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Investigation of C609T polymorphism in the *NQO1* gene in patients diagnosed with colorectal cancer in the Azerbaijani population

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This study aimed to determine the clinical significance of the *NQO1* C609T (rs1800566, Pro187Ser) polymorphism in patients with colorectal cancer (CRC) in the Azerbaijani population. 142 patients with CRC and 146 healthy individuals were included in the study. DNA from blood was isolated using the salting-out method, and genotypes were determined on agarose gel using the PCR-RFLP method. When comparing the patients and control groups, heterozygous CT (OR=1.813; 95%CI=1.097–2.995, P=0.020), under the dominant model (OR=1.842; 95%CI=1.137–2.983, P=0.013), and the mutant T allele (OR=1.644; 95%CI=1.096–2.465, P=0.016) were statistically associated with an increased risk of CRC. When comparing male patients with healthy men, heterozygous CT was associated with a higher risk of CRC (OR=2.219; 95%CI=1.079-4.565, P=0.029). However, the age, pathological tumor grade and stage, smoking, and alcohol consumption of the study groups were compared and no significant relationship was found (P>0.05). Our findings showed that the *NQO1* C609T polymorphism is related to the risk of colorectal cancer in the Azerbaijan population.

***Keywords:** Colorectal cancer, polymorphism, PCR-RFLP, NQO1, C609T*

INTRODUCTION

Colorectal cancer (CRC) is the most common cause of cancer death worldwide in both men and women (Siegel et al., 2018). Early detection and excision of precancerous lesions with screening programs have been associated with a reduced risk of CRC in different populations (Holme et al., 2013; Welch and Robertson, 2016). Genetic profiling of tumors and detection of somatic genetic variants are necessary and provide opportunities for effective treatment choices (Biller and Schrag, 2021). Furthermore, the underlying genetic causes of CRC include Chromosome instability (CIN) and Microsatellite instability (MSI). CIN phenotype includes structural abnormalities in chromosomes (copy number alterations, duplication, amplification, deletion, insertion, loss of heterozygosity) and chromosomal number changes such as aneuploidy which is present in 65-70% of all CRC cases (Nguyen et al., 2020). Microsatellites are DNA sequences containing repetitive motifs that tend to accumulate high mutation rates. MSI inactivates DNA repair system genes, including *MLH1*, *MSH2, MSH3, MSH6*, and *PMS2* (Li, 2008).

NQO1 is a multifunctional antioxidant encoding gene and plays an essential role in protecting cells from oxidative stress. NQO1 acts as an anti-cancer enzyme. In some cancers (breast, colon, lung cancer, etc.), the expression of the *NQO1* gene is increased (Yadav et al., 2018). *NQO1* is a gene of approximately 20 kb in length, consisting of 6 exons on chromosome 16q22.1 (Iskander and Jaiswal, 2005). Several polymorphisms have been identified in the *NQO1* gene (Nebert et al., 2002). However, the most studied and clinically significant is polymorphism C609T (rs1800566, P187S), which converts proline amino acid to serine in exon 6 (Yadav et

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al., 2018). *NQO1* C609T polymorphism has been widely investigated among different ethnic groups for CRC susceptibility, but the results are inconsistent.

Several different clinical studies have been performed on the *NQO1* gene C609 polymorphism, indicating that the TT genotype has an increased risk of disease in patients with kidney, urothelial, esophageal, bladder, breast and stomach cancers, pediatric and adult leukemia compared to healthy controls (Sameer et al., 2010). There are no consistent data available to show the association between *NQO1* gene polymorphism and CRC risk in our population. In this case-control study, we investigated for the first time the association between *NQO1* C609T (rs1800566, Pro187Ser) gene polymorphisms and subjects' age, gender, clinicopathological parameters, smoking and alcohol use parameters, and CRC susceptibility in the Azerbaijani population.

