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Evaluation of XRCC2 Expression in Breast Cancer

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Abstract: Breast cancer (BC) is a leading cause of mortality among women worldwide. The X-ray repair cross-complementing group 2 (XRCC2) gene is implicated in DNA repair processes, and its role in BC remains controversial. This study aimed to analyze XRCC2 mRNA expression in BC tissues compared to normal breast tissues to elucidate its potential role in BC pathogenesis. An observational analytical study with a case-control design was conducted at Zheen International Hospital, Erbil, Iraq, from 2021 to 2024. The study included 44 adult women diagnosed with BC, and XRCC2 mRNA levels were measured using real-time quantitative reverse transcription PCR (qRT-PCR). RNA was extracted, converted to cDNA, and analyzed by qRT-PCR. The XRCC2 mRNA expression levels were normalized using GAPDH and statistically analyzed using the $2^{-\Delta Ct}$ method. The study found a significant upregulation of XRCC2 expression in BC tissues compared with normal controls ($p<0.05$), especially in patients aged 40-55 years and those > 56 years ($p=0.0392$ and $p=0.0191$, respectively), and in higher BC grades II and III ($p=0.0013$ and $p=0.0051$, respectively). Invasive ductal carcinoma exhibited a notable increase in XRCC2 expression ($p=0.0006$). In conclusion, the increased XRCC2 mRNA expression in BC tissues suggests a possible oncogenic role of XRCC2 in BC development. The correlation between age and cancer grade indicates its potential as a marker for BC progression.

Keywords: Breast cancer, Gene expression, qRT-PCR, XRCC2

Introduction

Breast cancer (BC) is one of the most common (with 2.26 million cases in 2020) and the second leading cause of death (with 685,000 deaths in 2020) from cancer in women (Wilkinson & Gathani, 2022). It is a complex and heterogeneous disease, with various genetic and environmental factors contributing to its development and progression (Abiola et al., 2024). One of the key aspects of BC research is the study of DNA repair genes, as they play a crucial role in maintaining genome integrity and preventing the accumulation of mutations that can lead to cancer (Moon et al., 2023).

Among these genetic elements, the X-ray repair cross-complementing group 2 (XRCC2) gene has emerged as a candidate of interest due to its involvement in DNA repair mechanisms. The XRCC2 gene is integral to the homologous recombination (HR) repair pathway, a critical system for maintaining genomic stability and repairing DNA double-strand breaks, which, if left unrepaired, can lead to tumorigenesis (Andreassen and Hanenberg, 2019, Prime et al., 2024).

Recent studies have suggested that alterations in the expression levels of XRCC2 may be associated with the development of certain cancer types, including BC (Shi et al., 2022, Liu et al., 2023, Yu & Wang, 2023). The gene's product, XRCC2 protein, is a part of the RecA/Rad51-related protein family, is known for facilitating the exchange of strands between homologous DNA molecules (a key step in the repair process) (Liu et al., 2023). In

BC, the fidelity of DNA repair mechanisms is particularly crucial, as genetic mutations driving the disease are often a result of DNA repair errors (Alhmoud et al., 2020).

Recent advances in molecular techniques have presented new opportunities to dissect the complexities of cancer biology. mRNA expression analysis, in particular, has become a cornerstone in studying gene expression alterations in various cancers, including BC (Malone et al., 2020, Velaga & Toi, 2022). By quantifying mRNA levels, researchers can infer the activity of genes of interest and elucidate their potential involvement in tumorigenesis and progression. This approach is instrumental in validating biomarkers for cancer diagnosis and prognosis, as well as in identifying new therapeutic targets (Perron et al., 2018).

