WESTERN caspian UNIVERSITY

e-ISSN 3107-1546
p-ISSN 3107-1538

JOURNAL OF MOLECULAR BIOSCIENCES AND ENGINEERING

Vol 1, № 3, 2026



Baku - 2026

Western Caspian University

e-ISSN 3107-1546
p-ISSN 3107-1538

Journal of Molecular Biosciences and Engineering
Vol 1, No 3, 2026

DOI: 10.54414/PJVJ2367

© Western Caspian University, 2026

https://jmbe.wcu.edu.az/

Editor-in-Chief:

Prof. Dr. Hussein Baghirov

Western Caspian University, Azerbaijan

Scopus Link Scholar Link

Editorial Board

Prof. Dr. Subash Gopinath

Universiti Malaysia Perlis, Malaysia (Scopus Link Scholar Link)

Prof. Dr. Adil M. Allahverdiyev

Scientific Research Institute of Medical Prevention named after V. Akhundov, Azerbaijan (Scopus Link Scholar Link)

Assoc. Prof. Hijaz Ahmad

Near East University, Cyprus (Scopus Link Scholar Link)

Assoc. Prof. Umar Saeed

Foundation University Islamabad, Pakistan (Scopus Link Scholar Link)

Assoc. Prof. Ghodrat Mahmoudi

University of Maragheh, Iran (Scopus Link Scholar Link)

Assoc. Prof. Zebo Bahodirovna

Urgench State Tashkent Medical Institute, Uzbekistan (Scopus Link Scholar Link)

Assoc. Prof. Saltanat Aghayeva

Western Caspian University, Azerbaijan (Scopus Link Scholar Link)

Assist. Prof. Sevin Teoman Duran

Bursa Uludag University, Turkiye (Scopus Link Scholar Link)

Dr. Rashid Iqbal

The Islamia University of Bahawalpur, Pakistan (Scopus Link Scholar Link)

Dr. Ayaz Mammadov

Western Caspian University, Azerbaijan (Scopus Link Scholar Link)

Dr. Babek Alibayov

Independent Research, Azerbaijan (Scopus Link Scholar Link)

CONTENT

PCR-Based Molecular Diagnostics of Human Papillomavirus (HPV)

Aysu Guluzade, Ayaz Mammadov, and Elin Aliyev 4

Harmonization and Integration of Azerbaijan National GeneBank Passport Data into Genesys

Ilhama Mirzaliyeva 15

Cancer Genetics: Comprehensive Analysis of Genetic Variants and Molecular Biomarkers Associated with Cancer Progression and Patient Survival

Madina Hamidova and Saltanat Aghayeva 22

Early Blood-Based Diagnostic Criteria in Cardiovascular Diseases

Natavan Karamova and Irina Amiraslanova 27

Chronic Renal Complications and Their Risks in Thalassemic Patients Receiving Transfusion

Sahin Bayramli, Aysu Ismayilova, Aygun Suleymanova and Shamsiyya Asgarli 35

Forest Plant Diseases and Pests: Assessing Biological Risks in Tourism Areas

Gadir Mammadov and Najaf Mammadzada 43

https://doi.org/10.54414/UJVF1455

Journal of Molecular Biosciences and Engineering

Research Article

PCR-Based Molecular Diagnostics of Human Papillomavirus (HPV)

Aysu Guluzade[1], Ayaz Mammadov[1], and Elin Aliyev[2]

[1]Department of Natural Sciences, School of Innovative Engineering and Natural Sciences, Western Caspian University, Baku, AZ1072, Azerbaijan

[2] Baku Medical Plaza, International Medical Center Closed Joint Stock Company, Babek Avenue 92n, Baku, Azerbaijan

Received: 14.09.2026 Accepted: 09.10.2026 Published: 30.10.2026

https://doi.org/10.54414/NEHE3462

Copyright: © 2026 by the authors. Licensee: Journal of Molecular Biosciences and Engineering, Western Caspian University, Baku, Azerbaijan. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution 4.0 International License (CC BY 4.0).

Abstract

Human papillomavirus (HPV) is one of the most common sexually transmitted viruses in the world. HPV is a key etiological factor in the development of cervical cancer and other anogenital and oropharyngeal malignancies. Early detection and accurate genotyping of this virus are crucial for disease prevention and screening programs. Among current molecular diagnostic methods, the polymerase chain reaction (PCR) has become the gold standard due to its high sensitivity and specificity, as well as its ability to detect low viral loads. This review summarizes the principles, methodologies, and diagnostic applications of PCR-based HPV detection. The review also discusses traditional PCR, real-time PCR, multiplex PCR, and other PCR-based approaches. The main advantages, limitations, and clinical importance of these methods are explained. Furthermore, it has been determined that there are factors affecting diagnostic accuracy, including sample collection and DNA extraction, primer selection, and result interpretation. The role of PCR in HPV genotyping, epidemiological surveillance, and the prevention of cervical cancer is also being investigated. The obtained results indicate that PCR-based molecular diagnostics ensure the reliable and highly sensitive detection of HPV infection. Furthermore, it supports public health strategies aimed at the early diagnosis of the infection, patient management, and the reduction of HPV-related diseases.

Keywords: molecular diagnostics, cervical cancer, DNA amplification, viral detection, screening, diagnostic accuracy.

1. Introduction

Human papillomavirus (HPV) belongs to the Papillomaviridae family. It is a non-enveloped virus (lacking a lipid envelope) with a genome consisting of circular, double-stranded DNA approximately 7,000–8,000 base pairs in length. More than 200 genotypes of HPV have been identified. They are classified into low- and high-risk groups based on their oncogenic potential.

Low-risk types, particularly HPV6 and HPV11, are primarily associated with benign growths such as condyloma acuminata (genital warts). High-risk types, such as HPV16, HPV18, HPV31, HPV33, HPV45, HPV52, and HPV58, are associated with cervical intraepithelial neoplasia (CIN) and several cancer types. Persistent infection with high-risk HPV is a major risk factor for malignant transformation. For instance, HPV16 and HPV18 cause more than 70% of cervical cancer cases worldwide [1], [2], [3], [4], [5].

CIN represents precancerous changes in the cervical epithelium. Depending on the severity of the dysplasia, it is classified as CIN1, CIN2, or CIN3. Since high-grade CIN progresses to invasive cervical cancer if not treated in

time, this underscores the importance of the early detection of high-risk HPV. Due to its high sensitivity and specificity, the polymerase chain reaction (PCR) is widely used for the detection of HPV DNA [1], [5].

The HPV virion has a diameter of approximately 50–55 nm. They possess an icosahedral capsid composed of L1 and L2 proteins. As the primary structural component, L1 forms the basis of prophylactic HPV vaccines through the formation of virus-like particles (VLPs). L2, on the other hand, aids in capsid assembly and the entry of the viral genome into host cells. The absence of a lipid envelope increases the virus's environmental stability [4]. The HPV genome consists of early (E), late (L), and long control region (LCR) functional regions. The early region includes the E1, E2, E4, E5, E6, and E7 genes, which are involved in regulating viral replication and the interaction with the host cell. The E6 and E7 genes encode key oncoproteins that disrupt the function of tumor suppressor proteins such as p53 and pRb. The activity of these oncoproteins leads to uncontrolled cell proliferation, the development of genomic instability, and, ultimately, the promotion of carcinogenesis. The late region encodes the L1 and L2 capsid proteins, while the LCR regulates viral replication and transcription [4].

Molecular and phylogenetic analyses have demonstrated the existence of significant genetic diversity among HPV genotypes, as well as within high-risk genotypes such as HPV16. Genomic variations can significantly influence the virus's persistence, ability to evade the immune response, pathogenicity, and oncogenic potential. Phylogenetic analyses have not only enabled a more precise classification of HPV variants but have also facilitated a better understanding of their evolutionary characteristics and clinical significance [1], [3], [4]. The development of molecular diagnostics and next-generation sequencing technologies has expanded the study of HPV genomic diversity and created new opportunities in the fields of epidemiological surveillance, risk assessment, vaccine development, and disease management. Therefore, the accurate identification of HPV genotypes via PCR-based molecular approaches is crucial for ensuring the effectiveness of modern cervical cancer screening and prevention strategies [1], [4], [5].

Routes of Transmission and Epidemiology of Human Papillomavirus (HPV)

Human papillomavirus (HPV) is an epitheliotropic DNA virus that infects the basal epithelial cells of the skin and mucous membranes. The virus typically enters the body through microscopic lesions in the epithelial tissue, resulting in infection of the basal cell layer. The primary route of HPV transmission is sexual contact. The infection can be transmitted during vaginal, anal, or oral sexual contact. However, penetrative sexual intercourse is not strictly required for the transmission of the virus, as direct skin-to-skin contact between the anogenital areas can also lead to the spread of the infection. Main risk factors include having multiple sexual partners, early onset of sexual activity, oral-genital contact, and immunosuppressive conditions. The fact that HPV infection is asymptomatic in most cases can enable infected individuals to transmit the virus to others without being aware of the infection [5].

Although its epidemiological significance is more limited compared to sexual transmission, non-sexual transmission of HPV is also considered possible. The virus's DNA was detected on contaminated surfaces and medical instruments. Furthermore, vertical transmission from mother to child can occur during pregnancy and, particularly, during vaginal delivery. In rare cases, perinatal HPV infection is associated with the development of recurrent respiratory papillomatosis in newborns [5], [6].

The potential effects of HPV infection on reproductive health have also been investigated. The detection of HPV DNA in semen samples has been associated with increased sperm DNA fragmentation and reduced sperm motility. These changes suggest that HPV infection could potentially affect male reproductive function. However, the molecular and cellular mechanisms underlying this connection have not yet been fully elucidated.

HPV is the most common sexually transmitted viral infection worldwide. More than 200 HPV genotypes are known, with approximately 40 infecting the anogenital tract. High-risk types, especially HPV16 and HPV18, cause most HPV-related cancers and more than 70% of cervical cancer cases. Persistent HPV infection is detected in approximately 95–99% of cervical cancers [5], [6].

Most sexually active individuals acquire HPV at least once during their lifetime. Approximately 90% of infections clear spontaneously within one to two years. However, persistent infection with high-risk genotypes increases the

risk of malignant transformation. Cervical cancer usually develops 15–20 years after persistent infection but may progress within 5–10 years in immunocompromised individuals, particularly those living with HIV [5].

The prevalence of high-risk HPV varies geographically. The highest rates are reported in sub-Saharan Africa, followed by Latin America, the Caribbean, Eastern Europe, and Southeast Asia. HPV is most common among women of reproductive age but also remains prevalent in women over 50 years, highlighting the importance of continued HPV screening throughout adulthood [5].

Recent epidemiological studies have demonstrated that HPV infection is highly prevalent in men as well as women. According to the World Health Organization [7], approximately one in three men worldwide is infected with the human papillomavirus (HPV), and one in five carries a high-risk HPV genotype. It is reported that HPV16 is the most frequently detected genotype among men. These results highlight the significant global public health burden associated with HPV infection and demonstrate that the virus affects both men and women. Although comprehensive nationwide epidemiological data on HPV infection in Azerbaijan are limited, recent studies indicate a significant prevalence of HPV among women of reproductive age.

A clinical study was conducted in Baku between 2021 and 2023 among women aged 18 to 65. High-risk HPV was detected in a significant proportion of the samples that tested positive. Among the most frequently encountered genotypes, HPV16, HPV18, HPV36, and HPV45 predominated. HPV infection was most frequently detected in women aged 30–45, and a significant proportion of the infections were asymptomatic; this underscores the importance of regular cervical cancer screening. Limited vaccination coverage, low levels of public awareness, and limited access to organized screening programs remain significant public health issues in Azerbaijan [6].

Preventive vaccination is one of the most effective strategies for preventing HPV infection and related diseases. Currently available vaccines (including Gardasil 9, Gardasil, and Cervarix) protect against several high-risk HPV genotypes, particularly HPV16 and HPV18. Vaccination is usually recommended before the onset of sexual activity—that is, at a time when the likelihood of prior HPV infection is low. Different dosing schedules are applied depending on the age at vaccination and the applicable immunization guidelines. For older age groups, however, a three-dose schedule is usually recommended [8].

The Association Between HPV Infection and Oncological Diseases

Human papillomavirus (HPV) is one of the most significant oncogenic viruses in humans and is estimated to cause approximately 5% of all cancer cases worldwide. Persistent infection caused by high-risk HPV genotypes is strongly associated with cervical cancer. It plays a significant role in the development of anal, vulvar, vaginal, penile, and oropharyngeal cancers. Most HPV infections are temporary and are effectively cleared by the body's immune system. However, persistent infection with high-risk genotypes significantly increases the risk of malignant transformation [6], [9], [10].

The process of carcinogenesis caused by HPV is primarily mediated by the virus's E6 and E7 oncoproteins. Following the integration of the viral DNA into the host genome, the E6 protein promotes the degradation of the tumor suppressor protein p53, while the E7 protein disrupts the function of the retinoblastoma protein (pRb). These molecular changes disrupt the normal regulation of the cell cycle, increase genomic instability, and lead to the uncontrolled proliferation of cells. Recent studies also indicate that oxidative stress caused by HPV can contribute to DNA damage and further accelerate the process of carcinogenesis. Furthermore, the body's genetic susceptibility factors can influence the persistence of the virus [6], [9], [10].

Conventional Methods for HPV Diagnosis and Their Limitations

Since HPV infection is asymptomatic in most cases, persistent infections may go undetected until precancerous lesions or invasive cancer develop. For this reason, accurate laboratory diagnostics are of great importance for ensuring effective screening and early intervention. For many years, the Papanicolaou (Pap) smear has been one of the primary screening methods based on the detection of cytological changes in cervical epithelial cells. However, this method does not directly detect HPV DNA and may have limited sensitivity, particularly in the early stages of infection. Colposcopy allows for the visual assessment of the cervical epithelium. It helps identify suspicious areas for biopsy following the application of acetic acid or Lugol's iodine solution. However, this

method also does not directly detect HPV infection, and its diagnostic results depend significantly on the experience of the specialist performing the examination [11], [12], [13], [14].

The development of molecular diagnostic methods, particularly PCR, has significantly expanded the capabilities for detecting and genotyping HPV infections. PCR enables the highly sensitive detection of viral DNA and the identification of specific HPV genotypes. Modern approaches such as real-time PCR, multiplex PCR, and digital PCR enable the rapid, sensitive, and reliable detection of high-risk HPV genotypes. These characteristics have made PCR-based methods one of the important molecular approaches for HPV screening, laboratory diagnostics, and genotyping [12], [15].

Importance of Early Diagnosis of HPV Infection

Persistent high-risk HPV infection is one of the key risk factors for the development of cervical cancer. The fact that HPV infection is often asymptomatic and can remain in a latent state for many years makes early detection difficult. For this reason, early diagnosis is crucial for identifying infected individuals before precancerous lesions or invasive cancer develop. Timely detection of the infection enables adequate clinical management and prevents the progression of the disease. Ultimately, this approach contributes to a significant reduction in the incidence and mortality rates of cervical cancer [13], [16].

Molecular diagnostic methods have significantly expanded the possibilities for early diagnosis by detecting HPV DNA before cytological changes appear. PCR-based tests make it possible to detect even very small amounts of viral DNA. This feature facilitates the detection of infections, particularly at an early stage or in the absence of clinical symptoms, as well as the precise identification of high-risk HPV genotypes. Early molecular diagnostics enable risk stratification and personalized patient monitoring. This approach can contribute to the timely implementation of clinical interventions, the improvement of treatment outcomes, and a reduction in the need for invasive procedures [13], [16].

Limitations of Conventional Diagnostic Methods

The Papanicolaou (Pap) smear has formed the basis of cervical cancer screening for many years and has contributed to a significant reduction in deaths from this disease. However, the Pap test primarily detects the cytological changes caused by HPV infection, rather than the infection itself. For this reason, its sensitivity in detecting early or latent infections may be limited. Cytological findings may be normal in high-risk HPV carriers. This situation can lead to false-negative results and a delay in diagnosis [11], [13], [16].

