.9% V.S. 5.3%, respectively) and this relation is significantly protective (OR= 0.21, p=0.002). The genotype GA showed higher frequency within the control group than the patients and that relation was non-significant (33.9% V.S. 32.1%, p= 0.922). in another hand the genotype AA showed higher frequency in patients than in control (65.5% V.S. 57.1%, respectively) with an etiological factor equal to (OR= 2.63, p=0.012). the results of Hardy-Weinberg equation are shown in Table 3 and showed a great departure from hardy Weinberg suggestion for both group, control (chi square= 2.301) and patients (chi square= 4.103).

The comparison of patients with breast cancer and control was applied according to the serum level of RETN, as in Table 4.

As shown in the Table 4 the serum level of RETN were 126.52±2.36 in Brest cancer patients while it was 62.55 ±0.97 in control group this elevation was highly significant (p=0.0001).

Using the ΔΔCt approach, Table 5 compares the expression of the miRNA34a gene between breast cancer patients and control groups. the mean Ct of miRNA34-a in the patients group was 39.04 at the same time, The mean Ct of miRNA34-a in the control group was 37.73, which suggests that patients express less miRNA 34-a with fold change value of 0.51as shown in Table 5.

That The genetic polymorphism of RETN gene, SNP rs 3219175 may be interact with dysregulation of

Table 2. Genotypes Frequencies of rs3219175 and their Related Odds Ratio, p-value and Confidence Intervals

Genotype Frequency					
Genotype	Control	Patients	P-value	OR Ratio	95% Cl
	n = 50	n = 50			
GG	11 (8.9%)	5 (5.3%)	0.002	0.21	0.08 to 0.66
GA	19 (33.9%)	12 (32.1%)	0.922	0.812	0.42 to 2.01
AA	20 (57.1%)	33 (65.5%)	0.012	2.63	1.21 to 5.50
Allele frequency (%)					
Allele	Control	Patients	P-value	OR Ratio	95% Cl
	n = 50	n = 56			
G	23(25.9%)	24 (21.4%)	0.53	0.73	0.42 to 1.44
A	83 (74.1%)	88 (78.5%)			

Table 3. Genotypes Frequency and Hardy-Weinberg Equation

Groups			Genotypes			HWE p>0.05	Alleles	
			GG	GA	AA		G	A
Control	Observed	No.	11	19	20	2.301	41	59
		%	22	38	40		41	59
	Expected	No.	8.4	17.4	0.41			
		%	16.81	34.81	0.82			
Patients	Observed	No.	5	12	31	4.103	22	74
		%	10	24	62		22	74
	Expected	No.	2.52	16.95	28.52			
		%	5.04	33.91	57.04			

Table 4. RETN Serum Level in Breast Cancer Patients and Control Group

RETN (ng/ml)	Brest patients (No. = 50)	Control (No. = 50)
Mean ± S.E.	126.52 ±2.36	62.55 ±0.97
T-test	6.908 **	
P-value	0.0001	

(**) Significant Increase P < 0.01 in BC patient compared to control. S.E: Standard Error, No: Number, P: Probability.

Table 5. Comparison of Research Patients and Control Groups in miRNA34-an Expression

Group	Mean Ct Target Gene	Mean Ct Housekeeping Gene	Δ Ct	$2^{-\Delta Ct}$	Fold change
Controls	37.736	24.394	13.342	0.000095	1
Patients	39.04	24.41	14.63	0.000049	0.51

miRNA 34a. expression, that in fact lead to increase the aggressiveness of the tumor phenotype, so the enhancement of breast cancer growth and treatment resistance as shown in Figure 2.

Discussion

In breast cancer, resistin promotes tumor development, treatment resistance, and metastasis [12-14]. Resistin has been shown to affect epithelial to mesenchymal transition and stemness in breast cancer cells in a mechanical investigation, which could be mediated by cyclase-associated protein 1 (CAP1) [15]. This is first study investigates the breast cancer and RETN gene polymorphism (rs3219175) among patients from different regions of Baghdad's geographical location.

The RETN polymorphisms have been found in a variety of malignancies, including colon and lung tumors [7, 10, 16], although data on RETN polymorphisms in breast cancer is limited. To our knowledge, Wang et al., [4] study is the first to look into the distributions of the rs3219175 SNPs and their links to the development and progression of breast cancer in Chinese Han women. Women with the GG genotype of the RETN rs3219175 polymorphism were shown to have a higher risk of breast cancer, whereas those with at least one A allele in rs3219175 were found to have a higher risk of breast cancer than those with wild-type GG homozygotes, this agree with our study. A another study has reported that resistin level was found to be higher in the lung cancer (NSCLC) patients and associated with cancer cachexia [17].

