ISSN: 3029-0910

Research Article

Journal of Gynecological & Obstetrical Research

Association of Cyp1a1 Rs1048943 Polymorphism with Male Infertility: A Study in East Azerbaijan, Iran

Ronaz Mostafaei[1], Masoud Maleki[1] and Omid Pourbagherian[2*]

[1]*Department of Biology, Tabriz Branch, Islamic Azad University, Tabriz, Iran*

[2]*Immunology Research Center, Tabriz University of Medical Sciences, Tabriz, Iran*

[*]**Corresponding author**

Omid Pourbagherian, Immunology Research Center, Tabriz University of Medical Sciences, Tabriz, Iran.

Received: December 11, 2025; **Accepted:** December 22, 2025; **Published:** December 29, 2025

ABSTRACT

Introduction: Infertility is a disease of the reproductive system that is defined as non-pregnancy after 46 months of regular sexual intercourse without the use of contraceptive methods. Male infertility occurs in approximately 45% of couples. Various environmental and genetic factors play a role in male infertility. Among the genes involved in male infertility is the CYP1A1 gene, whose various polymorphisms are involved in infertility, including asthenospermia, which is one of the most important polymorphisms related to this polymorphic gene RS1048943. In this study, these polymorphisms in infertile men in northwestern Iran have been investigated.

Methods: For this purpose, sperm samples were prepared from Tabriz University Jihad Center and then DNA extraction was performed. Among the extracted DNAs, each sample of the desired quality was stored for other steps. Tetra ARMS PCR was used to study these polymorphisms. To perform PCR using primers designed for this gene, the desired region was amplified and after running on the gel, the genotypes were determined based on the obtained bands and statistical analyzes were performed using Excell software. $P< 0.05$ was significant.

Results: According to statistical studies, the percent of TT, AA and AT genotypes in the patient group was 52%, 4% and 44%, respectively, and the percent of genotypes in the control group was 32%, 52% and 16%, respectively.

Discussion: According to the results, genotypic frequency in the patient and control groups was insignificant and also the amount of allelic frequency in the patient and control groups was also insignificant.

Keywords: Male Infertility, CYP1A1 Gene, Rs1048943, Polymorphism

Introduction

Infertility is a major global health issue, affecting approximately 15% of couples worldwide who are trying to conceive. Male infertility is a contributing factor in up to 20% of cases directly and accounts for an additional 30-40% of infertility cases when combined with female factors [1]. This makes male infertility a significant public health challenge, especially given its increasing prevalence due to declining sperm counts and quality observed in industrialized nations [2]. Currently, 5-7% of the global male population experiences infertility, a figure projected to rise due to environmental exposures, lifestyle factors, and underlying health conditions. Despite remarkable advances in understanding human reproductive physiology, the primary cause of male infertility remains undetermined in approximately 50% of cases, often categorized as idiopathic infertility [3].

Idiopathic male infertility, which accounts for a significant proportion of unexplained reproductive failure, is widely believed to have a strong genetic foundation [4]. Spermatogenesis, the highly complex and tightly regulated process of sperm cell production, is governed by the coordinated expression of over 1,000 genes [5]. These genes regulate critical stages, including germ cell proliferation, meiosis, and differentiation, emphasizing

Citation: Ronaz Mostafaei1, Masoud Maleki, Omid Pourbagherian. Association of Cyp1a1 Rs1048943 Polymorphism with Male Infertility: A Study in East Azerbaijan, Iran. Open Access J Gyneco Obstet Res. 2025. 3(4): 1-6. DOI: doi.org/10.61440/JGOR.2025.v3.59

the potential for genetic involvement in unexplained infertility cases. Despite this, much of the genetic landscape contributing to male infertility remains undiscovered [6].

A few key genes have been definitively associated with specific forms of male infertility. For example, mutations in the CFTR gene are linked to congenital absence of the vas deferens, a condition commonly observed in men with cystic fibrosis [7]. Similarly, mutations in the androgen receptor (AR) gene can lead to androgen insensitivity syndrome, which disrupts spermatogenesis due to hormonal signaling deficiencies. However, these known genetic causes represent only a small fraction of the genetic underpinnings of male infertility [8].