The diagnostic effectiveness of the Pap test depends on the quality of the sample, the preparation of the specimen, and the experience of the cytologist. These factors can lead to increased inter-observer variability in the assessment of results. Given its relatively low sensitivity, repeat examinations may be required to improve diagnostic accuracy. This leads to an increase in healthcare costs and the burden on the patient. Furthermore, the Pap test does not allow for the identification of the HPV genotype or the differentiation between low-risk and high-risk virus types.

Advances in the field of molecular diagnostics have made it possible to overcome several limitations of the Pap test. PCR-based HPV DNA tests—including real-time PCR, multiplex PCR, and digital PCR platforms—provide high sensitivity and specificity while also enabling accurate genotyping of the virus. For this reason, several countries have incorporated HPV DNA testing into their primary cervical cancer screening programs. However, the approach taken has varied: in some instances, these tests have been implemented to replace cytological screening, while in others, they serve to complement it. The combination of PCR-based HPV tests with cytological examination holds high diagnostic value for the early detection, risk assessment, and clinical management of HPV infection [11], [12], [13], [15], [16].

2. Materials and Methods

Study Population and Clinical Samples

This study included cervical samples collected from 149 patients aged 18–60 years who had undergone HPV testing. Cervical smear samples were collected by qualified gynecologists using sterile cervical brushes and immediately placed into a liquid-based transport medium. Subsequently, the samples were delivered to the

Molecular Genetics Laboratory of "Baku Medical Plaza" (Babek branch) for molecular analysis. All laboratory procedures were performed according to the laboratory's standard operating procedures (SOPs).

Sample Collection, Storage, and Transportation

Cervical samples were obtained from the cervical transformation zone, the anatomical site where HPV-associated lesions most frequently develop. After collection, specimens were placed into sterile transport tubes containing preservation medium and transported to the laboratory under recommended conditions. Samples were processed immediately or stored at 2–8°C for short-term preservation until DNA extraction. All procedures complied with standard biosafety and laboratory quality-control guidelines [11], [17], [18].

DNA Extraction and Quality Assessment

Genomic DNA was extracted from cervical specimens using a commercial silica-based DNA extraction kit according to the manufacturer's instructions. The extraction procedure included cell lysis, nucleic acid adsorption onto a silica sorbent, washing to remove contaminants, and DNA elution. All pre-PCR procedures were carried out in a laminar airflow cabinet to minimize the risk of contamination. Negative controls were included throughout the extraction process to monitor possible contamination.

The concentration and purity of the extracted DNA were assessed before PCR analysis to ensure adequate template quality. DNA samples meeting the quality requirements were used for subsequent molecular testing, whereas extracted DNA was stored at −20°C until analysis, when immediate amplification was not performed [19], [20].

PCR Amplification & Statistical Analysis

HPV DNA detection was performed using a real-time polymerase chain reaction (Real-Time PCR) assay following the manufacturer's protocol. The reaction mixture contained template DNA, sequence-specific primers, fluorescent probes, nucleotides, reaction buffer, magnesium ions, and thermostable DNA polymerase. Amplification was carried out in a real-time PCR thermal cycler under optimized cycling conditions. Appropriate positive and negative controls were included in each PCR run to validate assay performance and exclude contamination.

PCR results were analyzed using standard laboratory software. Descriptive statistical methods were applied to summarize the frequency and distribution of HPV-positive samples according to age group and HPV genotype. Statistical analyses were performed using appropriate software, and results were considered statistically significant at $p < 0.05$.

HPV DNA was detected using a real-time polymerase chain reaction (Real-Time PCR) assay. This method relies on the exponential amplification of target HPV DNA sequences through repeated steps such as denaturation, primer annealing, and DNA chain extension. Fluorescence-labeled probes included in the reaction allow for real-time monitoring of the amplification process, facilitating the qualitative detection and accurate genotyping of HPV. These features enable the sensitive and specific identification of HPV DNA in clinical samples [21].

PCR analysis was performed using a commercially available "master mix" containing Taq DNA polymerase, reaction buffer, Mg^{2+} ions, deoxynucleotide triphosphates (dNTPs), genotype-specific forward and reverse primers, and fluorescence-labeled oligonucleotide probes. DNA purified from clinical samples was used as the template for amplification. To minimize the risk of cross-contamination, all reagents were prepared and used in a dedicated work area designated for the pre-PCR stage.

The primer pairs were designed to specifically target conserved regions of the HPV genome, enabling the sensitive detection of clinically significant high-risk HPV genotypes while minimizing non-specific amplification. Positive and negative controls were included in each amplification run to monitor assay performance and detect potential contamination. PCR amplification and fluorescence detection were performed using a real-time thermal cycler in accordance with the manufacturer's instructions. The resulting fluorescent signals were analyzed to determine the presence of HPV DNA and identify the specific HPV genotype [15], [19], [20], [22], [23], [24].

The PCR amplification protocol consisted of an initial denaturation step followed by repeated cycles of denaturation, primer annealing, and extension. Initial denaturation was performed at 94–95°C for 3–5 minutes to

ensure complete separation of the double-stranded DNA and activation of the DNA polymerase. Following this stage, 35–40 amplification cycles were performed. In each cycle, denaturation was performed at 94–95°C for 15–30 seconds to separate the DNA strands. Subsequently, the primer annealing step, which allows sequence-specific primers to hybridize with the target HPV DNA sequences, was performed at a temperature of 50–65°C for 30–60 seconds. Subsequently, an extension step was performed at 72°C for 30–60 seconds. At this stage, Taq DNA polymerase synthesized complementary DNA strands by incorporating deoxyribonucleotides. After the amplification cycles were completed, a final extension step was performed at 72°C for 5–10 minutes to ensure the complete synthesis of the amplified products. Subsequently, the reaction mixture was maintained at 4°C until the PCR tubes were removed from the instrument. Depending on the thermal cycler used and the specific amplification protocol, the total amplification time under these conditions was approximately 1.5–2 hours (Table 1).

Table 1. PCR setup

PCR Step	Temperature (°C)	Time	Purpose
Initial denaturation	94–95	3–5 min	DNA strand separation and enzyme activation
Denaturation	94–95	15–30 s	Separation of DNA strands
Annealing	50–65	30–60 s	Primer binding to target DNA
Extension	72	30–60 s	DNA synthesis by Taq polymerase
Final extension	72	5–10 min	Completion of DNA synthesis
Hold	4	Until sample removal	Preservation of PCR products

3. Results and Discussion

Interpretation of Real-Time PCR Results

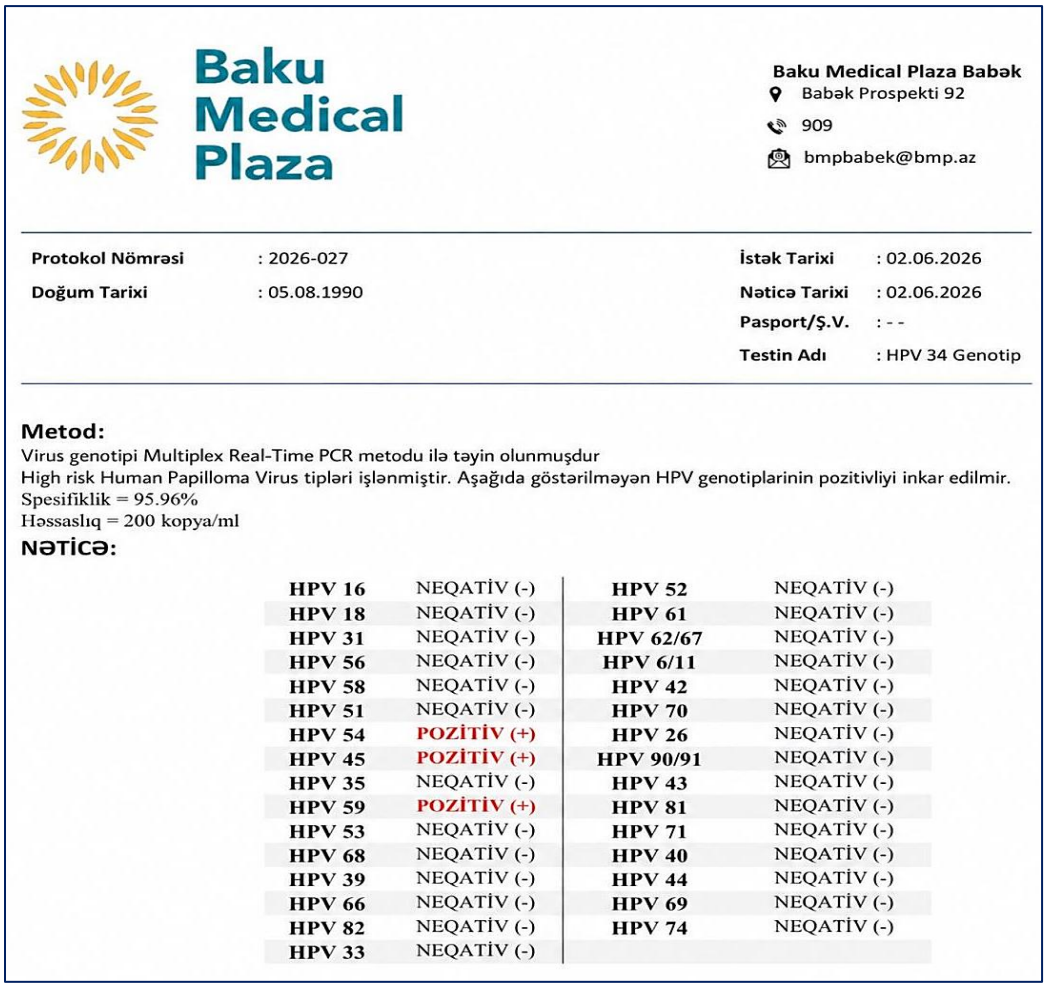
Real-time PCR results were interpreted in accordance with the manufacturer's instructions and established laboratory quality control criteria. Internal controls were used to verify successful DNA extraction and monitor for potential PCR inhibition, while external positive and negative controls were included in each amplification run to assess assay performance and monitor for potential contamination [25], [26].

Amplification curves and cycle threshold (Ct) values were evaluated to determine the presence or absence of HPV DNA. Samples showing amplification within the predetermined Ct range were considered positive. Provided that the corresponding internal control sample yielded a valid result, samples showing amplification outside the established threshold value or exhibiting no amplification were considered negative.

To ensure the reliability and diagnostic accuracy of the results, all findings were interpreted in conjunction with relevant quality control parameters and available clinical data (Figure 1). Real-time PCR provides a highly sensitive and specific method for the detection and genotyping of HPV, supporting early diagnosis, risk stratification, and appropriate clinical management [25], [26].

Analysis of overall HPV positivity rates

During the study, PCR analysis results of individuals who applied to the laboratory of the Baku Medical Plaza clinic between February 16 and May 25 for the detection of HPV infections were analyzed. During the study period, samples taken from a total of 149 individuals were examined. Of those examined, 135 were women, and 14 were men. In the vast majority of the analyzed samples, a test panel covering 14 high-risk HPV genotypes was used, and in only one patient was an extended HPV test covering 34 genotypes performed (Figure 2).



Baku Medical Plaza

Baku Medical Plaza Babək
Babək Prospekti 92
909
bmpbabek@bmp.az

Protokol Nömrəsi	: 2026-027	**İstək Tarixi**	: 02.06.2026
Doğum Tarixi	: 05.08.1990	**Nəticə Tarixi**	: 02.06.2026
		Pasport/Ş.V.	: - -
		Testin Adı	: HPV 34 Genotip

Metod:
Virus genotipi Multiplex Real-Time PCR metodu ilə təyin olunmuşdur
High risk Human Papilloma Virus tipləri işlənmiştir. Aşağıda göstərilməyən HPV genotiplarinin pozitivliyi inkar edilmir.
Spesifiklik = 95.96%
Həssaslıq = 200 kopya/ml

NƏTİCƏ:

HPV 16	NEQATİV (-)	**HPV 52**	NEQATİV (-)
HPV 18	NEQATİV (-)	**HPV 61**	NEQATİV (-)
HPV 31	NEQATİV (-)	**HPV 62/67**	NEQATİV (-)
HPV 56	NEQATİV (-)	**HPV 6/11**	NEQATİV (-)
HPV 58	NEQATİV (-)	**HPV 42**	NEQATİV (-)
HPV 51	NEQATİV (-)	**HPV 70**	NEQATİV (-)
HPV 54	**POZİTİV (+)**	**HPV 26**	NEQATİV (-)
HPV 45	**POZİTİV (+)**	**HPV 90/91**	NEQATİV (-)
HPV 35	NEQATİV (-)	**HPV 43**	NEQATİV (-)
HPV 59	**POZİTİV (+)**	**HPV 81**	NEQATİV (-)
HPV 53	NEQATİV (-)	**HPV 71**	NEQATİV (-)
HPV 68	NEQATİV (-)	**HPV 40**	NEQATİV (-)
HPV 39	NEQATİV (-)	**HPV 44**	NEQATİV (-)
HPV 66	NEQATİV (-)	**HPV 69**	NEQATİV (-)
HPV 82	NEQATİV (-)	**HPV 74**	NEQATİV (-)
HPV 33	NEQATİV (-)		

Figure 1. Evaluation of HPV results.

The results obtained showed that HPV infection was detected in the majority of the patients who underwent the analysis. In addition, a positive HPV result was recorded in 45 women. In addition, weak negative results were recorded in 2 women and 1 man. Weak negative results are usually associated with a low viral load or indicators close to the detection limit during the analysis. Such results are considered cases requiring clinical monitoring, and re-examination may be recommended after a certain period of time.

Based on the results of the study, HPV positivity was observed only in women. This indicator may be because women are more likely to be examined for the purpose of diagnosing HPV infection and the widespread use of cervical cancer screening programs. The lack of positive results among men may be due to the small number of male patients included in the study.

Various high-risk genotypes were identified among the HPV positive results. As a result of the genotyping, the highest frequency was observed in the HPV39 genotype. This genotype was detected in 13 people. The second highest was the HPV66 genotype, which was detected in 12 people. HPV16 and HPV52 were both detected in 7 people. HPV35 and HPV68 genotypes were recorded in 6 people, respectively.

Among other genotypes, HPV45, HPV56, and HPV59 genotypes were detected in 4 individuals each, HPV18 and HPV33 genotypes in 3 individuals each, and HPV31 and HPV51 genotypes in 2 individuals each. The lowest incidence was observed in the HPV58 genotype and was detected in only 1 individual.

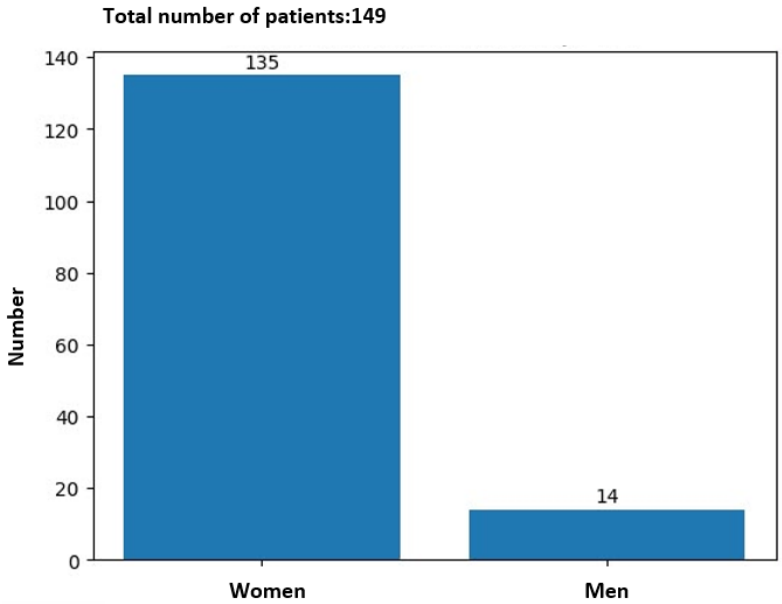


Figure 2. Total number of patients who underwent the analysis, separately for women and men.