The necessity to delve into the molecular landscape of BC is underscored by the heterogeneous nature of the disease, which impedes the efficacy of a one-size-fits-all approach to treatment. The present study is predicated on the hypothesis that XRCC2 expression levels may serve as a diagnostic and prognostic marker in BC and could offer insights into the disease's molecular phenotype. Therefore, the present study aims to provide a detailed analysis of XRCC2 mRNA expression in BC tissues, employing real-time quantitative reverse transcription polymerase chain reaction (qRT-PCR) to achieve high sensitivity and specificity.

Method

Study Design and Setting

In this observational analytical research study employing a case-control design, the focus was on investigating the XRCC2 gene's correlation with BC. Conducted between 2021 and 2024, this study involved the meticulous collection of specimens from Zheen International Hospital in Erbil, Iraq.

Participants

The study included adult women 18 years and older who had been diagnosed with BC through histological confirmation and had given informed consent. Patients with previous malignancies, undergoing chemotherapy or radiation therapy prior to sample collection, lacking complete medical records, or declining to participate were excluded from the study. All subjects provided informed permission before sample collection, and the Local Ethics Committee accepted the study procedure (Approval number: 05.01.2020\17). A total of 88 samples, comprising 44 normal and cancerous tissue samples from the breast, were analyzed using the prevailing sampling technique.

Table 1. The key reagents and conditions

Step	Component	Volume for reaction	Volume for negative control	Incubation conditions
One	Total RNA	1-6 µl	-	-
	Primer d(T)23 VN	2 µl	-	-
	Nuclease-free H2O	Variable	-	-
Two	Denaturation	-	-	5 min at 70°C
Three	M-MuLV Reaction Mix	10 µl	10 µl	-
	M-MuLV Enzyme Mix	2 µl	-	-
	Nuclease-free H2O	-	Variable	-
Four	Incubation	-	-	1 hr at 42°C
	(If random primer used)	-	-	5 min at 25°C
Five	Inactivation & Dilution	-	-	80°C, then dilute

RNA Extraction and Complementary DNA Synthesis

The extraction and synthesis of RNA into cDNA from breast tissue can be summarized in a streamlined process with attention to specific conditions and reagent volumes. Initially, RNA was extracted using a Thermofisher

kit, and its concentration was measured with a NanoDrop device. The conversion of RNA to cDNA involved the Ipsogen RT Kit and thermal cycling with the Master-cycler pro-PCR System. To prevent contamination, the workspace was cleaned with 70% ethanol, and filter tips were used throughout the process. A no-reverse transcriptase control was included to check for DNA contamination. For cDNA synthesis, the procedure began by mixing RNA with a primer d(T)23 VN in RNase-free conditions, followed by a denaturation step to improve yield, particularly for long mRNAs and CG-rich regions. This was achieved by heating the RNA-primer mixture at 70°C for 5 minutes, then quickly cooling it on ice. Next, reaction mixes and enzymes were added to initiate synthesis, with specific conditions for the negative control. Incubation at 42°C for an hour allowed for cDNA synthesis, with a preliminary step at 25°C for 5 minutes when using a random primer mix. The reaction was terminated by heat inactivation at 80°C. The final product was diluted (30 µl with H2O) and stored for PCR, ensuring the cDNA did not exceed 10% of the PCR reaction volume (Table 1).

Primer Design

In the study, the design of primers was a critical step for amplifying the XRCC2/Exp mRNA sequences. This task was accomplished using an online primer design tool (http://workbench.sdsc.edu). The primers were meticulously crafted to span the entire coding sequence of the gene of interest, incorporating one or two exon-exon junctions within the design. The inclusion of these junctions was strategic to ensure the amplification was specific to the RNA transcript, avoiding the unintended amplification of any possible genomic DNA contaminants. Details regarding the primer sequences, their specific annealing temperatures, and the expected sizes of the PCR products were systematically cataloged in Table 2.

Table 2. Primer sequences.