The cytochrome P450 (CYP) family of enzymes plays a crucial role in the metabolism of a wide array of endogenous and exogenous compounds, including hormones, xenobiotics, and environmental pollutants. These enzymes are involved in the phase I metabolic pathway, where they catalyze oxidation reactions, making hydrophobic compounds more water-soluble and, thus, more readily excreted. Among the CYP family, the CYP1A1 gene stands out due to its critical involvement in the biotransformation of polycyclic aromatic hydrocarbons (PAHs), a class of environmental pollutants commonly found in tobacco smoke, industrial emissions, and charred foods [9].

Located on chromosome 15q22-q24, the CYP1A1 gene encodes an enzyme that activates PAHs into their reactive intermediates. While this metabolic activity is essential for detoxification, it paradoxically also generates reactive oxygen species (ROS) and electrophilic intermediates, which can bind to DNA and proteins, causing cellular damage [10]. These reactive byproducts are particularly harmful to cells undergoing rapid division and differentiation, such as those involved in spermatogenesis. This process is highly sensitive to oxidative stress and DNA damage, both of which can impair the production of viable, motile sperm, contributing to male infertility.

One of the most studied genetic variations in the CYP1A1 gene is the rs1048943 polymorphism [11]. This single nucleotide polymorphism (SNP), characterized by an A→G substitution in exon 7, results in an amino acid change from isoleucine to valine at codon 462. This structural alteration significantly impacts the enzyme's function by increasing its metabolic activity. The heightened enzymatic activity enhances the bioactivation of PAHs, leading to an elevated generation of ROS. Although ROS play a physiological role at low levels, excessive production overwhelms the antioxidant defense mechanisms in the testes, resulting in oxidative stress [12].

Oxidative stress is a major contributor to male infertility, as it damages sperm DNA, proteins, and membranes, reducing sperm motility and viability [13]. ROS can disrupt the integrity of the sperm cell membrane, which is rich in polyunsaturated fatty acids and, thus, particularly susceptible to lipid peroxidation. Furthermore, ROS-induced DNA fragmentation in sperm cells can lead to impaired fertilization and embryonic development, increasing the risk of miscarriage. Studies have shown that individuals carrying the polymorphic variant of rs1048943 have higher levels of PAH-DNA adducts, a marker of oxidative DNA damage, in their reproductive tissues, further linking this SNP to adverse reproductive outcomes [14].

The prevalence of the rs1048943 polymorphism varies across populations [15]. It is notably higher in Asian populations, where it has been extensively studied in the context of various cancers and other diseases associated with oxidative stress. In these populations, the polymorphism has also been linked to altered reproductive outcomes, including impaired spermatogenesis and reduced sperm quality. However, the relationship between rs1048943 and male infertility remains underexplored in other populations, including those in the Middle East, where genetic and environmental factors may interact uniquely [11].

Given its potential role as a genetic susceptibility factor, the rs1048943 polymorphism represents an important area of research in the context of male infertility [16]. Investigating its prevalence and impact can provide insights into the molecular mechanisms underlying infertility and pave the way for more targeted approaches to diagnosis and treatment, particularly in regions with high exposure to environmental toxins. By understanding the interactions between genetic predispositions like CYP1A1 polymorphisms and environmental exposures, researchers can better address the growing public health challenge of infertility [17,18].

Several studies have investigated the potential association between CYP1A1 polymorphisms and male infertility, yielding mixed results. Some research suggests that individuals carrying the polymorphic variant of rs1048943 may be at a higher risk for infertility due to increased oxidative stress and impaired spermatogenesis [19]. However, other studies have reported no significant association, indicating that environmental and genetic interactions may play a critical role in determining susceptibility [20]. The prevalence of the rs1048943 polymorphism varies significantly across populations, with higher frequencies reported in Asian populations compared to Western populations [21]. This highlights the importance of population-specific studies to better understand the genetic basis of male infertility.

East Azerbaijan, located in Northwest Iran, represents a unique population with diverse genetic backgrounds and environmental exposures. Despite the high burden of male infertility in this region, limited studies have explored the genetic factors contributing to this condition. By investigating the rs1048943 polymorphism of the CYP1A1 gene in infertile men from this population, this study aims to shed light on its potential role as a genetic risk factor for male infertility. Using a case-control design, we compare the genotypic and allelic frequencies of this polymorphism between infertile and fertile men, providing valuable insights into its association with reproductive health outcomes.