Analysis of the distribution of genotypes shows that not only the classical high-risk HPV16 and HPV18, but also other high-risk genotypes such as HPV39, HPV66, HPV35, and HPV68 were widely distributed in the study group. This result indicates the genetic diversity of HPV infection and the importance of using a wide genotype panel in molecular diagnostics (Figure 3).

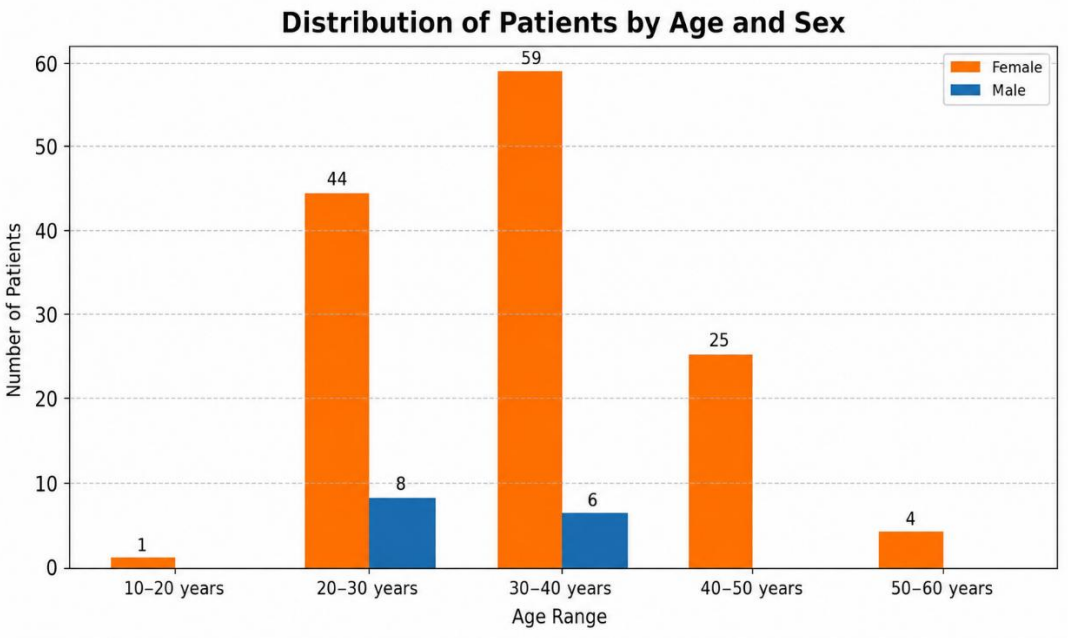


Figure 3. Distribution of those infected by age and gender.

It was found that in each of the patients who received a positive result, a minimum of 1 and a maximum of 3 genotypes were positive. This fact indicates that several genotypes can be positive in one patient at the same time. Cases of infection with multiple genotypes are of particular interest from a clinical point of view, since various

studies have shown that the presence of more than one high-risk HPV genotype in the same person can increase the risk of persistence and the likelihood of developing oncological changes.

Overall, the results obtained show that PCR technology is highly effective in accurately detecting HPV infection and its various genotypes. Due to the high sensitivity of the method, cases of infection with both single and multiple genotypes were identified. The analyses conducted show that HPV infection is quite widespread in the studied group, especially as high-risk genotypes prevail, and molecular diagnostics are of great importance in terms of early detection.

Thus, the study results confirmed the high informativeness of PCR technology in the diagnosis of HPV infection, allowing for the assessment of both overall positivity rates and the distribution of high-risk genotypes. The data obtained are of great importance for future epidemiological studies and the prevention of HPV-related diseases.

Comparative Analysis of HPV Prevalence Across Age Groups

The examined individuals were divided into different age groups and evaluated. This approach made it possible to determine the age group in which HPV infection is most common and to assess the epidemiological characteristics of the infection. According to the data obtained, the majority of the individuals participating in the study were between 20 and 40 years of age. A total of 52 individuals were examined in the 20-30 age group, of which 44 were women and 8 were men. This age group accounts for the majority of the total applicants. A total of 65 individuals in the 30-40 age group provided analysis. Of these individuals, 59 were women, and 6 were men. Thus, the largest number of applications was in the 30-40 age group. The number of people examined in the 40-50 age group was somewhat lower than the previous age group. In this age range, 26 women submitted an application, and no male patients were registered. In the 50-60 age group, only 4 women applied for examination. The least number of applications was in the 10-20 age range, and 1 woman in this age group submitted an analysis.

Analysis of the results by age groups showed that HPV positive results were observed only in people aged 21-36. This result indicates that HPV was detected in patients of reproductive age. As is known, HPV is one of the most common sexually transmitted infections, and the highest prevalence rates of infection are usually recorded among young and middle-aged people. The results obtained during the study also confirmed these epidemiological features. The higher number of positive results in the 20-30 age group is associated with the more active reproductive and social lifestyle of individuals in that age group. At the same time, the detection of positive cases in the 30-36 age group indicates that the viral infection remains relevant not only in young people, but also in the later reproductive period. In addition, the reason for the lack of positivity in people over 40 years of age is due to the low number of applicants in that age group.

The study results show that the 21-36 age group can be considered a risk group in terms of detecting infection. In this regard, regular gynecological examinations, participation in HPV screening programs and the use of molecular diagnostic methods in this age group can help in the early detection of infection and prevent possible complications.

4. Conclusion

This study demonstrated the high diagnostic performance of real-time polymerase chain reaction (PCR) for the detection and genotyping of human papillomavirus (HPV). The implementation of internal and external quality control methods has strengthened the robustness of the molecular diagnostic process by ensuring the reliability of DNA extraction, amplification, and result interpretation. Among the 149 individuals analyzed, HPV infection was detected exclusively in female participants, with positive results observed primarily in the 21–36 age group. The absence of HPV-positive results among male participants should be interpreted with caution, given the limited number of men included in the study. Genotype analysis revealed significant diversity. HPV39 and HPV66 were the most frequently detected high-risk genotypes, followed by HPV16, HPV52, HPV35, and HPV68. These results indicate that, alongside HPV16 and HPV18, other high-risk genotypes may play a significant role in the development of HPV infection within the studied population.

Both single and multiple genotype infections were identified, underscoring the importance of comprehensive HPV genotyping. The observed genotype distribution supports the use of broad-spectrum PCR tests capable of detecting multiple high-risk HPV types. HPV infection was most frequently detected in women of reproductive age, highlighting the importance of appropriate screening and early molecular diagnostics.

The study is limited by a relatively small sample size, a limited number of male participants, and a single-center design. Larger-scale, multicenter studies are needed to more accurately characterize HPV epidemiology and genotype distribution in Azerbaijan. Such studies can contribute to the optimization of screening strategies, vaccination policies, and other preventive measures.

Author Contributions

The sole author is responsible for conceptualizing, collecting, and analyzing data, and writing and editing the manuscript.

Conflict of Interest

The authors declare no conflicts of interest.

Funding

This research received no external funding.

References

[1] Katabathina, V. S., Ghannam, S., Chen, M., Desalme, B., Gabos, R., Emejulu, I., & Prasad, S. R. (2024). Update on pathologic conditions, imaging findings, prevention, and management of human papillomavirus–related neoplasms. *Radiographics*, *44*(8), e230179. https://doi.org/10.1148/rg.230179

[2] Li, Y., & Wu, X. (2025). Vaginal microbiome distinction in women with HPV+, cervical intraepithelial neoplasia, and cervical cancer, a retrospective study. *Frontiers in Cellular and Infection Microbiology*, *14*, 1483544. https://doi.org/10.3389/fcimb.2024.1483544

[3] Nikmanesh, N., Hosseini, S., Mirbagheri, F., Asadsangabi, K., Fattahi, M. R., Safarpour, A. R., ... & Nikmanesh, Y. (2024). Knowledge on human papillomavirus infections, cancer biology, immune interactions, vaccination coverage and common treatments: a comprehensive review. *Viral Immunology*, *37*(5), 221-239. https://doi.org/10.1089/vim.2023.0144

[4] Prétet, J.L., Charlot, J.F., Mougin, C. (2007). Aspects virologiques et carcinologiques des papillomavirus humains HPV [Virological and carcinogenic aspects of HPV]. *Bull Acad Natl Med., 191*(3):611-623.

[5] Wolf, J., Kist, L. F., Pereira, S. B., Quessada, M. A., Petek, H., Pille, A., & Nasi, L. A. (2024). Human papillomavirus infection: Epidemiology, biology, host interactions, cancer development, prevention, and therapeutics. *Reviews in Medical Virology*, *34*(3), e2537. https://doi.org/10.1002/rmv.2537

[6] Malagon, T., Franco, E. L., Tejada, R., & Vaccarella, S. (2024). Epidemiology of HPV-associated cancers past, present and future: towards prevention and elimination. *Nature Reviews Clinical Oncology*, *21*(7), 522-538. https://doi.org/10.1038/s41571-024-00904-z

[7] WHO (2023) One in three men worldwide are infected with genital human papillomavirus. https://www.who.int/news/item/01-09-2023-one-in-three-men-worldwide-are-infected-with-genital-human-papillomavirus

[8] Goldstone, S. E. (2023). Human papillomavirus (HPV) vaccines in adults: Learnings from long-term follow-up of quadrivalent HPV vaccine clinical trials. *Human vaccines & immunotherapeutics*, *19*(1), 2184760. https://doi.org/10.1080/21645515.2023.2184760

[9] Lu, Z., Haghollahi, S., & Afzal, M. (2024). Potential therapeutic targets for the treatment of HPV-associated malignancies. *Cancers*, *16*(20), 3474. https://doi.org/10.3390/cancers16203474
[10] Bhatia, R., Alcaniz Boada, E., Bonde, J. H., Quint, W. G., Xu, L., Ejegod, D. M., ... & Arbyn, M. (2023). Papilloplex HR-HPV test has non-inferior clinical performance for detection of human papillomavirus infection: assessment using the VALGENT framework. *Journal of Clinical Pathology*, *76*(3), 172-176. https://doi.org/10.1136/jclinpath-2021-207864
[11] Castle, P. E., Stoler, M. H., Wright, T. C., Sharma, A., Wright, T. L., & Behrens, C. M. (2011). Performance of carcinogenic human papillomavirus (HPV) testing and HPV16 or HPV18 genotyping for cervical cancer screening of women aged 25 years and older: a subanalysis of the ATHENA study. *The Lancet Oncology*, *12*(9), 880-890.
[12] Arbyn, M., Simon, M., Peeters, E., Xu, L., Meijer, C. J., Berkhof, J., & Poljak, M. (2021). 2020 list of human papillomavirus assays suitable for primary cervical cancer screening. *Clinical Microbiology and Infection*, *27*(8), 1083-1095. https://doi.org/10.1016/j.cmi.2021.04.031
[13] Perkins, R. B., Guido, R. S., Castle, P. E., Chelmow, D., Einstein, M. H., Garcia, F., & Schiffman, M. (2020). 2019 ASCCP risk-based management consensus guidelines for abnormal cervical cancer screening tests and cancer precursors. *Journal of lower genital tract disease*, *24*(2), 102. https://doi.org/10.1097/LGT.0000000000000525
[14] Schiffman, M., Hyun, N., Raine-Bennett, T. R., Katki, H., Fetterman, B., Gage, J. C., ... & Wentzensen, N. (2016). A cohort study of cervical screening using partial HPV typing and cytology triage. *International journal of cancer*, *139*(11), 2606-2615. https://doi.org/10.1002/ijc.30375
[15] Arbyn, M., Verdoodt, F., Snijders, P. J., Verhoef, V. M., Suonio, E., Dillner, L., & Anttila, A. (2014). Accuracy of human papillomavirus testing on self-collected versus clinician-collected samples: a meta-analysis. *The Lancet Oncology*, *15*(2), 172-183. https://doi.org/10.1016/S1470-2045(13)70570-9
[16] Nayar, R., & Wilbur, D. C. (2017). The Bethesda system for reporting cervical cytology: a historical perspective. *Acta Cytologica*, *61*(4-5), 359-372. https://doi.org/10.1159/000477556
[17] Arbyn, M., Cuschieri, K., Bonde, J., Schuurman, R., Cocuzza, C., Broeck, D. V., & Poljak, M. (2024). Criteria for second-generation comparator tests in validation of novel HPV DNA tests for use in cervical cancer screening. *Journal of Medical Virology*, *96*(9), e29881. https://doi.org/10.1002/jmv.29881
[18] Perkins, R. B., Wentzensen, N., Guido, R. S., & Schiffman, M. (2023). Cervical cancer screening: a review. *Jama*, *330*(6), 547-558.
[19] Fashedemi, O., Ozoemena, O. C., Peteni, S., Haruna, A. B., Shai, L. J., Chen, A., & Ozoemena, K. I. (2025). Advances in human papillomavirus detection for cervical cancer screening and diagnosis: challenges of conventional methods and opportunities for emergent tools. *Analytical Methods*, *17*(7), 1428-1450. https://doi.org/10.1039/d4ay01921k
[20] Bruni, L. B. R. L., Barrionuevo-Rosas, L., Albero, G., Serrano, B., Mena, M., Gómez, D., & De Sanjosé, S. (2019). Human papillomavirus and related diseases report. *ICO/IARC Information Centre on HPV and Cancer (HPV Information Centre)*, *7*, 26-42.
[21] Thornton, B., & Basu, C. (2011). Real-time PCR (qPCR) primer design using free online software. *Biochemistry and Molecular Biology Education*, *39*(2), 145-154. https://doi.org/10.1002/bmb.20461
[22] Ali, N., Rampazzo, R. D. C. P., Costa, A. D. T., & Krieger, M. A. (2017). Current nucleic acid extraction methods and their implications to point-of-care diagnostics. *BioMed Research International*, *1*, 9306564.
[23] Arya, M., Shergill, I. S., Williamson, M., Gommersall, L., Arya, N., & Patel, H. R. (2005). Basic principles of real-time quantitative PCR. *Expert review of molecular diagnostics*, *5*(2), 209-219. https://doi.org/10.1586/14737159.5.2.209
[24] Bustin S, Nolan T. (2017). Talking the talk but not walking the walk: RT-qPCR as a paradigm for the lack of reproducibility in molecular research. *Eur J Clin Invest., 47*:756–74
[25] Mackay, I. M., Arden, K. E., & Nitsche, A. (2002). Real-time PCR in virology. *Nucleic Acids Research*, *30*(6), 1292-1305. https://doi.org/10.1093/nar/30.6.1292
[26] Mackay, I. M. (2004). Real-time PCR in the microbiology laboratory. *Clinical microbiology and infection*, *10*(3), 190-212. https://doi.org/10.1111/j.1198-743X.2004.00722.x

Journal of Molecular Biosciences and Engineering

Research Article

Harmonization and Integration of Azerbaijan National GeneBank Passport Data into Genesys

Ilhama Mirzaliyeva

Genetic Resources Institute, Ministry of Science and Education of the Republic of Azerbaijan, Baku, Azadliq Avenue 155, AZ1106, Azerbaijan

Received: 21.09.2026 Accepted: 16.10.2026 Published: 30.10.2026

https://doi.org/10.54414/XOFO8878

Copyright: © 2026 by the authors. Licensee: Journal of Molecular Biosciences and Engineering, Western Caspian University, Baku, Azerbaijan. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution 4.0 International License (CC BY 4.0).

Abstract

The conservation and effective management of plant genetic resources for food and agriculture (PGRFA) depend not only on the maintenance of biological material but also on the availability of reliable and standardized information associated with individual accessions. Genebanks maintain passport data that support the identification, management, exchange, and utilization of conserved genetic resources. However, differences in database structures, terminology, and data formats can limit the interoperability and international exchange of such information. In Azerbaijan, passport information for plant genetic resources is maintained in the Central Database (CDB) of the Azerbaijan National Genebank. This study describes the preparation and harmonization of National Genebank passport data for integration into the Genesys PGR platform. The process included review and refinement of existing passport records, standardization of field content and formats, mapping of national database fields to the corresponding Multi-Crop Passport Descriptors (MCPD), verification of taxonomic and geographic information, and selection of accessions suitable for public sharing. The integration process established a standardized dataset suitable for international data exchange and improved the accessibility of information on Azerbaijan's conserved plant genetic resources. The experience demonstrates that successful integration into an international PGR information platform requires systematic data preparation, descriptor harmonization, quality verification, and appropriate management of public-access information. The resulting integration provides a basis for improved discoverability of national genetic resources and supports their use in research, breeding, and international collaboration.