Gene name		Primer sequence	Optimal annealing temperature	PCR product Size
XRCC2	F R	TGTTTGCTGATGAAGATTCAC TCGTGCTGTTAGGTGATAAAGC	59.2 °C	255 bp
GAPDH	F R	GGTCCACCACCCTGTTGCTGT AGACCACAGTCGATGCCATCAC	59,4 °C	456 bp

Real-time PCR

To optimize the PCR conditions for specific cDNA primers, a gradient PCR experiment was carried out using an ABI Vertti PCR System. The most effective annealing temperature was determined by evaluating the yield on a 2% agarose gel, which was identified to be 59.2°C for the XRCC2 primers. The PCR mixture, with a total volume of 25µL, included 15µL of dH2O, 2.75µL of 10X PCR buffer with ammonium sulfate, 2µL of 25 mM MgCl2, 1.5µL of 2 mM dNTP mix, 1µL each of 20 mM forward and reverse primers, 0.125µL of 5 U/mL Taq DNA polymerase, and 1.5µL of cDNA template. Table 3 shows the PCR gradient reaction conditions.

Table 3. Conditions of gradient PCR reaction.

No	Step	Temperature	Time
1	Pre denaturation at	94°C	7 minutes
2	Denaturation at	94°C	40 seconds
3	Primer annealing	55°C - 60°C	40 seconds
4	Extension	72°C(40 cycles)	40 seconds
5	Final extension	72°C for	5 minutes
6	Hold	4°C	0

After the PCR, the products were evaluated via 2% agarose gel electrophoresis stained with ethidium bromide, subjected to a 100-volt run for 60 minutes, and observed under UV light. Real-time PCR analysis was carried out on a RotorGene 5 plex system utilizing RT² SYBR Green ROX FAST Mastermix. The reaction mixture for evaluating XRCC2 expression was prepared with 10.5µL of the master mix, 1µL each of forward and reverse primers at 10 µM, 15.5µL of RNAse/DNAse free water, and 2.5µL of cDNA (50 ng), resulting in a total reaction volume of 30µL. The Real-Time PCR procedure involved an enzyme activation at 94°C for 10 min, succeeded by 40 cycles of denaturation at 94°C for 10 sec, primer annealing at the optimized 59.2°C for 40 sec, and extension at 72°C for 40 sec.

Normalization and Statistical Analysis

Normalization of gene expression data was conducted by employing GAPDH as an internal control gene. The relative quantification of XRCC2 mRNA levels was computed through the 2−ΔCt method, and statistical analysis was carried out utilizing SPSS software (version 22.0). The statistical significance of disparities in XRCC2 expression between tumor and normal tissue was assessed using Student's t-test, with a significance level set at 0.05.

Results and Discussion

Table 4 shows the basic data and statistical significance according to age, cancer grade, and cancer types of the patients. In terms of age distribution, most of the participants were 20 (45.45%) between 40- 55 years old. For the Grades variable, most of the participants 18 (40.9%) had grade II cancer. Additionally, in the cancer types, Metaplastic carcinoma, matrix producing type was observed in 12 participants (27.27%), invasive ductal carcinoma in 22 participants (50.0%), and carcinoma Medullary-like in 10 participants (22.73%).

The increased expression of XRCC2 exhibited notable significance within the age groups of 40-55 years and those aged above 56 ($P<0.05$). Similarly, a statistically significant elevation in XRCC2 expression was observed among patients diagnosed with BC grades II and III ($P<0.05$). Conversely, this augmentation did not reach statistical significance in individuals with cancer grade I ($P>0.05$). Moreover, a significant rise in XRCC2 expression was identified in cases of invasive ductal carcinoma ($P<0.05$) (Table 4).

Table 4. Statistical significance according to age, cancer grade and cancer types.