According to accumulating evidence, there are genetic or epigenetic alterations have shown in the RETN gene. Moreover, genetic polymorphisms in RETN have been identified in colorectal, colon, and breast cancers [16, 18]. The elevated RETN levels are significantly increased in breast cancer patients, suggesting a possible role of this adipokine in the pathogenesis and progression of the disease. Resistin is known to be secreted by adipose tissue and is associated with chronic inflammation, insulin resistance, and tumor-promoting microenvironments, which may enhance cancer cell proliferation, invasion, and metastasis.

The highly significant elevation of RETN in breast cancer cases compared to controls supports its potential use as a biomarker for disease detection or risk stratification. Moreover, this result is consistent with previous studies linking obesity, metabolic dysregulation, and elevated inflammatory mediators with a higher

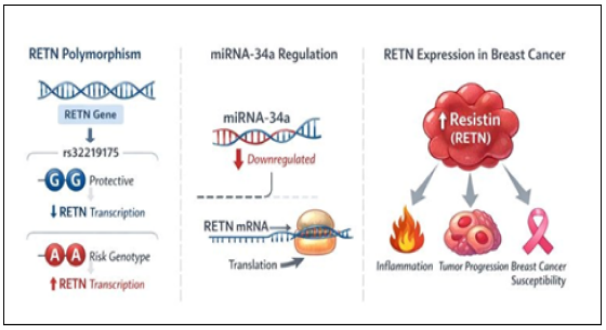

Figure 2. Schematic Representation of RETN Genetic Variation, miRNA-34a Regulation, and Resistin-Mediated Breast Cancer Susceptibility

incidence of breast cancer.

Based on the known biological functions of both molecules, an inverse relationship is expected between miRNA-34a expression and RETN protein levels. miR-34a acts as a tumor suppressor that inhibits inflammatory and EMT-related pathways, while resistin promotes inflammation, proliferation, and metastasis. Therefore, decreased miR-34a expression may allow for increased RETN activity, contributing to breast cancer progression.

Therefore, RETN may not only reflect an inflammatory state in breast cancer but could also serve as a promising target for therapeutic or prognostic applications in clinical practice [19].

The finding of the present study strongly supports the Role of MiRNA 34a down regulation in Breast cancer pathogenesis by indirect effect-on increase resistin level, these finding support the role of MiRNA 34a as a potential genetic biomarker for breast cancer while resistin may be considered as the mediator linking between inflammation to breast cancer progression.

In Conclusion, this study demonstrates that RETN rs3219175 polymorphism, with elevated resistin levels, and reduced miRNA-34a expression are significantly associated with breast cancer in Iraqi women. The AA genotype was identifying as a risk factor, whereas the GG genotype showed a protective effect against breast cancer. Resistin levels were markedly higher in patients, indicating its role in tumor-related inflammation and disease progression. The downregulation of miRNA-34a and its inverse correlation with resistin suggest a regulatory interaction contributing to breast cancer development. These findings highlight the potential of RETN genetic variation and miRNA-34a as complementary biomarkers for breast cancer susceptibility and progression, warranting further validation in larger and functional studies.

Recommendations and Future Perspectives

Future studies should include larger, multicenter cohorts to validate the association of RETN rs3219175 polymorphism, serum resistin levels, and miRNA-34a expression with breast cancer risk and prognosis. Functional in vitro and in vivo studies are recommended to elucidate the molecular mechanisms linking miRNA-34a regulation to RETN expression and downstream inflammatory and oncogenic pathways. Longitudinal studies are also needed to assess the prognostic value of these biomarkers in disease progression, treatment response, and survival outcomes. Clinically, integrating RETN genetic and molecular profiling into breast cancer risk assessment may improve early detection and personalized therapeutic strategies, particularly in populations with high obesity-related inflammatory burden.

Ethics Statement

The protocol of the study was received by the Council of the Institute of Genetic Engineering and Biotechnology for Postgraduate Studies at the University of Baghdad. And the Ministry of Health and Environment–Baghdad.

Author Contributions

H.A.J. and A.A.S. designed the study. H.A.J., R.A.O., and S.J.K. performed the experiments. S.J.K. and H.F.L. analyzed the data. A.A.S. supervised the work and revised the manuscript. All authors approved the final manuscript.

Data Availability

Data are available from the corresponding author upon reasonable request.

Conflict of Interest

Authors declare that there is no conflict.

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