Keywords: plant genetic resources, Genebank, Genesys, passport data, MCPD, data harmonization, data standardization, data integration.

1. Introduction

Plant genetic resources for food and agriculture (PGRFA) provide the genetic basis for crop improvement, adaptation, and sustainable agricultural production. Genebanks play a central role in conserving this diversity and maintaining the passport information required to identify, manage and exchange individual accessions [1]. The quality and completeness of such information are important for the effective management and use of ex situ collections [2].

The increasing use of digital information systems has created new opportunities for making Genebank collections more discoverable and facilitating access to information by researchers, breeders, and other users [3]. Genesys PGR is an international information platform that provides access to passport information on plant genetic resources maintained by Genebanks and other institutions. Effective information management is an essential component of long-term genetic resource conservation, as documentation provides the link between conserved biological material and its potential use [4]. International catalogues such as EURISCO demonstrate the value of

standardized passport information for making Genebank material searchable across institutional and national boundaries [5], [6].

Effective international exchange of passport information requires compatible data structures and standardized terminology. The Multi-Crop Passport Descriptors (MCPD) provide a common framework for documenting passport information and facilitate interoperability between national and international plant genetic resource information systems [7].

In Azerbaijan, passport information for the national plant genetic resources collection is maintained in the Central Database (CDB) of the Azerbaijan National Genebank. The dataset was reviewed for data consistency and metadata quality, and the MCPD-compatible passport records were prepared for submission to Genesys, with accessions selected according to their public accessibility status. The development of the national Genebank has supported the conservation and management of plant genetic resources in Azerbaijan [8], [9]. Previous work has also demonstrated the use of international descriptor systems for documenting characterization data from the national collection [10], [11].

The objective of this study was to describe the harmonization and preparation of Azerbaijan National Genebank passport data for integration into the Genesys PGR platform and to assess the resulting contribution to the international accessibility of national plant genetic resources information.

2. Materials and Methods

The study was based on passport data maintained in the Central Database (CDB) of the Azerbaijan National Genebank. The dataset contains information associated with accessions conserved in the national ex situ collection, including accession identification, taxonomy, geographic origin, collection and conservation-related information. Following database review and inventory assessment, passport records for 15286 registered accessions were used as the basis for data preparation. A subset of 10296 accessions was maintained under medium- and long-term storage conditions.

The passport records were maintained in a structure compatible with the Multi-Crop Passport Descriptors (MCPD). The preparation process therefore focused on reviewing the existing fields, standardizing field content and formats where necessary, mapping national database fields to the corresponding MCPD descriptors, and verifying the consistency of taxonomic and geographic information [7]. The data preparation process included:

- review of the existing passport data structure;
- refinement and standardization of data fields and formats;
- mapping of national database fields to MCPD descriptors;
- verification of taxonomic and geographic information
- identification and correction of inconsistencies where applicable; and
- preparation of the standardized dataset for Genesys integration.

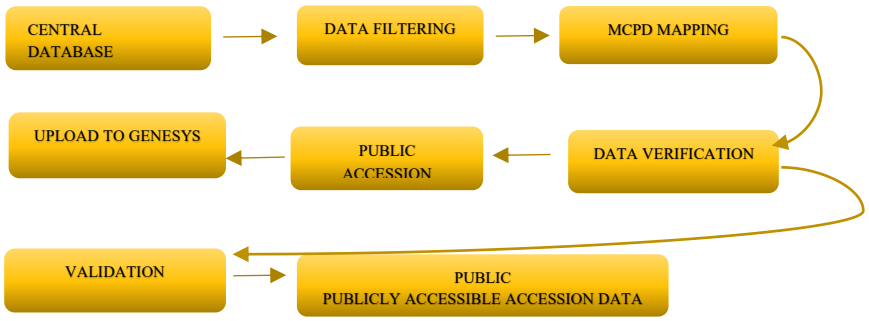


Figure 1. Workflow for the harmonization and integration of passport data into Genesys.

Before integration into Genesys, accession records were reviewed according to their public-sharing status. The “Is public” filter was set to “Yes” to identify accessions eligible for public dissemination. This filtering step ensured that only records designated as publicly accessible were submitted for integration into the international platform. The harmonized passport dataset was structured for exchange with Genesys using appropriate structured data formats. The integration workflow consisted of the stages shown in Figure 1.



Figure 2. Workflow for the preparation and integration of passport data into Genesys.

The selected passport records were uploaded to Genesys using the Genesys Uploader Tool. Following upload, the submitted records were validated to confirm successful integration and data consistency. After successful validation, the accession records became publicly accessible through Genesys.

3. Results

The review of the national passport dataset identified differences in field structure, terminology, and formatting that required harmonization before international data exchange. National database fields were mapped to the corresponding MCPD descriptors, and the resulting records were prepared in a standardized structure for Genesys integration.

The preparation process established a consistent data structure linking the national passport database with the MCPD framework. Taxonomic and geographic information were also reviewed during data preparation to improve the consistency and reliability of the submitted records.

The public-accessibility filtering procedure was applied before data submission to Genesys. The "Is public = Yes" criterion was used to identify accession records eligible for international dissemination. A total of 15,286 accessions were uploaded, representing 117 families, 460 genera, 892 species, and 367 subspecies. Of these, 10,296 accessions were maintained in medium- and long-term cold storage facilities (Figure 3).

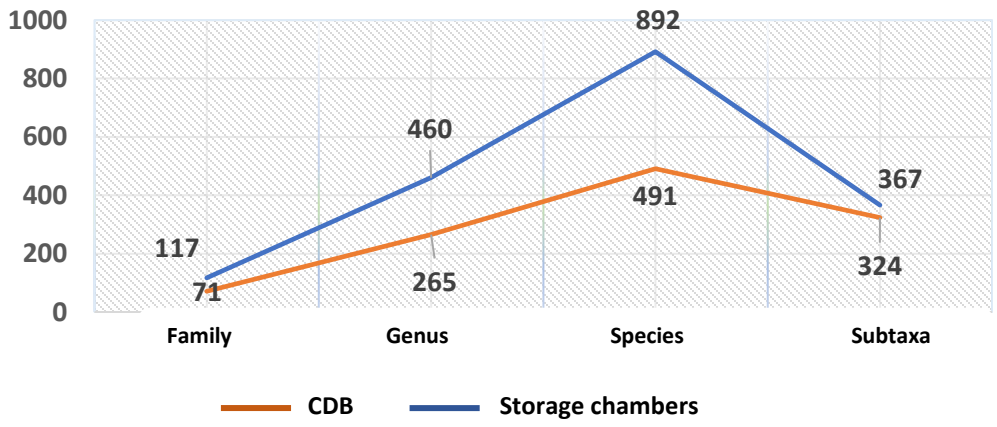


Figure 3. Distribution of accessions in the CDB and in medium- and long-term cold storage according to taxonomic groups.

The integration made eligible passport information from the Azerbaijan National Genebank available through an international PGR information environment. The standardized records can be discovered using the information made available through Genesys and can support further research, breeding, and comparison of plant genetic resources.

The CDB contains 15286 registered accessions belonging to a broad range of plant genetic resources. For the purpose of contextualizing the integration dataset, the collection comprised cereals, legumes, medicinal plants, forage plants, industrial crops, vegetable crops, and fruit crops (Figure 4).

The registered accessions comprised 6123 cereals (40.1%), 2112 legumes (13.8%), 849 medicinal plants (5.6%), 815 forage plants (5.3%), 1670 industrial crops (10.9%), 937 vegetable crops (6.1%), and 2780 fruit crops (18.2%).

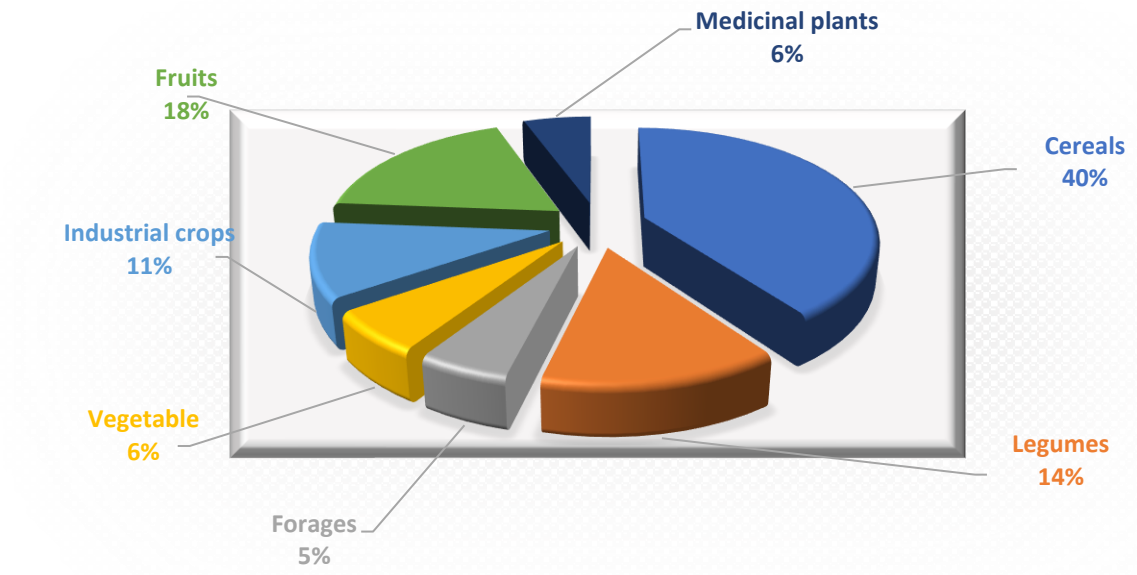


Figure 4. Distribution of registered accessions among major crop groups.

4. Discussion

The preparation of Azerbaijan National Genebank passport data for integration into Genesys demonstrates that international accessibility of Genebank information depends on the quality and structure of the underlying national data. Integration is therefore not limited to the technical transfer of records; it requires prior data refinement, standardization, verification, and appropriate selection of publicly accessible information. This is consistent with the broader role of information management in the long-term conservation and use of plant genetic resources [4].

The use of MCPD provided a common framework for mapping national passport fields to an internationally recognized descriptor structure. Standardized passport information facilitates the exchange and interpretation of accession data across information systems and contributes to the broader interoperability of PGR documentation [2], [7].

The verification of taxonomic and geographic information was an important component of the preparation process. Accurate passport information is essential for the interpretation and discovery of Genebank accessions, while inconsistencies in passport data can reduce their value for users and affect the reliability of information exchanged between databases [2].

The use of a public-accessibility filter also demonstrates the importance of distinguishing between information maintained within a national database and information that can be disseminated through an international platform. The “Is public = Yes” criterion provided a practical mechanism for selecting records eligible for public sharing.

Integration into Genesys makes Azerbaijan's plant genetic resource information discoverable beyond the national database environment. The experience is consistent with the role of international PGR information systems such as EURISCO in facilitating the discovery and use of conserved genetic resources through standardized passport information [5], [6]. The availability of national passport information through such systems can also complement ongoing efforts to document and conserve Azerbaijan's genetic resources [8], [9], [12].

The composition of the integrated collection, including cereals, legumes, medicinal, forage, industrial, vegetable, and fruit crops, illustrates the breadth of the national ex situ resource base. Such diversity provides a documented foundation for further characterization and utilization activities. Previous characterization studies from the

Azerbaijan National Genebank have similarly demonstrated the value of structured descriptor-based information for documenting conserved plant material [10].

The integration process also highlights the importance of maintaining passport-data quality after the initial submission. Changes to accession records, taxonomic information, and other passport fields require continued review and updating so that information available through international platforms remains consistent with the national collection. Regular maintenance is particularly important for the long-term reliability and usefulness of Genebank information systems [4], [13].

Future work should therefore focus on regular updating of passport records, improving data completeness, maintaining MCPD compatibility, integrating additional datasets, and strengthening links between passport, characterization and evaluation information. Such developments would further improve the value of the national information infrastructure for research, breeding and international collaboration.

4. Conclusion

The harmonization and integration of Azerbaijan National Genebank passport data into Genesys established a standardized pathway for international data exchange and improved the accessibility of information on conserved plant genetic resources. The application of MCPD, together with data refinement, field mapping, metadata verification, and public-accessibility filtering, provided a practical basis for preparing national passport information for integration into an international PGR information platform. The experience demonstrates that effective digital integration requires coordinated attention to data structure, terminology, quality, and public-access conditions. Continued maintenance and updating of the national dataset will support the reliability and long-term usefulness of the information made available through Genesys and will facilitate the wider discovery and potential use of Azerbaijan's conserved plant genetic resources.

Author Contributions

The sole author is responsible for conceptualizing, collecting, and analyzing data, and writing and editing the manuscript.

Conflict of Interest

The authors declare no conflicts of interest.

Funding

This research received no external funding.

References

[1] Commission on Genetic Resources for Food. (2010). The second report on the state of the world's plant genetic resources for food and agriculture (Vol. 2). Food & Agriculture Organization of the UN (FAO).

[2] Van Hintum, T., Menting, F., & Van Strien, E. (2011). Quality indicators for passport data in ex situ genebanks. *Plant Genetic Resources*, *9*(3), 478-485.

[3] Oppermann, M., Weise, S., Dittmann, C., & Knüpffer, H. (2015). GBIS: the information system of the German Genebank. *Database*, *2015*, bav021.

[4] Weise, S., Lohwasser, U., & Oppermann, M. (2020). Document or lose it—On the importance of information management for genetic resources conservation in genebanks. *Plants*, *9*(8), 1050. https://doi.org/10.3390/plants9081050

[5] Weise, S., Oppermann, M., Maggioni, L., van Hintum, T., & Knüpffer, H. (2017). EURISCO: The European search catalogue for plant genetic resources. *Nucleic Acids Research*, *45*(D1), D1003-D1008. https://doi.org/10.1093/nar/gkw755

[6] Kotni, P., van Hintum, T., Maggioni, L., Oppermann, M., & Weise, S. (2023). EURISCO update 2023: the European Search Catalogue for Plant Genetic Resources, a pillar for documentation of genebank material. *Nucleic Acids Research*, *51*(D1), D1465-D1469. https://doi.org/10.1093/nar/gkac852

[7] Alercia, A.; Diulgheroff, S.; Mackay, M. (2015) FAO/Bioversity Multi-Crop Passport Descriptors V.2.1 [MCPD V.2.1] - December 2015. Bioversity International, 11 p.

[8] Akparov, Z. I., Mammadova, S. A., & Mammadov, A. T. (2013). Mobilization and conservation of the seed pool of plant genetic resources in Azerbaijan. *Russian Journal of Genetics: Applied Research*, *3*(1), 26-29. https://doi.org/10.1134/S2079059713010024

[9] Akparov, Z. I., Mammadova, S. A., Kalantarova, N. S., Mirzaliyeva, I. A., & Mammadov, A. T. (2025). National genebank of Azerbaijan. In *Plant Gene Banks: Genetic Resources Collections, Conservation and Sustainable Utilization* (pp. 1-44). Singapore: Springer Nature Singapore. https://doi.org/10.1007/978-981-99-4236-7_31-1

[10] Mirzaliyeva, I. (2024). Characterization Data of Cereal, Legumes and Technical Plants by the International Descriptors Lists. *International Journal of Innovative Approaches in Agricultural Research, 8*, 85–93. https://doi.org/10.29329/ijiaar.2024.1049.3

[11] Mirzaliyeva, I.A., Asadova, A.I. & Akparov, Z.I. (2017). Characterization and evaluation data of some leguminous plants stored in the National Genebank. *International Journal of Innovative Approaches in Agricultural Research, 1*, 30–39

[12] Engels, J. M., Ebert, A. W., & van Hintum, T. (2024). Collaboration between private and public genebanks in conserving and using plant genetic resources. *Plants*, *13*(2), 247. https://doi.org/10.3390/plants13020247

[13] Hanson, J., Jamora, N., & Bramel, P. (2026). Identifying gaps and challenges for conserving crop diversity in genebanks: Lessons from the reviews of national genebanks. *Plant Genetic Resources*, *24*(4), 245-257. https://doi.org/10.1017/S1479262125100427

Journal of Molecular Biosciences and Engineering

Review Article

Cancer Genetics: Comprehensive Analysis of Genetic Variants and Molecular Biomarkers Associated with Cancer Progression and Patient Survival

Madina Hamidova [1] and Saltanat Agayeva[1]

[1]Department of Natural Sciences, School of Innovative Engineering and Natural Sciences, Western Caspian University, Baku, AZ1072, Azerbaijan

Received: 23.09.2026 Accepted: 19.10.2026 Published: 30.10.2026

https://doi.org/10.54414/CWVN5915

Copyright: © 2026 by the authors. Licensee: Journal of Molecular Biosciences and Engineering, Western Caspian University, Baku, Azerbaijan. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution 4.0 International License (CC BY 4.0).