Variables	NO. (%)	XRCC2 Expression (p-value)	Mean of differences	SD of differences	SEM of differences
			Age		
<40 years	13 (29.54)	0.8149	0.050	0.7832	0.2093
40- 55 Years	20 (45.45)	0.0392	0.0280	0.5653	0.1264
> 56 years	11 (20.45)	0.0191	0.3200	0.3553	0.1123
Cancer Grade					
I	9 (20.45)	0.8273	-0.06667	0.8874	0.2958
II	18 (40.9)	0.0013	0.3722	0.4099	0.09661
III	17 (38.65)	0.0051	0.2412	0.5789	0.1404
Brest cancer type					
Invasive ductal carcinoma	22 (50.0)	0.0006	0.3682	0.4247	0.09055
Carcinoma Medullary like	10 (22.73)	0.8088	0.0500	0.6346	0.2007
Matrix producing metaplastic	12 (27.27)	0.6647	0.1083	0.8426	0.2432

SEM: Standard error of means

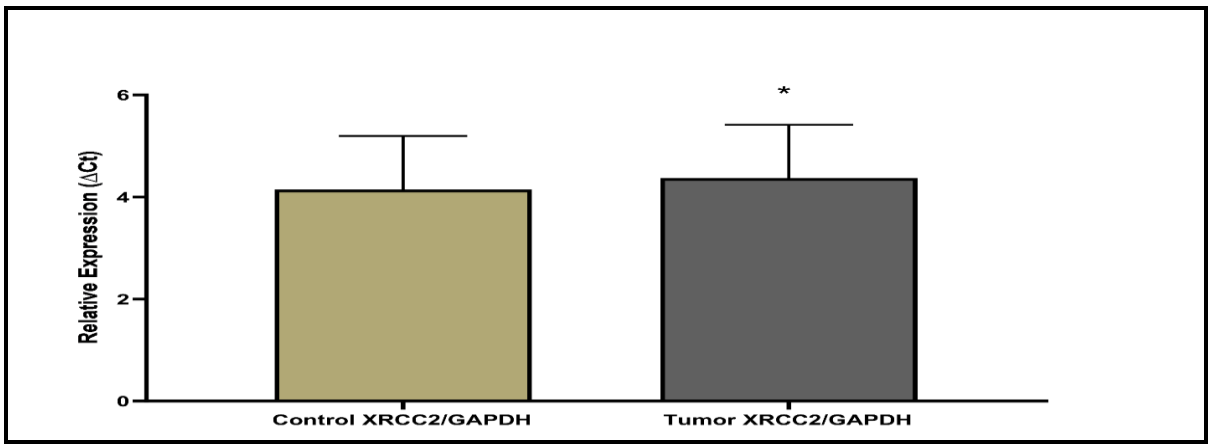

Figure 2. The level of expression of mRNA

Figure 2 illustrates the statistical results of the level of expression of mRNA in both normal controls and tumors. The results reveal that the expression of the XRCC2 gene is significantly increased (upregulated) in tumor samples compared with normal tissues ($p<0.01$). In addition to the statistical data, Figure 3 presents the amplification curve of XRCC2 in real-time PCR. Also, Figure 4 shows the melting curve of XRCC2 in real-time PCR.