The findings from this study will contribute to the growing body of evidence on the genetic and environmental determinants of male infertility. Understanding the role of CYP1A1 polymorphisms in infertility could pave the way for targeted interventions, such as genetic screening, lifestyle modifications, and personalized treatment strategies. Furthermore, this research underscores the importance of investigating gene-environment interactions in reproductive health, particularly in regions with unique environmental and genetic profiles.

Materials and Methods

Sample Collection

This study was conducted using sperm samples obtained from the Infertility Center at Jahad Daneshgahi, Northwest Iran. Fifty infertile men were recruited as the case group. Control samples were collected from fertile men with confirmed fertility, as validated by the infertility center. The fertile participants had no history of infertility-related conditions, and their semen analysis indicated normal parameters.

DNA Extraction

DNA was extracted from sperm samples using the FAVERGEN kit. After centrifugation, the pellet was treated with TBE buffer, proteinase K, FABG buffer, and ethanol. The solution was passed through a mini-column, washed with W1 and Wash buffers, and dried via centrifugation. Finally, DNA was eluted with preheated elution buffer and stored at -20°C for further analysis.

Assessment of DNA Quality and Quantity

The quality and quantity of the extracted DNA were evaluated using spectrophotometry and agarose gel electrophoresis. The absence of smearing and contamination confirmed the suitability of the DNA for subsequent analysis.

Primer Design

Specific primers for Tetra-ARMS PCR were designed to target the rs1048943 polymorphism in the CYP1A1 gene. Primer design was performed using OLIGO7 and GENERUNNER software to ensure specificity and complementarity to the target DNA sequence. Primer binding specificity was validated through the NCBI Primer-BLAST tool. The sequences and annealing temperatures of the primers used in the study are shown in [Table 1].

Table 1: Primer's Sequence

Gene	Primer Type	Sequence	Annealing Temp (°C)
CYP1A1	Forward In Primer	TCCCAGCGGGCAATGGAC	62.42
	Reverse In Primer	CATGGGCAAGCGGAAGTGTATC	61.89
	Forward Out Primer	GTCATGTCCACCTTCACGCC	61.30
	Reverse Out Primer	GTCAACCCATCTGAGTTCCTACCT	61.61

Genotyping

The rs1048943 polymorphism was analyzed using the Tetra-ARMS PCR method, which involves four primers to detect single nucleotide polymorphisms (SNPs). PCR was carried out in a thermocycler, and the products were separated via agarose gel electrophoresis. The genotypes were identified based on specific banding patterns observed under UV illumination.

Statistical Analysis

Data analysis was performed using SPSS software. Genotypic and allelic frequencies were compared between the case and control groups using appropriate statistical tests. A p-value < 0.05 was considered statistically significant. The results were interpreted to determine any association between the rs1048943 polymorphism and male infertility.

Results

Statistical Analysis of Genotypes

Based on the analyzed data, in the patient group, 52% had the homozygous TT genotype, 44% had the heterozygous TA genotype, and 4% had the homozygous AA genotype. In the control group, 32% had the homozygous TT genotype, 52% had the heterozygous TA genotype, and 16% had the homozygous AA genotype. According to this data, no significant difference was observed between the control and patient groups (p>0.05) (Table 2).

Table 2: Genotypic Frequency in Patient and Control Groups

dbSNP	Genotypes/allele	Case(n=25)	Control(n=25)	OR (95%CI)	p-value
Codominant	TT	13(52%)	8(32%)	Ref	-
	TA	11(44%)	13(52%)	1.920(0.583-6.324)	0.281
	AA	1(4%)	4(16%)	6.500(0.613-68-957)	0.091
Dominant	TT	13(52%)	8(32%)	Ref	0.152
	TA+AA	12(48%)	17(68%)	2.302(0.729-7.268)	
Recessive	TT+TA	24(96%)	21(84%)	Ref	0.157
	AA	1(4%)	4(16%)	4.571(0.473-44.170)	
Overdominant	TT+AA	14(56%)	12(48%)	Ref	0.571
	TA	11(44%)	13(52%)	1.379(0.453-4.197)	

Chi-Square Test for Genotypic Analysis

The chi-square test results for genotypic frequency between the control and patient groups showed a p-value greater than 0.05, indicating no significant difference between the two groups (Table 3).