MATERIALS AND METHODS

This study included 142 patients diagnosed with colorectal cancer at the Azerbaijan Medical University and the Scientific Surgery Center named after M. Topchubashov and 146 healthy people as a control group. The control group was selected from individuals with no history of inflammatory bowel disease or cancer. Cancer biopsy samples taken from patients were evaluated due to pathohistological analysis, and the stage and grade of the tumor were determined. Venous blood was taken from the study groups in tubes with EDTA. DNA extraction was carried out in the Laboratory of Human Genetics of ANAS Institute of Genetic Resources. DNA extraction from blood samples was performed according to the protocol by the *Salting-out* method. Quantitative and qualitative parameters of DNA were measured by the spectrometric method (Thermo Scientific™ NanoDrop™ 2000/2000c). Then the amplification process was performed by PCR. The sequence of specific primers used in PCR follows forward 5'-AAGCCCAGACCAACTTCT-3', reverse 5'-TCTCCTCATCCTGTACCTCT-3. The PCR reaction with a total volume of 20 µl consisted of the following: 2µl DNA (200 ng/µl), 2 µl 10X buffer [10 mMTris–HCl pH 8.0, 50 mM KCl], 2 µl $MgCl_2$, dNTP mixture 0.2 µl (20 mM), 0.5 µl (100 µM) and 0.2 µl (5 U/µl) from each primer, 0.2 µl (5 U/µl) Taq DNA polymerase (Solis BioDyne, Tartu, Estonia) and 12.6 µl distilled water (dH_2O). The amplification condition for PCR (Applied Biosystems, USA) consisted of 5 minutes at 95ºC, 35 cycles of 30 seconds at 95ºC, 45 seconds at 59ºC and 2 minutes at 72ºC, then final elongation of 7 minutes at 72ºC. *HinfI* (NEB, New England Biolabs) restriction enzymes were used to identify *NQO1* C609T genotypes. Restriction fragments were visualized on 2% agarose gel stained with Ethidium Bromide under the UV gel documentation system. As a result of digestion of PCR products with the restriction enzyme, wild type CC 271 bp, heterozygous CT 271 bp, 151 bp and 120 bp, and homozygous mutant TT genotype 151 bp and 120 bp (Figure).

Statistical analysis. Statistical analysis using *SPSS* version 17.0 (SPSS Inc, Chicago, USA) Pearson's chi-squared test was used for genotype and allele frequency comparison. Odds ratios (OR) and confidence intervals (CI) were calculated to estimate relative risk. A *P* value less than 0.05 was considered statistically significant.

RESULTS AND DISCUSSION

The study involved 142 patients with colon cancer and 146 practically healthy individual control groups. Demographic parameters for patients and controls are shown in Table 1. The patient group consisted of 84 men and 58 women, and the control group consisted of 64 men and 82 women, respectively. The age range of patients was 25-85, and in the control group, it was 27-82 years. No statistical association was observed when the study groups were compared by age (P=0.256). Furthermore, 8.2% of patients had G1, 91% G2 and 24.7% G3 tumors, and 2.1% T1, 11.3% T2 and 78.9% T3, and 7.7% had a tumor stage. In addition, no significant statistical difference was observed when comparing research groups in terms of data on smoking and alcohol consumption (P>0.05).

The genotype and allele frequencies of the C609T polymorphism of the *NQO1* gene are shown in Table 2. Genotypic frequencies were found in 54.9% CC, 39.5% CT and 5.6% TT in patients, and 69.2% CC, 27.4% CT and 3.4% TT in the control group, respectively. Heterozygous

CT and homozygous mutant TT were higher in patients. A statistical association was found between heterozygous CT and increased risk of colon cancer (OR=1.813; 95% CI=1.097–2.995; P=0.020). Although the association was found in the dominant model (OR=1.842; 95% CI=1.137–2.983; P=0.013), no association was found in the recessive model (P>0.05). In addition, 74.6% of patients had normal C allele, and 25.4% had mutant T allele. 82.9% had a wild type C allele in the control group, and 17.1% had a mutant T allele. A statistical association was observed between the mutant T allele (OR=1.644; 95% CI=1.096–2.465; P=0.016) and the risk of colorectal cancer.