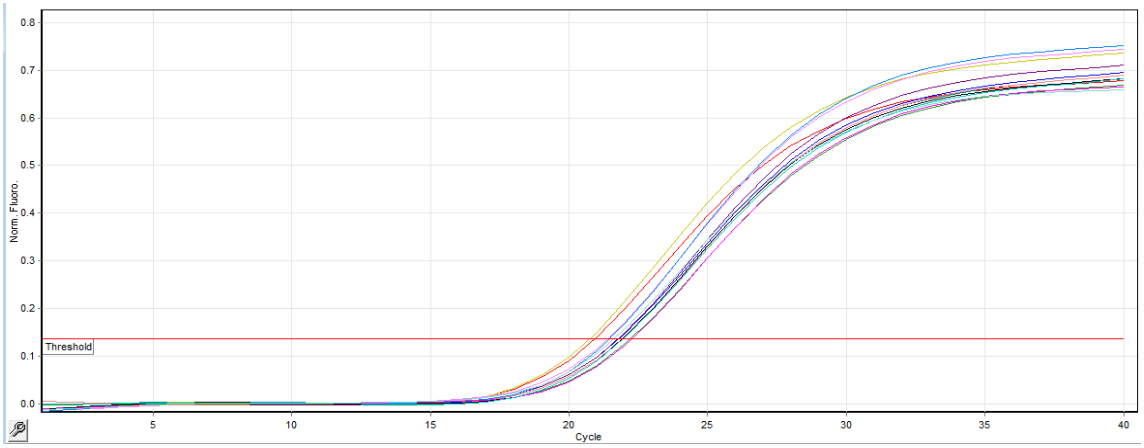

Figure 3. Amplification curve of XRCC2 in real-time PCR.

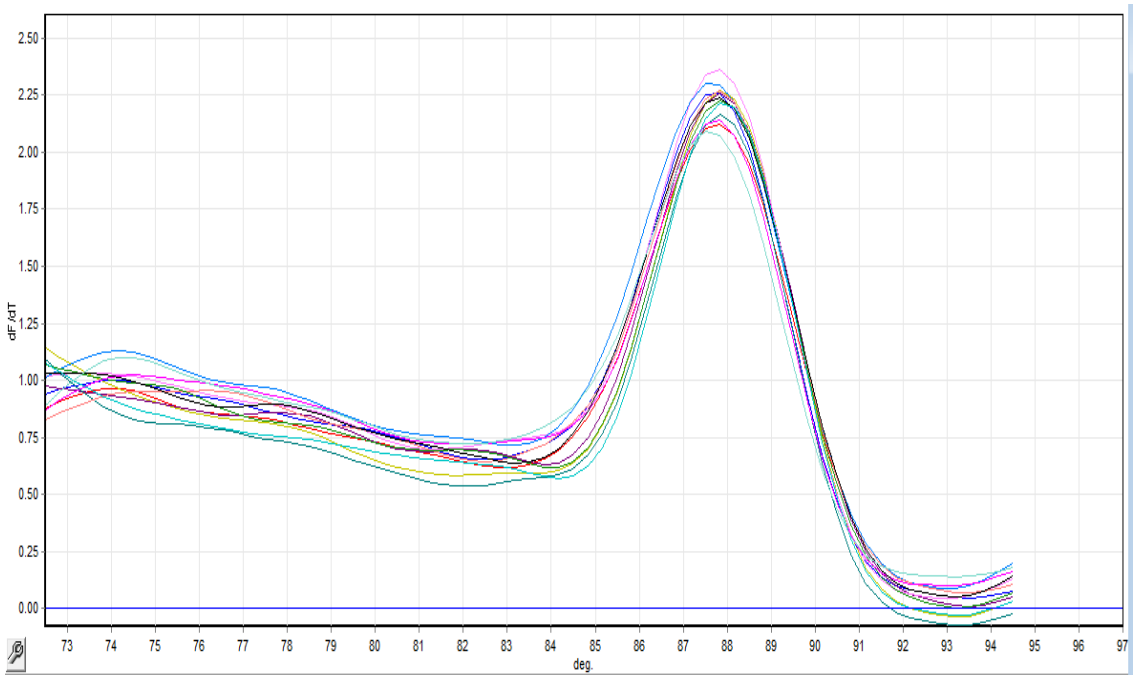

Figure 4. Melting curve of XRCC2 in real-time PCR

Breast cancer stands as the prevalent form of cancer in women and a primary contributor to female mortality rates (Siegel et al., 2024). Studies have shown that XRCC2 gene polymorphisms can impact BC susceptibility (Wei-Yu et al., 2011). Additionally, meta-analyses have highlighted the significance of genetic variability in DNA repair genes like XRCC2 in BC risk (Yu & Wang, 2023).