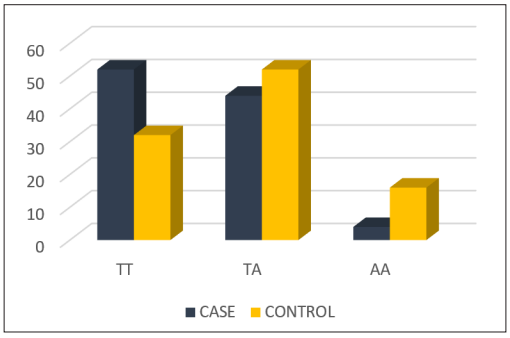

Chart 1: Genotypic Frequency Percentages in Patient and Control Groups

Table 3: Chi-Square Test Results for Genotypic Frequency

	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	3.1571	2	0.20627

Allelic Frequency in Control and Patient Groups

Based on the analyzed data, the patient group showed 74% allele A and 26% allele T, while the control group showed 58% allele A and 42% allele T. The p-value for allelic frequency was greater than 0.05, indicating no significant difference in allelic frequency between the control and patient groups (Table 4).

Table 4: Allelic Frequency in Patient and Control Groups

dbSNP	Allele	Case (n=50)	Control (n=50)	OR (95%CI)	p-value
ALLELE	A	37(74%)	29(58%)	Ref	0.091
	T	13(26%)	21(42%)	2.061 (0.885-4.800)	

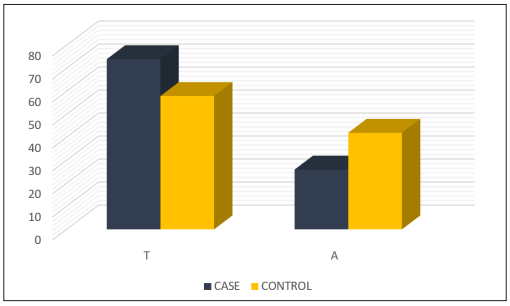

Chart 2: Allelic Frequency Percentages in Patient and Control Groups

Chi-Square Test Results for Allelic Frequency

The Pearson chi-square test for Hardy-Weinberg equilibrium yielded a p-value greater than 0.05, indicating no significant difference in allelic frequency between the two groups (Table 5).

Table 5: Chi-Square Test Results for Allelic Frequency

	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	2.852	2	0.091258

Results of Hardy-Weinberg Equilibrium Analysis

The comparison of observed genotypic frequencies between the patient and control groups with the expected frequencies showed a p-value of 0.7732, which is greater than 0.05, indicating that Hardy-Weinberg equilibrium is maintained.

Expected Frequency (%)	Observed Frequency	Observed Frequency (%)	Genotype	Expected Frequency
14	52	13	TT	56
10	44	11	TA	40
1	4	1	AA	4
25	100	25	Total	100

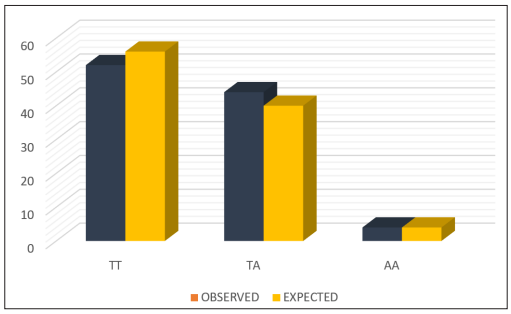

Chart 3: Observed and Expected Genotypic Frequency Percentages

Discussion

Infertility is defined as the inability to achieve pregnancy after 12 months of unprotected intercourse, affecting 10–15% of couples in the United States [21]. Male infertility accounts for approximately 30–55% of all infertility cases. Azoospermia, the absence of sperm in the ejaculate, constitutes 10–15% of male infertility cases and affects about 1% of the male population [22].