Table 1. Clinical and demographic parameters of study groups

	Patients N=142 (%)	**Controls N=146 (%)**	***P* value**
Gender			
Male	84 (59.2)	64 (43.8)	0.256
Female	58 (40.8)	82 (56.2)	
Age			
Age interval	25-85	27-82	
Mean	61±9.1	60±10.3	
Histological Grade			
G1	13 (8.2)		
G2	91 (67.1)		
G3	38 (24.7)		
Tumor Stage			
T1	3 (2.1)		
T2	16 (11.3)		
T3	112 (78.9)		
T4	11 (7.7)		
Smoking Status			
Smokers	49 (34.4)	51 (34.9)	0.942
Non-Smokers	85 (60)	89 (60.9)	
Unknown	8 (5.6)	6 (4.2)	
Alcohol consumption			
Yes	45 (31.7)	46 (31.5)	0.368
No	89 (62.7)	94 (64.4)	
Unknown	8 (5.6)	6 (4.1)	

When comparing genotypes by gender factor (Table 3), 53.6% of wild-type CC, 40.5% of heterozygous CT, and 5.9% of mutant TT were found in male patients. In healthy men, the genotypic frequencies were 73.4% CC, 25% CT and 1.6% TT, respectively. A statistically significant association was found between heterozygous CT genotype and increased risk of colorectal cancer among men included in the study (OR=2.219; 95%CI=1.079-4.565; P=0.029). Moreover, genotypic frequencies in female patients were 56.9% CC, 37.9% heterozygous CT, and 5.2% mutant TT, respectively. It was determined as 65.9% CC, 29.3% CT, and 4.8% TT in healthy women. Although both CT and TT genotypes were more common in female patients, no statistical difference was found between genotypes and disease risk (P<0.05).

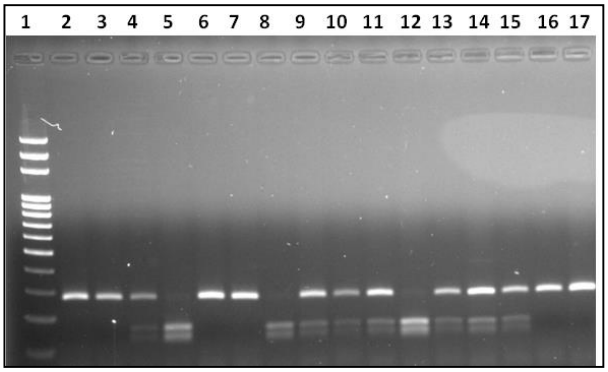

Fig. Genotypes of *NQO1* C609T polymorphism determined by PCR-RFLP methods in agarose gel **DNA Ladder (100 bp):** Lane-1; **Wild type CC:** Lane-2, 3, 6, 7, 16, 17; **Heterozygous CT:** Lane-4, 5, 9, 10, 11, 13, 14, 15; **Homozygous mutant TT:** Lane-5, 8, 12.

The distribution of genotypes according to the average age in the study groups is presented in Table 4. The distribution of genotypes in patients under 60 years of age was determined by 57.7% CC, 36.5% CT and 5.8% TT, and in the control group, 68.9% CC, 28.3% CT and 2.8% TT, respectively. Although heterozygous CT (OR=1.541; 95%CI=0.754-3.150; P=0.234) and mutant TT (OR=2.433; 95%CI=0.465-4.514; P=0.364) genotypes were more common in patients compared with the control group. A statistical relationship between genotypes and disease risk was not found when comparing patients under 60 with the control group. However, in patients over 60 years of age, normal CC, heterozygous CT, and mutant TT genotypes were 53.3%, 41.1%, and 5.6%, while these frequencies were 70%, 25%, and 5% in the controls, respectively. Both CT (OR=2.158; 95%CI=0.932-4.998; P=0.069) and TT (OR=1.458; 95%CI=0.265-3.021; P=0.663) genotypes are more common in patients over 60 years of age. No statistically significant results were obtained between genotypes and disease risk when comparing study groups over 60 years of age (P>0.05).