Abstract

The development and expansion of cancer are mainly due to heterogeneous genetic structures caused by somatic mutations and several epigenetic changes. here, the expansion and development of the tumor cell and at the same time how long the metastatic artis and cancer patients can survive and at the same time the molecular markers that determine this are seriously evaluated. In addition, the transformation of malignant cells and the basic changes that occur in oncogenes and cells, as well as indels and point mutations, are discussed in a broad way. One of the researched areas is the effect of polymorphism on cancer cells and their treatment, its instability, mutational burden, and transcriptomic expressions; and the use of biomarkers for various purposes, such as prognostic and predictive purposes, is mainly investigated. In addition, many studies- next-generation sequencing, finding biomarkers of genome association studies, and new changes in the identification of genes up to the molecular level are also included. In order to improve the treatment results of cancer patients and to optimize the treatment, the correlation of multi-omic data with medical information is widely used.

Keywords: cancer genetics, germline variants, tumor progression, patient survival, molecular biomarkers, somatic mutations.

1. Introduction

Cancer is considered a very dangerous disease that has caused the death of many people in the world, and it is said that it is related to the cases of genetic disorders or mutations that usually occur in normal cells. Malignant tumor transformation depends on changes in 2 types of genes. Oncogenes can be activated as a result of many changes, which can be caused by genetic mutation and a number of epigenetic changes. However, despite this, cis suppressor genes can fully regulate cell proliferation under normal conditions and maintain the overall stability of the genome [1]. Although Knudson's '2-hit' model has taken a number of important steps to explain information about the loss of activity of sis genes, subsequent studies have shown that inactivation of sis genes can occur by more complex means, such as insufficiency and negative dominant effect [2]. Recently, the development of the next-generation sequencing method and the genetic characteristics of different species of shark have been possible. Essentially, The Cancer Genome Atlas has revealed somatic mutations, genetic structural differences, and changes in gene expression in various ways in many malignant tumors [3]. This type of research has helped to find many changes, including insertions, deletions, and chromosomal anomalies. These differences mainly manifested themselves in several genes - oncogenes such as EGFR, KRAS, and BRCA1, BRCA2 in tumor cells [1], [4]. The genetic basis of the risk of cancer is derived from many elements, cloned sis consists of differences and germlines that increase susceptibility to sis. It has been investigated that hereditary genetic cancer risk and cancer treatment can have a

high impact on mammary gland and ovarian cancer and Lynch syndrome [4], [5]. Recent studies show that germline genetic differences have a 20% chance of ovarian cancer and 5% of endometrial cancer [6]. In addition, molecular biomarkers have an important role in the diagnosis and treatment of cancer. Biomarkers can be used as more useful tools than previously thought; such biomarkers include microsatellite instability, cis mutational burden, and gene expression markers. Through this, stratification of patients and making therapeutic decisions can be easier [7], [8].

Liquid biopsy has emerged as a minimally invasive method for the genetic assessment of tumor cells in the blood, as well as for monitoring treatment responses and the changes that occur during therapy [7]. Tumor development primarily arises from the progressive accumulation and interaction of numerous genetic alterations during cancer evolution. Differences in genetic characteristics among various cancer cell subpopulations within the same tumor can lead to the formation of tumor heterogeneity. Such genetic diversity can influence a range of clinically significant characteristics, including tumor aggressiveness, metastatic potential, risk of recurrence, and response to medical treatments [9]. Furthermore, treatment-resistant subclones may be present within the tumor even before therapy is initiated. During treatment, these resistant cell populations can rapidly multiply, ultimately leading to treatment failure and, in some cases, disease progression and death. Genomic profiling has become increasingly important in determining the molecular characteristics that underlie tumor behavior and influence clinical treatment outcomes. Among the genomic alterations identified to date, driver mutations are considered the primary molecular anomalies. The reason for this is considered to be their role in the disruption of key signal transduction pathways associated with aberrant proliferation and tumor development. Constitutive activation of the RAS-RAF-MEK-ERK and PI3K-AKT-mTOR signaling pathways can accelerate metastasis by completely halting apoptotic activity in malignant tumors [10].

As a result, genetic alterations affecting KRAS and BRAF have attracted particular attention. The primary reason for this is that these alterations can significantly influence the efficacy of molecularly targeted therapies. Moreover, genetic alterations affecting DNA damage response and repair pathways, particularly in genes such as BRCA1, BRCA2, ATM, and PALB2, may contribute to cancer susceptibility and influence the response of tumor cells to specific therapeutic approaches [11]. The disruption of DNA repair mechanisms in cancer cells can lead to the emergence of molecular vulnerabilities. This disruption may create opportunities for the development of more selective and effective treatment methods.

2. Genetic Variants in Cancer & Somatic Mutations

Mutations occurring in somatic cells lead to genetic changes that play a decisive role in the onset and development of cancer in humans. This process can occur in various cells in humans throughout their lives [12]. First of all, they arise from various intracellular processes, such as anomalies occurring during DNA replication or problems caused by oxidative stress. However, such changes can also occur due to the influence of factors such as smoking and intense ultraviolet (UV) radiation [13].

Driver and passenger mutations differ significantly from each other in terms of their mutational mechanisms. Thus, driver mutations contribute to the survival of cancer cells by delaying programmed cell death and activating cellular processes associated with proliferation, growth, metastasis, and tumor formation. Unlike driver mutations, passenger mutations are considered to be of minor biological significance. Since they do not directly participate in tumor development, they are understood to have a limited functional impact on cancer progression [14]. The frequency and nature of somatic mutations can vary significantly depending on the tissue type. These changes reflect differences in environmental impacts and tissue-related biological processes [14]. These changes reflect differences in environmental impacts and in the biological processes occurring within the tissue [14]. In this context, the importance of comprehensive genomic profiling in the assessment and clinical management of cancer is emphasized [15].

Genetic abnormalities can disrupt a range of cellular processes by altering the structure or expression of proteins involved in the regulation of cell proliferation, division, and survival, ultimately affecting normal cellular functions.

A range of genetic alterations, including missense, nonsense, and frameshift mutations, along with chromosomal abnormalities, may alter gene expression or impair protein function. Thereby contributing substantially to the malignant transformation of normal cells. As a result of these genetic changes, a direct link is established between genomic instability and both the initiation and progression of cancer. Changes occurring in somatic cells can disrupt the activity of numerous genes that play a crucial role in fundamental biological processes such as apoptosis, DNA damage repair, cell cycle regulation, cell differentiation, and the maintenance of genomic stability. Mutations affecting the activity of genes involved in these mechanisms can disrupt normal regulatory pathways, thereby promoting uncontrolled and abnormal cell proliferation. Alterations in the oncogene activation mechanism can trigger sustained signal transduction pathways that promote intensive cell proliferation. Otherwise, the complete inactivation of tumor suppressor genes could disrupt the functioning of essential cellular mechanisms that ensure cell division and the maintenance of genomic integrity [16]. The functional mechanism of a genetic alteration is determined by the characteristics of the affected gene, the precise location of the mutation within the genome, and its effects on protein synthesis and transcriptional regulation.

The fundamental essence of the clonal evolution of tumors is characterized by the accumulation of somatic mutations. As cancer cells progress through the stages of proliferation, new genetic changes occur within them. This results in the emergence of genetically distinct subpopulations within the same tumor. Sometimes, tumor subclones can possess specific biological advantages. These advantages include increased metastatic potential of cells and their proliferative capacity, as well as high resilience to environmental stress factors and to anti-cancer treatments [17].

3. Molecular Biomarkers

Germline variants arise from changes occurring in the cell of one of the parents. Specifically, the resulting alteration establishes heritable genetic differences that persist in all cells of the next generation, originating from the early stages of reproductive cell development [2]. These changes are observed in various forms, ranging from single nucleotide polymorphisms (SNPs) to larger-scale structural genomic alterations. In addition to susceptibility to cancer, this factor may also influence the course of the disease and the response to treatment [4].

Studies show that hereditary breast and ovarian cancer, Li-Fraumeni syndrome, and Lynch syndrome arise from mutations in genes such as BRCA1, BRCA2, and TP53, as well as in the mismatch repair genes MLH1 and MSH2 [5]. The impact of genetic variations on the risk of developing cancer depends primarily on the biological function of the genes in which the alterations occur. Furthermore, a link has been established between cancer development and the level of risk associated with the specific type of cancer.

Mutations in genes responsible for maintaining genome stability can be considered one of the key factors significantly increasing the likelihood of malignant tumor development in the organism. The BRCA1 and BRCA2 genes, which are responsible for the repair of DNA double-strand breaks, are considered key components of the homologous recombination pathway. Defects occurring during the repair process create an additional risk of genomic anomaly accumulation. This can lead to an increased likelihood of developing various malignant tumors, such as breast and ovarian cancers [18].

The presence of hereditary defects in DNA repair pathways reflects a molecular link between genetic predisposition and the development of cancer. However, the impact of inherited genetic alterations on the functioning of DNA mismatch repair systems is considered a key factor. Consequently, this process can lead to increased susceptibility to cancer. Lynch syndrome is considered one of the factors that significantly increase various health risks. Pathogenic germline variants in mismatch repair genes disrupting the DNA repair mechanism and increasing genomic instability is an example of this. This also leads to an increased risk of developing endometrial, colorectal, ovarian, and other malignant tumors [19]. Cancer cells arising from defects in DNA repair often exhibit microsatellite instability. This is considered a molecular characteristic of significant biological and clinical importance.

4. Conclusion

Significant strides have been made in cancer research through advances regarding molecular genetic variants and

biomarkers. These achievements have substantially broadened scientific understanding and perspectives concerning the molecular biology, pathogenesis, and clinical characteristics of cancer.

A comprehensive analysis of germline variants and somatic genomic alterations enables a more precise molecular characterization of tumors, the refinement of prognostic assessment mechanisms, and the development of personalized treatment strategies. Comprehensive analysis of germline variants and somatic genomic alterations enables a more precise molecular characterization of tumors. Such characterization lays the groundwork for improving the prognostic assessment of these alterations and developing personalized treatment strategies.

The application of key molecular biomarkers, such as tumor mutational burden (TMB), microsatellite instability (MSI), and gene expression profiles, in clinical practice is of great importance in medicine. In recent years, significant progress has been made using these markers to select optimal treatment approaches, refine diagnostic assessments, and more effectively evaluate treatment responses. This increasingly enhances the importance of these molecular markers in modern oncology. In particular, the use of molecular biomarkers plays a crucial role in identifying patients who may potentially benefit from immunotherapy and in making decisions regarding the selection of adjuvant systemic treatment approaches.

Advancements in the fields of cancer genetics, bioinformatics, and artificial intelligence will play a crucial role in the future integration of genomic and clinical data. In particular, the use of bioinformatics and artificial intelligence is expected to contribute to improvements in early disease diagnosis and treatment decision-making. This, in turn, will lead to significant progress in oncology. At the same time, attention must be paid to key aspects regarding the integration of molecular research findings into daily clinical practice. Specifically, integrating research results into routine clinical practice requires comprehensive clinical validation, as well as thorough consideration of multidisciplinary collaboration, ethical issues, and technical and practical limitations. Such achievements in scientific fields are accelerating the transition from traditional treatment to personalized therapy tailored to the patient's clinical indicators and the molecular-biological characteristics of the tumor.

Author Contributions

The sole author is responsible for conceptualizing, collecting, and analyzing data, and writing and editing the manuscript.

Conflict of Interest

The authors declare no conflicts of interest.

Funding

This research received no external funding.

References

[1] Wang, X., Shi, X., Liao, S., Su, T., Gu, Y., Zhang, H., & Li, Z. (2026). Multidimensional effects of genetic mechanisms in cancer occurrence, development, and treatment. *Cancer Genetics*, 304–305, 58–75. https://doi.org/10.1016/j.cancergen.2026.03.006

[2] de Voer, R. M., & Valle, L. (2026). Integrating germline and tumor sequencing to improve hereditary cancer diagnosis and care. *European Journal of Human Genetics*, 1-11. https://doi.org/10.1038/s41431-026-02046-5

[3] Ananda, H., Sahana, S. R., Murthy, S. R., Kunnath, A. N., Prashant, A., Kaifi, J. T., & Suvilesh, K. N. (2026). Genomic innovations in cancer prevention, diagnosis, prognosis and precision therapeutics. *Frontiers in Genetics*, *17*, 1828450. https://doi.org/10.3389/fgene.2026.1828450

[4] Zhu, H., Shao, J., Wang, Y., Gu, C., & Liu, J. (2026). Molecular landscape and clinical translation of DNA damage response alterations in solid tumors: A pan-cancer perspective on precision oncology. *Neoplasia*, *77*, 101315. https://doi.org/10.1016/j.neo.2026.101315
[5] Agüera-Sánchez, A., Peña-Ros, E., Martínez-Martínez, I., & García-Molina, F. (2026). Comprehensive landscape of diagnostic, prognostic and predictive biomarkers in colorectal cancer: From genomics to multi-omics integration in precision medICIne. *Journal of Personalized Medicine*, *16*(1), 48. https://doi.org/10.3390/jpm16010048
[6] Watanabe, T., Soeda, S., Okoshi, C., Fukuda, T., Yasuda, S., & Fujimori, K. (2023). Landscape of somatic mutated genes and inherited susceptibility genes in gynecological cancer. *Journal of Obstetrics and Gynaecology Research*, *49*(11), 2629-2643. https://doi.org/10.1111/jog.15766
[7] Basheeruddin, M., Qausain, S., Anjankar, A., & Khan, F. I. (2026). Advancements in cancer biomarker discovery over fifteen years: diagnostics and prognostic approach in somatic cancer. *Oncology Reviews*, *20*, 1809836. https://doi.org/10.3389/or.2026.1809836
[8] Fisher, P. B., Tew, K. D., Kumar, U., & Das, V. (2026). Biomarkers for cancer screening, diagnosis and targeted therapeutic approaches. Elsevier.
[9] McGranahan, N., & Swanton, C. (2017). Clonal heterogeneity and tumor evolution: past, present, and the future. *Cell*, *168*(4), 613-628.
[10] Hanahan, D. (2022). Hallmarks of cancer: new dimensions. *Cancer Discovery*, *12*(1), 31-46.
[11] Lord, C. J., & Ashworth, A. (2016). BRCAness revisited. *Nature Reviews Cancer*, *16*(2), 110-120.
[12] Yoshida, K. (2026). Somatic mutations and clonal evolution in normal tissues and cancer development. *Experimental & Molecular Medicine*, *58*(4), 961. https://doi.org/10.1038/s12276-025-01592-0
[13] Ng, A. S., & Chan, D. K. H. (2023). Commonalities and differences in the mutational signature and somatic driver mutation landscape across solid and hollow viscus organs. *Oncogene*, *42*(37), 2713-2724. https://doi.org/10.1038/s41388-023-02802-7
[14] Jin, C., Li, W., Liu, B., Cao, L. Q., Stefan, S. M., Yuan, L., & Yu, H. (2026). Emerging trends and converging evidence in tumor evolution: A comprehensive review. *Drug Resistance Updates*, 101380. https://doi.org/10.1016/j.drup.2026.101380
[15] O'Malley, D. E., Raspin, K., Melton, P. E., Burdon, K. P., Dickinson, J. L., & FitzGerald, L. M. (2024). Acquired copy number variation in prostate tumours: a review of common somatic copy number alterations, how they are formed and their clinical utility: Genetics and Genomics. *British Journal of Cancer*, *130*(3), 347-357. https://doi.org/10.1038/s41416-023-02485-7
[16] Vogelstein, B., Papadopoulos, N., Velculescu, V. E., Zhou, S., Diaz Jr, L. A., & Kinzler, K. W. (2013). Cancer genome landscapes. *science*, *339*(6127), 1546-1558. https://doi.org/10.1126/science.1235122
[17] Greaves, M., & Maley, C. C. (2012). Clonal evolution in cancer. *Nature*, *481*(7381), 306-313. https://doi.org/10.1038/nature10762
[18] Kuchenbaecker, K. B., Hopper, J. L., Barnes, D. R., Phillips, K. A., Mooij, T. M., Roos-Blom, M. J., ... & BRCA1 and BRCA2 Cohort Consortium. (2017). Risks of breast, ovarian, and contralateral breast cancer for BRCA1 and BRCA2 mutation carriers.
[19] Lynch, H. T., & De la Chapelle, A. (2003). Hereditary colorectal cancer. *New England Journal of Medicine*, *348*(10), 919-932. https://doi.org/10.1056/NEJMra012242

Journal of Molecular Biosciences and Engineering

Review Article

Early Blood-Based Diagnostic Criteria in Cardiovascular Diseases

Natavan Karamova [1] ✉ **and Irina Amiraslanova** [1] ✉

[1]Department of Natural Sciences, School of Innovative Engineering and Natural Sciences, Western Caspian University, Baku, AZ1072, Azerbaijan

Received: 25.09.2026 Accepted: 20.10.2026 Published: 30.10.2026

https://doi.org/10.54414/YPJU1653

Copyright: © 2026 by the authors. Licensee: Journal of Molecular Biosciences and Engineering, Western Caspian University, Baku, Azerbaijan. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution 4.0 International License (CC BY 4.0).