Quantitative real-time reverse transcription polymerase chain reaction (qRT-PCR) is a highly sensitive and specific method for measuring gene expression levels, including the expression of XRCC2 in BC (Castelló et al., 2002, Ho-Pun-Cheung et al., 2009). The gene expression change of XRCC2 was done by the RT-qPCR method and the data was analyzed by the ΔCT method. The results showed that the expression level of this gene in tumor samples upregulated significantly compared to the adjacent healthy tissue. Considering the regenerative function associated with this particular gene (Andreassen and Hanenberg, 2019), it is anticipated that the level of its expression would diminish throughout the progression of tumorigenesis, exhibiting a tumor-

suppressive characteristic; However, findings from this investigation indicate an upregulation of this gene in cases of BC, suggesting an oncogenic attribute.

The results showed that XRCC2 mRNA expression was significantly upregulated in BC tissues compared to normal controls, suggesting that XRCC2 plays a role in the development of BC. In line with the results of the present study, Shi et al. (2023) support the role of XRCC2 in BC, but what draws attention is that, in their study, they point out that the expression of XRCC2 mRNA decreased; It is contrary to the results of the present study (Shi et al., 2022). Bashir et al.'s (2014) study also discovered that XRCC2 mRNA expression is decreased in BC (Bashir et al., 2014). This difference may be caused by the grade and type of BC and the number of participants and the methods used. Therefore, more studies are needed to investigate this difference. In contrast, other studies also confirm the findings of the present study and state that XRCC is overexpressed in glioblastoma (Liu et al., 2021), colorectal (Xu et al., 2014), stomach cancer (Gok et al., 2014), and BC (Mohamed et al., 2021).

Statistical studies showed that the expression of this gene has a significant relationship with the age of the patients so it showed a higher level of expression in patients 40 years old and above. The studies conducted by Qureshi et al. (2015) and Kluźniak et al. (2019) also confirm this finding, because most people who had mutations in the XRCC2 gene were over 40 years old (Qureshi et al., 2015, Kluźniak et al., 2019).

In line with the present study, the study by Chen et al. (2018), and Zhang et al. (2012) showed that XRCC2 is highly expressed in human tumor cell lines and tissues (Zheng et al., 2012, Chen et al., 2018). Additionally, the results of the present study showed that the increase in cancer grade is related to the higher expression of XRCC2. A study undertaken by Zhang et al. (2017) demonstrated a notable elevation in XRCC2 expression in colorectal cancer (CRC) tissues compared to normal tissues. This heightened XRCC2 expression was linked with more advanced T staging, M staging, TNM staging, Duke's staging, and increased liver and lymph node metastases. The study suggests that XRCC2 expression could serve as an independent prognostic marker for patients with CRC (Zhang et al., 2017). As mentioned, despite accepting the carcinogenicity of XRCC2, some studies suggest that it is premature to consider XRCC2 as a BC-predisposing gene (Kluźniak et al., 2019). Therefore, more studies on the carcinogenesis of XRCC2 in BC are needed.

Conclusion

In conclusion, in the present study, the expression level of XRCC2 was notably higher in tumor samples than in adjacent healthy tissues, suggesting an oncogenic role for XRCC2 in BC development. Discrepancies in the literature regarding the role of XRCC2 in BC highlight the complexity of its function, as some studies report a decrease in its expression. However, conflicting data and variability in study designs necessitate further research to fully elucidate the role of XRCC2 in BC carcinogenesis.

Recommendations

Further investigations are recommended to elucidate the inconsistent findings regarding XRCC2 expression in BC across different studies, with particular attention to cancer grade and type. Larger, more diverse cohorts should be included in subsequent research to validate the findings and account for variability in gene expression due to cancer grade, type, and patient demographics. Additionally, functional studies are warranted to understand the mechanistic role of XRCC2 in tumor development and progression.

Scientific Ethics Declaration

The authors declare that the scientific ethical and legal responsibility of this article published in EPHELS journal belongs to the authors.

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