Among the critical mechanisms explored in genetic studies is the role of cytochrome P4501A1 (CYP1A1), which plays a significant role in Phase I metabolism of polycyclic aromatic hydrocarbons (PAHs) into biologically active intermediates [23]. These intermediates can potentially impact male fertility. CYP1A1 is involved in metabolizing substrates through the catalysis of β-estradiol hydroxylation at the C-2 position. PAH metabolites can form DNA adducts, which in sperm cells may lead to severe DNA damage and disruptions in meiosis during spermatogenesis. This has been linked to male infertility [24].

Recent studies suggest that CYP1A12C genetic polymorphisms, which influence xenobiotic metabolism, might play a crucial role in male infertility. The rs1048943 CYP1A12C polymorphism involves an A-to-T substitution at nucleotide 2455, resulting in an amino acid change from isoleucine to valine at codon 462 in exon 7 [25] . This polymorphism is more common in Asian populations. Various studies highlight the importance of this polymorphism in influencing male reproductive health. CYP1A1 serves as a key enzyme in activating PAHs, which exhibit reproductive toxicity and are linked to male infertility risk [26].

Male reproductive functions can be affected by numerous environmental, physiological, and genetic factors. Most environmental factors are xenobiotics. These xenobiotics exert adverse effects via covalent interactions between intermediate metabolites and cellular macromolecules like DNA and proteins. These compounds are metabolized by CYP1A1, which can also induce enzyme activity. Apart from xenobiotic metabolism, CYP1A1 participates in testosterone inactivation, potentially influencing testicular function. However, the relationship between genetic variability in xenobiotic metabolism and male reproductive functions remains underexplored [27]. The interplay between environmental and genetic factors in infertility is not fully understood.

In this study, we examined the frequency of the CYP1A1 single nucleotide polymorphism in infertile men. The results showed no significant difference in the genotypic frequency of the rs1048943 polymorphism between infertile and fertile men. Similarly, there was no significant association between allelic frequency of this polymorphism and male infertility.

In contrast, Gudimella Tirumala Vani et al. investigated this polymorphism in infertile men in Yazd and found that individuals carrying the CYP1A1*2A CC allele had an increased risk of infertility [28]. These discrepancies could be attributed to differences in the studied populations and sample size.

Zakieh Javidan et al. reported an association between the CYP1A12A polymorphism and azoospermia, suggesting that this single nucleotide polymorphism may play a significant role in male infertility. They proposed that the CYP1A12A CC genotype could be recognized as an effective agent in azoospermia, although its precise function depends on interactions with other genetic and environmental factors. Their findings highlight the importance of further molecular studies to better understand the genetic mechanisms underlying male infertility, particularly at other genetic levels and in different populations [29].

Recently, the critical role of estrogen in male infertility has been highlighted. Estrogens are metabolized by CYP1A1, which converts them into catechol estrogens, such as 2-hydroxyestradiol and 4-hydroxyestradiol. CYP1A1 also plays a role in metabolizing xenobiotics and activating environmental toxins. A complex interplay exists between CYP1A1, estrogen receptor alpha, and the aryl hydrocarbon receptor, which exhibits anti-estrogenic properties [30]. CYP1A1 expression is induced by various endogenous and exogenous chemicals via the aryl hydrocarbon receptor. Additionally, CYP1A1 interacts with estrogen receptor alpha and the aryl hydrocarbon receptor to influence gene expression. Polymorphisms in CYP1A1 can alter enzyme activity and expression, potentially leading to reproductive disorders in men [31]. Furthermore, the association of CYP1A1 and estrogen polymorphisms with disruptions in spermatogenesis suggests that genetic and environmental factors play critical roles in testicular dysfunction, ultimately causing sperm damage, abnormal morphology, and male infertility.

Conclusion

While our findings do not support a significant role for the rs1048943 polymorphism in male infertility, they highlight the importance of population-specific studies. Further research, particularly large-scale and multi-ethnic studies, is needed to clarify the role of CYP1A1 polymorphisms and their interaction with environmental and physiological factors in male infertility. This will help bridge the gaps in understanding the complex relationship between genetic variability and reproductive health.

Acknowledgments

Authors extend their appreciation to the staff and researchers at the Infertility Center at Jahad Daneshgahi, Tabriz, for their cooperation and for providing the sperm samples used in this study.

Author contributions

All authors approved the final manuscript.

Funding

Not applicable

Competing interests

The authors declare that they have no competing interests

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