Abstract

The present era is characterized not only by an increase in the number of diseases but also by their occurrence at younger ages. Modern medical advances are sometimes unable to prevent an increase in mortality. Naturally, the growing number of diseases also directly depends on social and environmental, medical and biological, ecological, and other factors. In such a period, early diagnosis of diseases means successful treatment and longer human life. Cardiovascular diseases are among the most serious public-health problems both worldwide and in Azerbaijan. According to statistical data, cardiovascular diseases account for a large proportion of deaths in the country. The results of blood tests performed at Memorial Clinic in April 2026 once again confirm the high prevalence of these diseases and show that approximately 80% of the examined individuals suffer from them. A comparative analysis of the results of patients who underwent troponin testing revealed elevated levels mainly in elderly men. This is a key diagnostic criterion for myocardial infarction and other acute heart diseases. Triglyceride and LDL test results were also elevated mainly in middle-aged individuals. The same change in HDL levels was observed in all women and men who underwent blood testing. The obtained indicator confirms a high risk of ischemic heart disease. Modern lifestyles and stress factors also contribute to the spread of the disease. Early diagnosis and preventive measures play an important role in preventing complications. Timely examinations can significantly reduce the risk of myocardial infarction and stroke. In this respect, the presented article may be regarded as a step toward protecting human health and is statistically significant.

Keywords: CVD, IHD, troponin, LDL, HDL, triglyceride, physiological age.

1. Introduction

Despite the fundamental achievements of medical science and technological progress in the modern era, humanity continues to struggle with cardiovascular diseases (CVDs), which remain among the most serious threats to human health. In the world today and in our country, pathologies of the cardiovascular system rank among the leading causes of death and disability. According to official World Health Organization (WHO) data, approximately 32% of all deaths each year, or 17.9 million lives, are lost because of this group of pathologies. Global environmental transformations, the intensification of anthropogenic effects, the impact of poor nutrition on metabolism, chronic distress factors that disrupt homeostatic adaptation mechanisms, and the sharp decline in daily physical activity are the main triggers accelerating this group of diseases. Cardiovascular pathologies, previously encountered mainly among older generations, are now rapidly becoming widespread among younger people as well. This indicates a “rejuvenation” of CVDs. The methodological opportunities provided by modern molecular biology, genetics, bioinformatics, and other medical sciences have demonstrated that the fundamental basis of many CVDs is hereditary predisposition, specific gene polymorphisms, pathological changes in biochemical signaling

pathways, or other similar genetic disorders. The prevalence of cardiovascular pathologies in Azerbaijan is analyzed mainly by region. In highly urbanized areas, such as Baku and the Absheron Peninsula, the incidence of CVDs is higher than in mountainous regions.

Cardiovascular diseases are a broad group of pathologies that are among the most prevalent and fatal diseases worldwide and encompass structural and functional disorders of the heart, the principal organ of the cardiovascular system, and of the blood vessels extending from it to organs and tissues. According to the latest WHO statistics, approximately 32% of deaths each year, or 17.9 million lives, are lost because of this group of pathologies. CVDs are most frequently encountered in large, developed countries. This is due to expanding urbanization, unhealthy nutrition, and other factors. Although cardiovascular diseases were formerly observed only in older individuals, they are now also encountered in young people. This phenomenon is referred to as the "rejuvenation" of the disease.

According to modern medicine, cardiovascular diseases rank first among causes of death worldwide. The principal causes of cardiovascular diseases include atherosclerosis, arterial hypertension, diabetes mellitus, changes in the rheological properties of blood, and others. A sedentary lifestyle, or physical inactivity, also leads to CVD. Based on global prevalence and high mortality, CVDs are classified as follows:

1. Ischemic heart disease (IHD): the most common form within the CVD group;

2. Cerebrovascular diseases: the second leading cause of death within the CVD group. Cerebrovascular disease has two main forms: ischemic stroke (thrombosis of cerebral vessels) and hemorrhagic stroke (intracerebral bleeding caused by rupture of a vessel).

3. Peripheral arterial disease: although direct mortality is low, these patients have a high risk of stroke and myocardial infarction;

4. Rheumatic heart disease: streptococcal infection causes damage to the heart valves. This form is regarded as one of the causes of death and disability at a young age;

5. Congenital heart defects: congenital heart defects account for one-third of all forms of CVD;

6. Arrhythmias;

7. Heart failure;

8. Deep vein thrombosis (DVT), etc. [1], [2].

Ischemic heart disease (IHD): in modern medicine, elevated levels of "bad" cholesterol in the coronary arteries that supply the heart muscle cause narrowing or complete blockage of the vessels. Consequently, blood flow slows, and an imbalance develops between the oxygen delivered to the heart and the heart's oxygen demand. Among CVDs, ischemic heart disease (IHD) ranks first among conditions that most frequently cause disability and death.

IHD differs markedly across age groups. According to WHO data, there are 10 deaths per 100,000 people aged 25–34 years, while this figure rises to 1,000 in the 55–64-year age group. IHD is more common in men than in women [3], [4]. When the degree of coronary artery narrowing exceeds 50%, IHD manifests clinically; that is, clinical symptoms appear. In addition to narrowing of the coronary arteries due to "bad" cholesterol, other factors—including smoking, alcoholism, consumption of unsaturated fats, physical inactivity, bronchial asthma, diabetes mellitus, hypothyroidism, gallstone disease, and others—also play an important role in the development of IHD [4], [5], [6].

Numerous clinical studies demonstrate that biomarkers such as atherogenic lipoproteins, fibrinogen, homocysteine, and TSH (thyroid-stimulating hormone) correlate closely with major risk factors such as dyslipidemia, hypertension (high blood pressure), obesity, and diabetes mellitus. At the same time, assessing inflammatory indicators such as C-reactive protein (CRP) together with standard biochemical tests makes it possible to predict disease risk more accurately. Specific markers such as high-sensitivity troponin T (hsTnT) and NT-proBNP play a major role in the early diagnosis of heart failure and myocardial infarction. These substances begin to rise above normal blood levels only 1–2 hours after an acute cardiovascular event [7], [8]. The most

important markers of myocardial injury are troponin T and troponin I. An increase in blood TnT and TnI above normal levels leads to increased pressure in the right ventricle and, consequently, acute myocardial injury. An elevated serum troponin I (TnI) level in patients with cardiac rhythm disorders is not merely a laboratory indicator; it is also a clinical warning that the patient may experience a stroke or myocardial infarction at any time. Serum troponin is considered one of the principal markers indicating the latent development of vascular atherosclerosis [9], [10].

The lipid profile is of great importance in CVD. Lipoproteins are complex molecules that transport lipids such as cholesterol and triglycerides throughout the body in the bloodstream and consist of hydrophobic and hydrophilic layers. A lipoprotein is a spherical molecule with cholesterol esters and triglycerides inside and an outer covering composed of protein and membrane components. Fat particles are covered externally by a protein layer. This causes them to aggregate and obstruct blood vessels. The ratio of protein to fat determines whether the lipoproteins are HDL or LDL, that is, their type.

In a healthy individual, the optimal LDL level should be below 100 mg/dL. If this level rises to 100–129 mg/dL (2.6–3.3 mmol/L), it is considered a risk factor. Unlike other disorders of lipid metabolism, particularly familial hypercholesterolemia manifested by high cholesterol levels, atherosclerosis or coronary heart disease does not necessarily develop in every patient with acute hypertriglyceridemia or chylomicronemia syndrome. Initially, this paradox raised doubts about the role of triglycerides in cardiovascular pathologies; however, it was explained by the fact that at extremely pathological concentrations (triglycerides >50 mmol/L), triglyceride-rich lipoproteins are too large to enter the inner layer of the vessels and therefore cannot cause atherosclerosis. Conversely, at mildly and moderately elevated pathological triglyceride concentrations (2–10 mmol/L), lipoprotein particles are small enough to penetrate the arterial intima; consequently, they accumulate in the tunica intima and have the potential to cause atherosclerosis. For this reason, clinical epidemiology focuses primarily on subclinical elevations in triglycerides. Triglyceride levels are higher in men than in women. However, when women and men with cardiovascular diseases were compared, it became clear that at the maximum triglyceride level, affected women had a higher risk of developing cardiovascular pathologies than men. Analysis of blood tests in women and men of different ages and investigation of their role in the early diagnosis of CVD. The purpose of the study was to analyze the test results affecting CVD according to the number, sex, and age of the patients

2. Materials and Methods

Various scientific methods were used in the study. Theoretical analysis and synthesis were applied to systematically examine and generalize the available scientific literature on the topic. Comparative and pathophysiological analyses were conducted to assess the diagnostic significance of specific biochemical blood biomarkers that play an important role in the early diagnosis of cardiovascular diseases (cardiac troponins, high-sensitivity C-reactive protein [hs-CRP], lipid profile, and other metabolic indicators). Statistical methods were used to analyze the incidence of pathological conditions and the correlations among biomarkers across various indicators. In addition, the study and systematization of clinical and laboratory data made it possible to obtain practical findings aimed at detecting cardiovascular diseases at an early stage.

The study was conducted on the basis of laboratory results from Memorial Clinic. Venous blood samples were collected from patients who attended the clinic in April 2026, depending on the type of test prescribed. The samples obtained from patients were analyzed using biochemical and immunochemical analyzers. To maintain the accuracy of the study, particular attention was paid to the pre-analytical stage, which is the initial stage of the investigation. During the pre-analytical stage, blood samples were centrifuged at 35,000 rpm for 10 minutes to separate the serum. The patients' lipid profiles (triglycerides, HDL, LDL) and enzyme activity (AST, ALT) were determined on a fully automated biochemical analyzer using enzymatic-colorimetric and kinetic UV methods. To assess acute myocardial injury and inflammatory processes, troponin and hs-CRP markers were analyzed on a fully automated immunochemical analyzer using electrochemiluminescence immunoassay (ECLIA) and immunoturbidimetric methods, respectively.

3. Results

Analysis of the results of the scientific study. The blood test results of patients who attended Memorial Clinic are presented after division into different groups:

Analysis of troponin values

Troponin I and T are among the most important markers of myocardial injury. Even a slight change in the amount of troponin in the blood indicates narrowing of the cardiac vessels, because troponin T and I are regulatory proteins involved in the actin–myosin interaction in the heart muscle. Therefore, in modern laboratories, serum troponin T and I levels are considered the "gold standard" for CVDs, especially for the timely detection of myocardial infarction. During the study, 285 patients underwent troponin testing at Memorial Hospital in April 2026 (Figure 1).

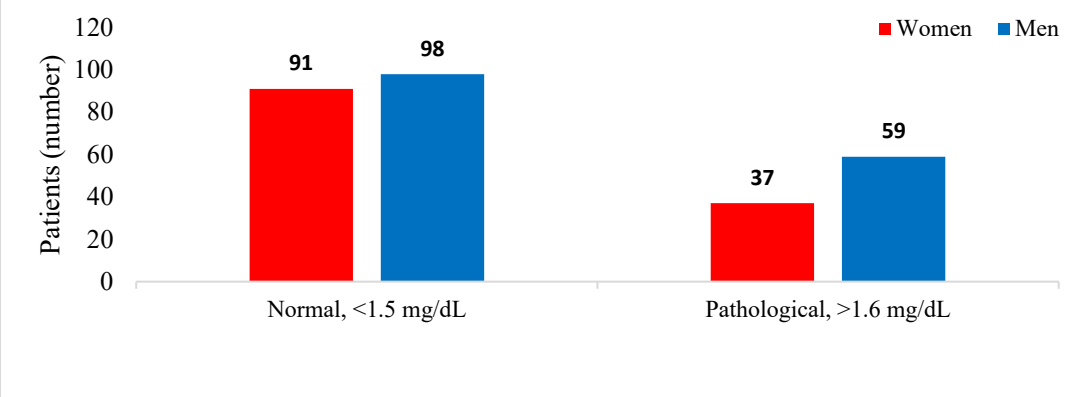


Figure 1. Comparative presentation of the test results of patients who underwent troponin testing at Memorial Clinic in April 2026.

Of the tests, 128 belonged to women and 157 to men. Among the 128 female patients, 91 had results below 1.5 mg/dL, that is, within the normal range, while 37 had results above 1.5 mg/dL, indicating a pathological value. Among the 157 male patients, 98 had results within the normal range, and 59 had pathological values. The number of male patients in the pathological group was greater than the number of female patients in that group. Analysis by physiological age showed that the patients' ages ranged from 15 to 77 years. According to physiological age, the patients were divided into two groups: Group I included 92 individuals aged 15–35 years. Troponin levels were <1.5 mg/dL in 71 individuals (33 women and 38 men) and >1.5 mg/dL in 21 individuals (8 women and 13 men). The number of patients in Group II exceeded that in Group I. In Group II, among patients aged 36–77 years, troponin levels were <1.5 mg/dL in 118 individuals (58 women and 60 men) and >1.5 mg/dL in the remaining 75 individuals. Analysis of the results showed that the measured indicator was higher in male patients, which is a factor that increases the risk of CVD.

Analysis of patients based on triglyceride test values

It is known from the literature that an increase in the amount of triglycerides in a patient's blood indicates a high risk of myocardial infarction. Therefore, assessing triglycerides is one of our primary objectives in blood-test analysis. Interesting findings were obtained in studies conducted at Memorial Clinic (Figure 2), where 309 patients underwent triglyceride testing. Of these tests, 149 belonged to women and 160 to men. In 124 examined patients (64 women and 60 men), the triglyceride level was 70–150 mg/dL, which is considered within the normal range. The physiological age groups of patients whose triglyceride levels were within the normal range were as follows: 1–25 years: 27 individuals (15 women and 12 men); 26–45 years: 58 individuals (31 women and 27 men); 46–65 years: 26 individuals (14 women and 12 men); and 66–80 years: 13 individuals (5 women and 8 men).

Pathological triglyceride levels were also observed. Overall, 71 patients (33 women and 38 men) had triglyceride levels of 150–190 mg/dL, which is considered the borderline range. In medicine, the borderline range is also called the "gray zone." Grouping by physiological age showed 4 patients aged 1–25 years (2 women and 2 men), 17 patients aged 26–45 years (8 women and 9 men), 21 patients aged 46–65 years (10 women and 11 men), and 29 patients aged 66–80 years (13 women and 16 men).