Genotypic frequencies were found in non-smokers compared to non-smokers (Table 5), with 27.3% CC, 52.7% CT and 20% TT in smokers. In non-smokers, the distribution of genotypes was 38.9% CC, 42.2% CT and 18.9% TT, respectively. Although heterozygous CT (OR=0.952; 95%CI=0.460-1.972; P=0.895) and mutant TT (OR=0.667; 95%CI=0.121-3.678; P=0.642) are more common in smokers, this difference was not statistically significant. Additionally, comparing genotypes in terms of alcohol use, heterozygous CT (49%) and homozygous TT (20.4%) were more prevalent in alcohol users. However, no association was found between genotypes CT (OR=1.619; 95%CI=0.772-3.397; P=0.201) and TT (OR=0.971; 95%CI=0.175-5.407; P=0.974) and disease risk.

The distribution of genotype frequencies in tumor grade and the stage is presented in Table 6. Both heterozygous CT genotype (47.4%) and mutant TT genotype (7.9%) were prevalent in malignant tumor G3. The heterozygous CT genotype was more common in the T3 and T4 stages of the tumor, and the mutant TT genotype was more common in T3. No statistical correlation was found between tumor grade and stages and C609T polymorphism (P>0.05).

Table 2. Distribution of genotype and allele frequencies of C609T polymorphism of *NQO1* gene in subject groups

	Patients (N=142 (%))	**Controls (N=146 (%))**	**OR (95%CI)**	***P* value**
Genotypes				
CC	78 (54.9)	101 (69.2)	1	-
CT	56 (39.5)	40 (27.4)	1.813 (1.097-2.995)	**0.020**
TT	8 (5.6)	5 (3.4)	2.072 (0.652-6.581)	0.209
Dominant				
CC	78 (54.9)	101 (69.2)	1	-
CT+TT	64 (45.1)	45 (30.8)	1.842 (1.137-2.983)	**0.013**
Recessive				
CC+CT	134 (94.4)	141 (96.6)	1	-
TT	8 (5.6)	5 (3.4)	1.684 (0.537-5.275)	0.367
Allele				
C	212 (74.6)	242 (82.9)	1	-
T	72 (25.4)	50 (17.1)	1.644 (1.096-2.465)	**0.016**

Table 3. Distribution of genotype frequencies between men and women

Males	**Patients (N=84 (%))**	**Controls (N=64 (%))**	**OR (95%CI)**	***P* value**
Genotypes				
CC	45 (53.6)	47 (73.4)	1	-
CT	34 (40.5)	16 (25)	2.219 (1.079-4.565)	**0.029**
TT	5 (5.9)	1 (1.6)	2.315 (0.587-3.396)	0.205
Females	**N=58** (%)	**N=82** (%)		
Genotypes				
CC	33 (56.9)	54 (65.9)	1	-
CT	22 (37.9)	24 (29.3)	1.500 (0.728-3.090)	0.270
TT	3 (5.2)	4 (4.8)	1.227 (0.258-5.831)	0.798

Table 4. Distribution of genotype frequencies by age factor

Age, ≤ 60	**Patients (N=52 (%))**	**Controls (N=106 (%))**	**OR (95%CI)**	***P* value**
CC	30 (57.7)	73 (68.9)	1	-
CT	19 (36.5)	30 (28.3)	1.541 (0.754-3.150)	0.234
TT	3 (5.8)	3 (2.8)	2.433 (0.465-4.514)	0.364
>60	N=90 (%)	N=40 (%)		
CC	48 (53.3)	28 (70)	1	-
CT	37 (41.1)	10 (25)	2.158 (0.932-4.998)	0.069
TT	5 (5.6)	2 (5)	1.458 (0.265-3.021)	0.663