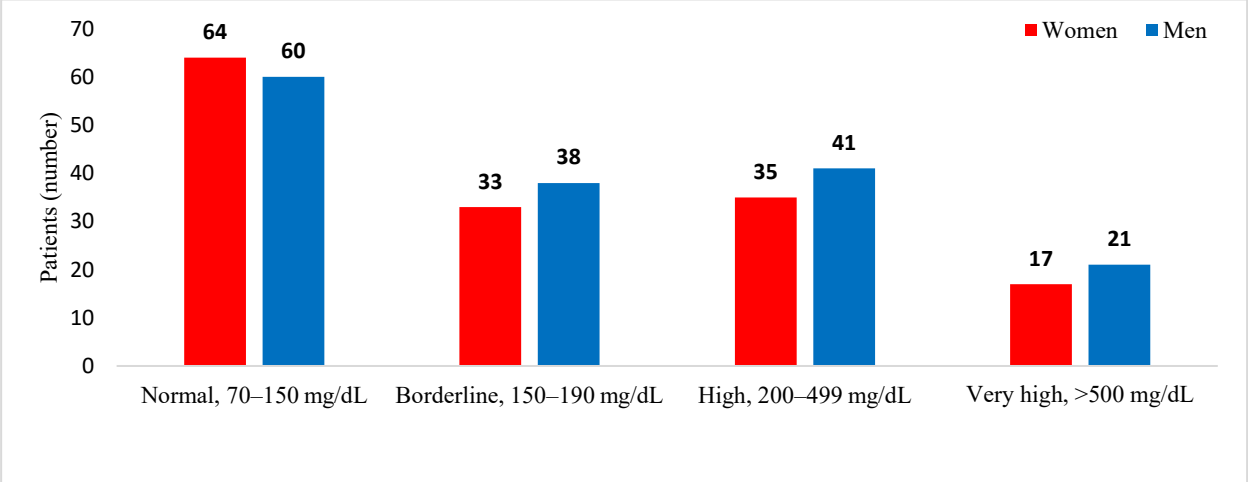


Figure 2. Results of 309 patients who underwent triglyceride testing at Memorial Clinic in April 2026.

An elevated triglyceride level is one of the risk factors. Thus, 76 patients (35 women and 41 men) had high triglyceride levels. Grouping them by physiological age showed 1 patient aged 1–25 years (a man), 18 patients aged 26–45 years (6 women and 12 men), 26 patients aged 46–65 years (12 women and 14 men), and 31 patients aged 66–80 years (15 women and 14 men).

Among the 309 patients studied, 38 had very high triglyceride values. Of these results, 17 belonged to women and 21 to men. When divided into physiological age groups, very high triglyceride values were identified in 6 individuals aged 26–45 years (2 women and 4 men), 23 individuals aged 46–65 years (6 women and 7 men), and 19 individuals aged 66–80 years (9 women and 10 men).

Thus, comparative analysis of the obtained results showed that pathological values of the studied indicator were more common at advanced ages and particularly among male patients, which increases the risk of myocardial infarction and CVD.

Analysis based on HDL test values

Lipid disorders and atherosclerosis are also major risk factors that cause pathologies of the cardiovascular system. The principal function of HDL in the human body is to remove excess cholesterol particles from the vessel wall and transport them to the liver. For this reason, HDL is also called "good cholesterol" in medicine. In HDL disorders, excess cholesterol particles accumulated in the vessel wall are not removed, and an atherosclerotic plaque forms, resulting in the development of CVDs (Figure 3).

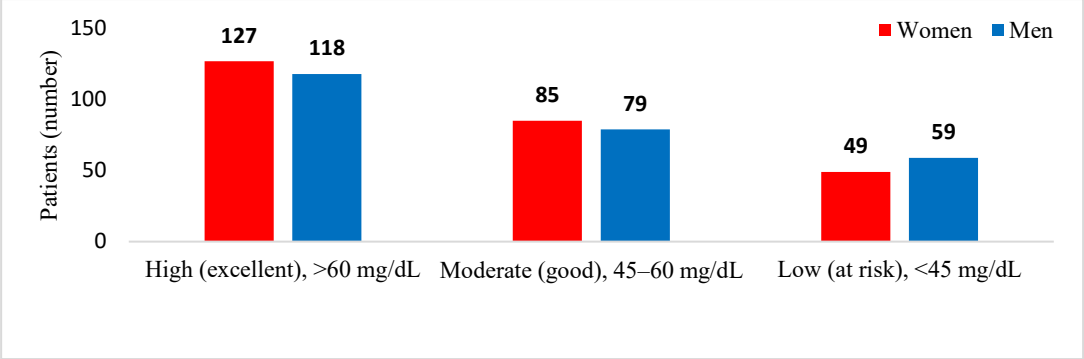


Figure 3. Graphical presentation of the test results of 517 patients who underwent HDL testing at Memorial Clinic in April 2026.

Of the 517 patients who underwent HDL testing, 261 were women, and 259 were men. In 245 individuals (127 women and 118 men), the HDL result was above 60 mg/dL, which is a good result. Patients with excellent results were divided into four physiological age groups: 64 individuals aged 1–25 years (34 women and 30 men), 78 individuals aged 26–45 years (41 women and 37 men), 58 individuals aged 46–65 years (27–29), and 47 individuals aged 66–85 years (25 women and 22 men). Thus, among the 517 patients who attended Memorial Clinic, HDL-related protection of the cardiovascular system was observed in 142 individuals aged 1–45 years.

The test results of 164 patients (85 women and 79 men) showed moderate HDL levels, and certain somatic diseases (type 2 diabetes, chronic kidney disease, etc.) were identified. The physiological age groups of these patients were as follows: 41 individuals aged 1–25 years (19 women and 22 men), 55 individuals aged 26–45 years (28 women and 27 men), 40 individuals aged 46–65 years (21 women and 19 men), and 28 individuals aged 66–85 years (17 women and 11 men).

In 108 of the examined patients (49 women and 59 men), the HDL level was below 45 mg/dL. These patients have a very high probability of developing cardiovascular diseases, and all belong to the risk group. Distribution by physiological age was as follows: only 2 male patients were aged 1–25 years; 26 individuals were aged 26–45 years (11 women and 15 men); 35 individuals were aged 46–65 years (16 women and 19 men); and 45 patients were aged 66–85 years (22 women and 23 men). Thus, comparative analysis showed that HDL levels were approximately equal in women and men and differed markedly from the norm across all age groups. The obtained indicator demonstrates a high risk of CVD.

Investigation of blood LDL levels

In medical terminology, LDL is also called “bad cholesterol.” LDL is formed when VLDL loses most of its fat content. LDL transports cholesterol from the liver to peripheral tissues. An elevated blood LDL level is a principal indicator of cardiovascular pathologies (Figure 4).

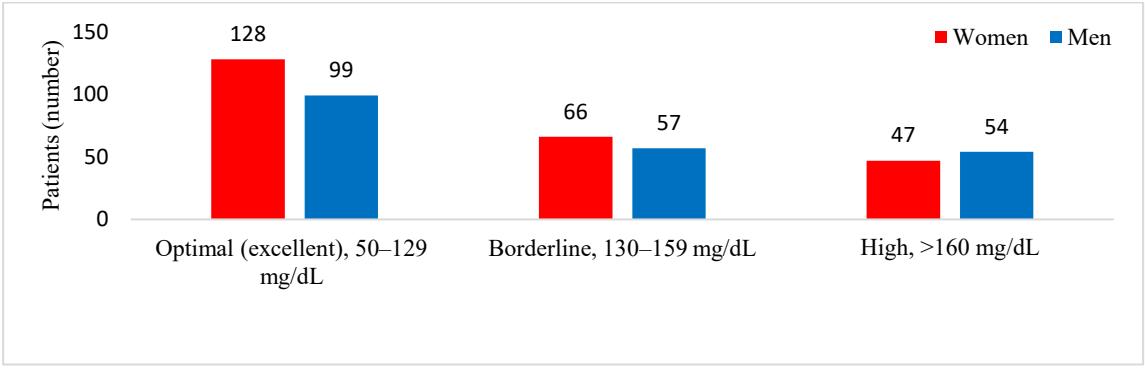


Figure 4. Results of the investigation of blood LDL levels in 451 patients tested at Memorial Clinic in April 2026.

In April, 451 patients (241 women and 210 men) underwent LDL testing at Memorial Clinic. In 227 of these patients (128 women and 99 men), the test result was 50–129 mg/dL, which is considered the normal range. Values within the normal range were recorded most frequently among patients aged 1–45 years (134 individuals) and in 93 individuals aged 46–85 years. These figures show that blood LDL levels increase with age. In 123 patients (66 women and 57 men), the test result ranged from 130 to 159 mg/dL, which is considered the borderline range. Of the patients in this range, 86 were aged 46–85 years. In 101 patients (47 women and 54 men), the test result was above 160 mg/dL. The vascular structure is altered in these patients, which may lead to atherosclerosis. The largest number of patients was in the 66–85-year age group (54 individuals: 23 women and 31 men).

Thus, analysis of LDL levels showed higher values among male patients. When taking the patients' life histories (anamnesis vitae), we determined that alcoholism was one of the principal reasons for the predominance of men. This increases the risk of atherosclerosis, IHD, and other cardiovascular diseases.

Cardiovascular diseases are considered one of the most serious public-health problems in Azerbaijan. According to statistical data, cardiovascular diseases account for a large proportion of deaths in the country. Approximately 60% of all deaths in Azerbaijan are associated with cardiovascular diseases. In recent years, increases have been observed in arterial hypertension, ischemic heart disease, and myocardial infarction. Cardiovascular diseases are widespread both among people of working age and among the elderly population. Heart and vascular diseases rank first among causes of death in the country. Unhealthy nutrition, physical inactivity, smoking, and excess weight are considered important risk factors in the development of these diseases. High blood pressure and high blood cholesterol increase the risk of developing cardiovascular diseases. According to specialists, part of the population is unaware of their blood pressure and does not undergo regular medical monitoring. Modern lifestyles and stress factors also contribute to the spread of the disease. Early diagnosis and preventive measures play an important role in preventing complications. Timely examinations can significantly reduce the risk of myocardial infarction and stroke. State programs and awareness-raising measures aimed at preventing cardiovascular diseases are being implemented in Azerbaijan. Nevertheless, the prevalence of the disease keeps the problem relevant. Cardiovascular diseases are regarded not only as a medical problem but also as a socioeconomic one. Early detection and effective prevention of these diseases are particularly important for improving the population's quality of life and reducing mortality. Promoting a healthy lifestyle in Azerbaijan is considered one of the main approaches to combating cardiovascular diseases. The study conducted once again demonstrated the high risk level of these diseases.

4. Conclusion

Cardiovascular diseases are considered one of the most serious public health problems in Azerbaijan. According to statistical data, cardiovascular diseases account for a large proportion of deaths in the country. The results of blood tests performed at Memorial Clinic in April 2026 confirm the high prevalence of the diseases and show that approximately 80% of the examined individuals suffer from them. Comparative analysis of the results of patients who underwent troponin testing at Memorial Clinic in April 2026 identified elevated levels mainly in elderly men. This is a key diagnostic criterion for myocardial infarction and other acute heart diseases. Triglyceride and LDL test results were also elevated mainly in middle-aged individuals. The highest incidence among men was recorded at advanced ages, which may be associated with harmful habits, an unhealthy lifestyle, stress, and other factors. The same change in HDL levels was observed in all women and men who underwent blood testing. This increases the risk of atherosclerosis, IHD, and other cardiovascular diseases. Modern lifestyles and stress factors also contribute to the spread of the disease. Early diagnosis and preventive measures play an important role in preventing complications. Timely examinations can significantly reduce the risk of myocardial infarction and stroke.

Author Contributions

The sole author is responsible for conceptualizing, collecting, and analyzing data, and writing and editing the manuscript.

Conflict of Interest

The authors declare no conflicts of interest.

Funding

This research received no external funding.

References

[1] Goldsborough, E., Tasdighi, E., & Blaha, M. J. (2023). Assessment of cardiovascular disease risk: a 2023 update. *Current opinion in lipidology*, *34*(4), 162-173. https://doi.org/10.1097/MOL.0000000000000887

[2] Lopez, E. O., Ballard, B. D., & Jan, A. (2023). Cardiovascular disease. In *StatPearls [Internet]*. StatPearls Publishing.

[3] Azizov, V. A., Hajiyeva, G. I., Gahramanova, S. M., Rustamova, Y. K., Mahammadli, S. M., Immanov, Q. G., Khalilov, S. J., Shiraliyeva, G. Sh., Ibadova, F. A. (2024). Cardiology. Baku, 416 p.

[4] Antman, E. M., & Braunwald, E. (2020). Managing stable ischemic heart disease. *New England Journal of Medicine*, *382*(15), 1468-1470.

[5] Hussain, S., Sattar, U., & Azhar, M. A. (2013). Risk factors for ischemic heart disease in southern Punjab. *Pakistan Heart Journal*, *46*(4).

[6] Carballo, D., Visseren, F. L., Mach, F., & Smulders, Y. (2022). The ‘ten commandments' for the 2021 ESC Guidelines on CVD prevention.

[7] Garady, L., Soota, A., Shouche, Y., Chandrachari, K. P., KV, S., Shankar, P., & Soota Sr, A. (2024). A narrative review of the role of blood biomarkers in the risk prediction of cardiovascular diseases. *Cureus*, *16*(12). https://doi.org/10.7759/cureus.74899

[8] Al-Hindawi, M. S., Mohsen, A. M., & Abdulameer, Z. K. (2026). Biochemical Markers for Early Detection of Cardiovascular Diseases. *JM Med Stu 2026*, *3*(1), 261-266. https://doi.org/10.51219/JMMS/Mohammed-s-al-hindawi/61

[9] Alaour, B., Liew, F., & Kaier, T. E. (2018). Cardiac Troponin-diagnostic problems and impact on cardiovascular disease. *Annals of Medicine*, *50*(8), 655-665. https://doi.org/10.1080/07853890.2018.1530450

[10] Jakubiak, G. K. (2024). Cardiac troponin serum concentration measurement is useful not only in the diagnosis of acute cardiovascular events. *Journal of Personalized Medicine*, *14*(3), 230. https://doi.org/10.3390/jpm14030230

Journal of Molecular Biosciences and Engineering

Research Article

Chronic Renal Complications and Their Risks in Thalassemic Patients Receiving Transfusion

Sahin Bayramli [1], Aysu Ismayilova [1], Aygun Suleymanova [1] and Shamsiyya Asgarli [2]

[1] Department of Natural Sciences, School of Innovative Engineering and Natural Sciences, Western Caspian University, Baku, AZ1072, Azerbaijan

[2] Thalassemia Division of the National Center of Hematology and Transfusion, Ministry of Health of the Republic of Azerbaijan

Received: 27.09.2026 Accepted: 26.10.2026 Published: 30.10.2026

https://doi.org/10.54414/XDTY5462

Copyright: © 2026 by the authors. Licensee: Journal of Molecular Biosciences and Engineering, Western Caspian University, Baku, Azerbaijan. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution 4.0 International License (CC BY 4.0).

Abstract

Our study aimed to describe the prevalence, main clinical features, and risk factors for renal and urinary tract complications in patients with thalassemia. This study assessed renal complications and their main clinical features in 177 thalassemic patients evaluated between 2025 and 2026 and compared the results with renal complications observed in sickle cell anemia. This study evaluated 177 patients with thalassemia examined between 2025 and 2026. Findings from renal ultrasonography alongside clinical data regarding age contribution, transfusion therapy, and detected renal pathologies were analyzed. Additionally, the clinical utility of clinical parameters and ultrasound findings in assessing renal function was reviewed. Finally, this study investigated the primary mechanisms of renal damage in thalassemia, highlighting key differences from the renal changes characteristic of sickle cell anemia.

Keywords: thalassemia, renal complications, microlithiasis, chronic transfusion, iron overload, renal function, eGFR, renal ultrasonography.