Table 5. Comparison of genotype frequencies in terms of smoking and alcohol use

Genotypes	Smokers (N=49 (%))	Non-smokers (N=85 (%))	OR (95%CI)	*P* value
CC	27 (27.3)	45 (38.9)	1	-
CT	20 (52.7)	35 (42.2)	0.952 (0.460-1.972)	0.895
TT	2 (20)	5 (18.9)	0.667 (0.121-3.678)	0.642
	Drinkers (N=45)	**Non-drinkers (N=89)**		
CC	21(30.6)	51 (36.5)	1	-
CT	22 (49)	33 (44.7)	1.619 (0.772-3.397)	0.201
TT	2 (20.4)	5 (18.8)	0.971 (0.175-5.407)	0.974

Table 6. Distribution of genotype frequencies in the degree and stage of the tumor

Tumor Grade	CC (N (%))	CT (N (%))	TT (N (%))	*P* value
G1	7 (53.8)	6 (46.2)	0	0.397
G2	54 (59.3)	32 (35.2)	5 (5.5)	
G3	17 (44.7)	18 (47.4)	3 (7.9)	
Tumor Stage				
T1	0	3	0	0.201
T2	10 (62.5)	6 (37.5)	0	
T3	61 (54.5)	43 (38.4)	8 (7.1)	
T4	4 (36.4)	7 (63.6)	0	

In summary, in our study, we investigated the C609T polymorphism of the *NQO1* gene in patients diagnosed with colorectal cancer and in the control group. We determined the relationship between polymorphism and disease risk and clinical parameters. Thus, there was a statistically significant association between heterozygous CT, dominant model (CC/CT+TT) and mutant T allele and colorectal cancer risk in our study. Similar findings supporting our findings were reported in a study presented by Wang and colleagues. Researchers have provided strong evidence for an association between the *NQO1* gene C609T polymorphism and the risk of colorectal cancer. They have shown that the mutant T allele is a significant risk factor for disease (Wang et al., 2013). Another study found that the T allele was significantly associated with susceptibility to colorectal cancer in both Asians and Caucasians. There was also a positive association between *NQO1* C609T polymorphism and susceptibility to disease in smokers compared to non-smokers (Zheng et al., 2014). In our study, no association was found between genotypes and disease risk compared to smokers and non-smokers. A meta-analysis of 12 studies that included 5,525 patients and 6,272 healthy individuals reported that the mutant T allele was a high-risk factor for colorectal cancer and received a positive statistical correlation for the dominant model (Ding et al., 2012). A meta-analysis of another Asian population showed a clear association between *NQO1* Pro187Ser polymorphism and the risk of colorectal cancer (Zhao et al., 2014). In a study by Yadav and colleagues, C609T polymorphism in the *NQO1* gene was found to increase the risk of colorectal cancer and other parts of the digestive tract, especially gastric cancer (Yadav et al., 2018). In addition, no association was observed in our study between *NQO1* gene polymorphism and disease risk in patients who smoked and consumed alcohol. A study by Peng and colleagues found that *NQO1* 609 C>T polymorphism increased the risk of colorectal cancer in the Chinese population, especially in patients who smoked and drank alcohol (Peng et al., 2013). A 2010 study of the Kashmir population also found an association between *NQO1* C609T polymorphism and the risk of developing colorectal cancer and a statistical relationship between smoking, age, cancer stage, and degree of homozygous mutant genotype (Sameer et al., 2010). In comparison with this study, our study did not find a statistically significant relationship between cancer stage and degree, age factor, smoking and alcohol use factors, and *NQO1* C609T polymorphism.

In summary, in our case-control study, heterozygous CT and mutant T alleles were associated with an increased risk of CRC in our population. In particular, a statistically significant association was found between the *NQO1* CT genotype and in-

creased CRC risk when male patients were compared to healthy men. Thus, our results emphasize that the *NQO1* gene C609T polymorphism may play an essential role in the molecular pathogenesis of colorectal cancer and suggest that it may be a genetic marker in early detection.

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Azərbaycan populyasiyasında kolorektal xərçəng diaqnozu qoyulan xəstələrdə *NQO1* genində C609T polimorfizminin tədqiqi

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Bu tədqiqatın əsas məqsədi kolorektal xərçəng (KRX) diaqnozu qoyulan xəstələrdə *NQO1* geni C609T (rs1800566, Pro187Ser) polimorfizminin klinik əhəmiyyətini tədqiq etməkdir. Tədqiqat işinə 142 KRX diaqnozu qoyulan xəstə və 146 sağlam insan daxil edilmişdir. Qandan DNT-nin ekstraksiyası *Salting-out* üsulu ilə həyata keçirilmiş, genotiplər isə PZR-RFLP üsulu və aqaroz gel elektroforezi vasitəsilə müəyyən edilmişdir. Xəstə və nəzarət qruplarını müqayisə etdikdə heteroziqot CT (OR=1,813; 95% CI=1,097–2,995, P=0,020), dominant model (OR=1,842; 95% CI=1,137–2,983, P=0,013) və mutant T alleli (OR=1,644; 95% CI=1,096–2,465, P=0,016) ilə artmış KRX riski arasında statistik əhəmiyyətli asossasiya aşkar edildi. Həmçinin kişi xəstələri sağlam kişilərlə müqayisə etdikdə, heteroziqot CT (OR=2,219; 95% CI=1,079-4,565, P=0,029) yüksək KRX riski arasında statistik əlaqə müəyyən edildi. Bununla belə, tədqiqat qruplarının yaşı, patoloji şiş dərəcəsi və mərhələsi, siqaret və spirtli içki qəbulu müqayisə edildikdə əhəmiyyətli statistik əlaqə aşkar edilməmişdir (P>0,05). Nəticələrimiz *NQO1* C609T polimorfizminin Azərbaycan populyasiyasında kolorektal xərçəng riski ilə əlaqəli olduğunu göstərdi.

Açar sözlər: *Kolorektal xərçəng, polimorfism, PZR-RFLP, NQO1, C609T*

Исследование полиморфизма C609T гена *NQO1* у больных с диагнозом колоректальный рак в популяции азербайджана

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Основная цель настоящего исследования состояла в том, чтобы определить клиническую значимость полиморфизма C609T гена NQO1 (rs1800566, Pro187Ser) у пациентов с колоректальным раком (КРР). В исследование были включены 142 пациента с КРР и 146 здоровых лиц. ДНК из крови выделяли методом Salting-out, а генотипы определяли на агарозном геле методом ПЦР-ПДРФ. При сравнении пациентов и контрольных групп гетерозиготная CT (OR=1,813; 95%CI=1,097-2,995, P=0,020), доминантная модель (OR=1,842; 95%CI=1,137-2,983, P=0,013) и мутантная T аллель (OR=1,644; 95% CI=1,096-2,465, P=0,016) были статистически связаны с повышенным риском КРР. При сравнении пациентов мужского пола со здоровыми мужчинами гетерозиготная CT была связана с более высоким риском КРР (OR=2,219; 95% CI=1,079-4,565, P=0,029). Однако при сравнении возраста, степени и стадии патологической опухоли, курения и употребления алкоголя в исследуемых группах достоверной статистической связи (P>0,05) выявлено не было. Результаты исследования позволили сделать вывод о том, что риск развития колоректального рака у населения Азербайджана связан с полиморфизмом C609T гена *NQO1*.

Ключевые слова: *Колоректальный рак, полиморфизм, ПЦР-ПДРФ, NQO1, C609T*