1. Introduction

Thalassemia is a group of hereditary hematological disorders caused by defects in the synthesis of hemoglobin globin chains. Severe forms of the disease require regular erythrocyte transfusions, which serve as the primary therapeutic approach to extend patients' lifespan. However, long-term transfusion therapy leads to systemic iron overload, resulting in tissue damage and ultimately organ failure. Among the organs affected, the kidneys are particularly vulnerable. Renal complications in thalassemic patients stem from multiple factors, including prolonged anemia, hypoxia, oxidative stress, and iron overload. Chronic anemia can induce renal hemodynamic alterations and early-stage hyperfiltration, whereas long-term disease progression may lead to tubular dysfunction, proteinuria, and other renal manifestations. In addition to iron toxicity, the use of certain iron chelators can also exert nephrotoxic effects.

Evaluating renal status in thalassemic patients relying solely on serum creatinine levels is insufficient. Comprehensive assessment—including estimated glomerular filtration rate (eGFR), urinary albumin and protein levels, calcium, phosphate, uric acid parameters, and renal ultrasonography—enables early detection of subtle renal changes.

Thalassemia patients require long-term blood transfusion therapy. Regular transfusions, along with accompanying

comorbidities, impair renal function. Tissue hypoxia, hemolytic processes, and iron overload play pivotal roles in the deterioration of renal function. Dysfunction of the renal tubules is among the primary drivers of the onset and progression of renal damage. Therefore, regular monitoring of the kidneys, ureters, and urinary bladder is essential in thalassemia patients receiving chelation therapy [1], [2].

Renal injury in thalassemic patients should be evaluated by considering both glomerular and tubular alterations. Chronic anemia in thalassemia leads to reduced oxygen delivery to the kidneys. The resulting hypoxic state negatively impacts renal hemodynamics and the filtration system. Consequently, compensatory hemodynamic adaptations emerge in thalassemic patients, manifesting in early stages as an elevated estimated glomerular filtration rate (eGFR)—a hyperfiltration phenomenon that serves as an early marker for the eventual development of renal failure. When the organism is exposed to prolonged, continuous hypoxic and hemodynamic alterations, progressive damage to the renal parenchyma ensues. Recurrent hemolytic events further exacerbate renal injury. During hemolysis in thalassemic patients, the breakdown of erythrocytes releases toxic cell debris and free hemoglobin, which impairs renal function and induces tissue injury. Furthermore, renal hypoxia adversely affects cellular energy metabolism. Decreased oxygen availability impairs mitochondrial oxygenation, leading to a marked reduction in ATP synthesis. In the setting of ATP depletion, intracellular accumulation of fluid and sodium (Na^+) occurs, ultimately triggering renal cell swelling and tissue damage [2], [3]. Can be expressed as below: Hemolysis – Oxidative stress – Renal cell damage – tubular dysfunction – kidney damage

During hemolysis, reactive oxygen species (ROS) and oxidative stress are generated, which exacerbate renal injury. Hemolysis-induced phosphaturia, hypercalciuria, and other metabolic byproducts directly target and damage the tubular structures. Another critical mechanism contributing to renal damage in thalassemic patients is iron overload. In patients receiving chronic erythrocyte transfusions, systemic iron accumulates and can also enhance intestinal iron absorption. Excess iron heightens oxidative stress, subsequently damaging various tissues, including renal glomeruli and particularly renal tubular structures [3].

Another key mechanism is hypercalciuria. In thalassemia patients, disrupted bone metabolism and altered mineral homeostasis lead to increased urinary calcium (Ca^{2+}) excretion [4], [5], [6].

In chronically transfused patients, hyperoxaluria represents an additional mechanism of renal injury. Increased urinary excretion of oxalate in these patients stimulates the precipitation of calcium oxalate crystals. Furthermore, hyperuricosuria plays a significant role: persistent erythrocyte destruction during hemolysis accelerates purine metabolism, leading to elevated uric acid levels that create a lithogenic environment. Hypocitraturia—a decrease in urinary citrate—further increases the risk of stone formation [5], [7], [8].

Tubular dysfunction is another vital mechanism. Elevated urinary protein and albumin excretion, accompanied by excessive urinary excretion of calcium and phosphate, predominantly signal the onset of proximal renal tubular damage [3], [9].

Additionally, iron chelation therapy can contribute to renal tissue damage. In thalassemic patients, chelation regimens frequently utilize deferasirox-containing agents, which can induce serum creatinine elevation. Deferasirox administration is associated with excessive urinary excretion of phosphate, uric acid, and other solutes, culminating in hypophosphatemia, hyperphosphaturia, and hypercalciuria [10], [11], [12], [13].

2. Materials and Methods

This retrospective observational study used the examination of hospital medical records of 177 patients with thalassemia assessed between August 2025 and August 2026. Data was collected with the permission of the healthcare institution. The study aimed to describe the frequency and distribution of renal and urinary tract abnormalities in patients with thalassemia, with particular emphasis on renal ultrasonographic findings and available clinical characteristics.

Data were retrospectively collected from patients' medical histories, available laboratory analysis results, medical records, and renal and abdominal ultrasonography reports. The extracted information included age, documented

renal abnormalities, and available records on erythrocyte transfusion therapy. Laboratory results were reviewed according to their availability in the medical records; therefore, not all laboratory parameters were necessarily available for every patient.

Renal ultrasonography reports were assessed to identify documented renal and urinary tract abnormalities. The recorded findings included microlithiasis, renal calculi, parenchymal and sinus cysts, increased renal parenchymal echogenicity, and other reported abnormalities. Of the 177 patients enrolled in the study, renal ultrasonographic abnormalities were documented in 137 patients. As more than one abnormality could be present in each patient, the number of recorded findings was not necessarily equivalent to the number of affected patients. The analysis relied on the findings documented in the available ultrasonography reports.

Patients were categorized into five age groups: 0–10 years, 11–18 years, 19–30 years, 31–50 years, and ≥50 years. The distribution of documented renal abnormalities across these age groups was summarized using the available medical records (Table 1) and (Figure 1).

Table 1. Statistics on renal complications.

Age Group	Number of Patients	Number of Pathology-Detected Patients
0-10	30	15
11-18	43	31
19-30	48	41
31-50	38	35
50+	18	15

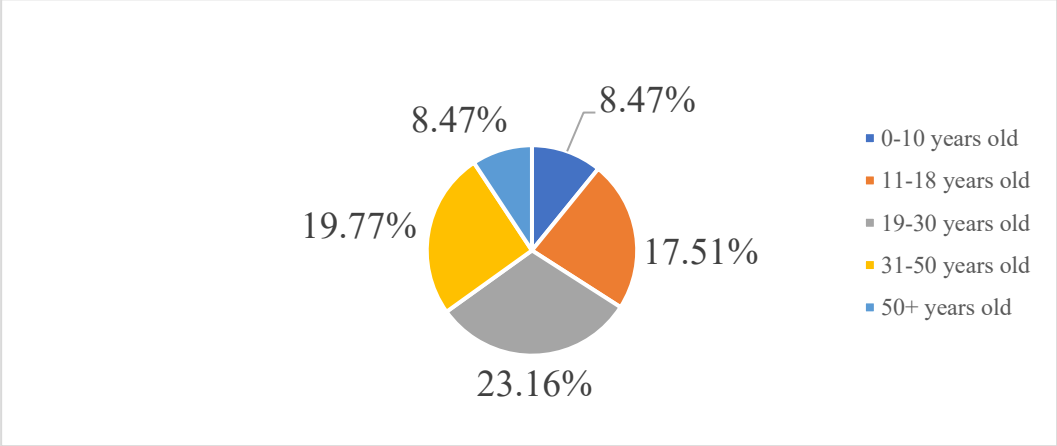


Figure 1. Statistics on renal complications.

The data were evaluated using descriptive statistics. Absolute numbers and percentages were used to summarize patient characteristics, age-group distributions, and renal ultrasonographic findings. No inferential statistical methods were applied, and adjusted estimates of the relationships between potential risk factors and renal abnormalities were not obtained. Estimated glomerular filtration rate (eGFR) was not estimated in this study.

The study was conducted with the permission of the healthcare institution. The use of patient medical records complied with the applicable institutional requirements for privacy and confidentiality and protection of personal information. Details of ethics committee approval, including the committee's name and approval reference, should be reported if applicable and documented.

3. Results

Out of the 177 thalassemic patients evaluated in the study, renal ultrasonographic (USM) abnormalities were detected in 137 individuals. It should be noted that multiple concurrent renal pathologies were identified in several patients. Based on renal ultrasonography findings evaluated between August 2025 and August 2026, microlithiasis (fine urinary crystals) emerged as the most prevalent abnormality among thalassemic patients with identified renal pathologies, affecting 68.93% of the cohort (Figure 2).

In patients receiving chronic blood transfusions, elevated urinary excretion of lithogenic substances creates a favorable biochemical environment for the nucleation and development of renal microcrystals and small calculi. Hypercalciuria and hyperuricosuria play pivotal roles in this process. Furthermore, chronic hemolysis and high erythrocyte turnover accelerate purine catabolism and uric acid excretion, thereby promoting urate crystal formation and facilitating the crystallization of calcium-bearing (Ca2+) salts. Over time, the aggregation and growth of these microcrystals can lead to frank nephrolithiasis, mechanical irritation of the urinary tract mucosa, and occasional hematuria [4], [5], [14].

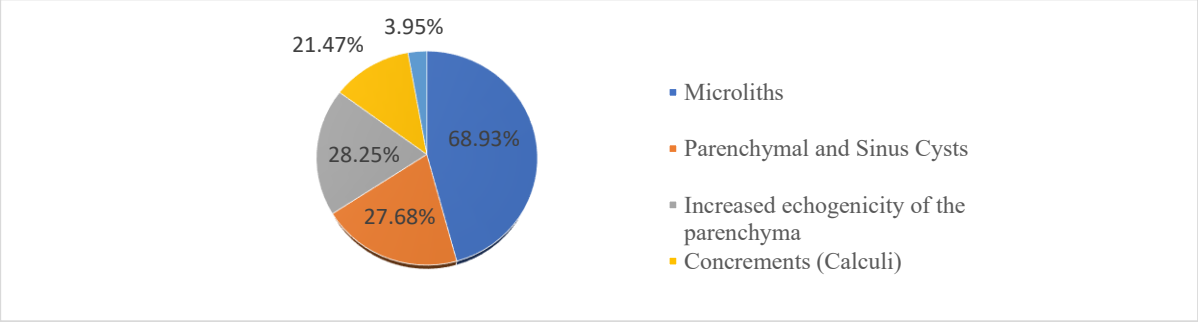


Figure 2. Most common kidney complications

Prolonged transfusion therapy duration in patients receiving long-term blood transfusions is directly correlated with progressive renal dysfunction. Suboptimal adjustment of inter-transfusion intervals induces renal tissue injury through two primary pathophysiological mechanisms [1], [2], [3]:

1) ***Hypoxia and Ischemia:*** Extended intervals between transfusions lead to protracted anemia and tissue hypoxia. The renal cortex and medulla are exquisitely sensitive to oxygen deprivation. Consequently, chronic hypoxia induces glomerular hyperfiltration, tubular atrophy, and interstitial fibrosis [1], [3].

2) ***Iron Overload and Hemosiderosis:*** Excessive iron accumulation in renal tubular epithelial cells drives the generation of free radicals (via Fenton chemistry), causing increased parenchymal echogenicity and cystic parenchymal changes [1], [3], [10].

According to data collected between August 1, 2025, and August 1, 2026, 89 out of 177 thalassemic patients (50.28%) received regular transfusion therapy (Figure 3). Extending transfusion intervals or administering therapy irregularly exacerbates hypoxic injury within the renal parenchyma. Conversely, optimizing transfusion schedules alongside appropriate iron chelation therapy significantly mitigates the risk of developing renal complications. Among the cohort of 177 thalassemic patients evaluated for renal complications, the overall mortality rate was found to be 1.69%.

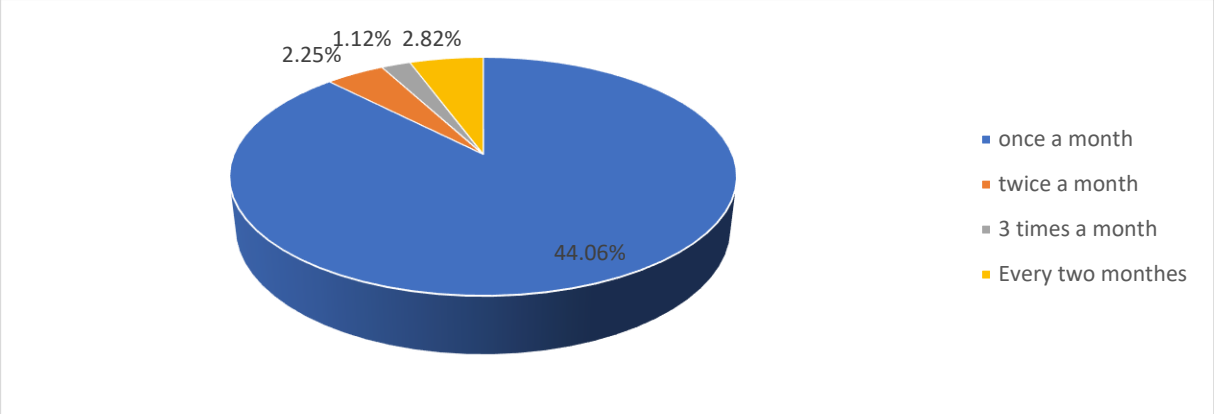


Figure 3. Annual percentage of patients receiving regular transfusion therapy.

Renal complications in patients with sickle cell anemia (SCA) differ significantly from those observed in thalassemic patients due to distinct underlying pathophysiological mechanisms. In SCA, chronic hemolysis coupled with the presence of hemoglobin S (HbS) triggers erythrocyte sickling, particularly within the vasa recta of the renal medulla where localized microvascular occlusion impairs blood flow. The unique microenvironment of the renal medulla—characterized by low oxygen tension, high osmolality, and an acidic pH—provides optimal conditions for HbS polymerization and sickling. Initial renal impairment in SCA typically presents as a loss of urinary concentrating capacity (hyposthenuria) due to ischemic damage within the renal medulla. Concurrently, compensatory glomerular hyperfiltration develops. Persistent hyposthenuria and chronic hyperfiltration accelerate glomerular structural injury, leading to progressive albuminuria and eventually chronic kidney disease (CKD). Moreover, focal ischemic microinfarctions in the medullary pyramids frequently result in renal papillary necrosis and gross or microscopic hematuria. In contrast, renal involvement in \beta-thalassemia is primarily driven by chronic anemia-induced hypoxia, systemic iron overload secondary to regular blood transfusions, tubular dysfunction, and the nephrotoxic effects of certain chelation regimens.

Overall, the cardinal features that distinguish renal involvement in sickle cell anemia from that in beta-thalassemia include medullary vaso-occlusion, early-onset albuminuria, renal papillary necrosis, and hematuria [15], [16]. In patients with chronic anemia, laboratory testing serves as the initial diagnostic step for evaluating renal complications. The primary biomarkers include serum creatinine, urinary albumin, estimated glomerular filtration rate (eGFR), proteinuria, and routine urinalysis. However, serum creatinine alone is insufficient for detecting early hyperfiltration. Comprehensive diagnostic assessment requires adjunctive monitoring of serum calcium (Ca2+), phosphate (P), and uric acid parameters.

In addition to biochemical testing, renal ultrasonography is routinely utilized to identify structural abnormalities such as renal cysts and hydronephrosis. Concurrent evaluation of both laboratory markers and ultrasonographic findings enables early detection of renal parenchyma damage [2], [3], [17], [18]. Continuous erythrocyte transfusion therapy in patients with thalassemia inevitably leads to systemic iron accumulation and severe iron overload. Excess labile iron catalyzes the generation of reactive oxygen species (ROS), triggering reactive oxidative stress that damages renal tissue and other parenchymal organs.

To mitigate iron toxicity, iron chelation therapy is initiated in thalassemic patients. The primary chelation agents utilized in clinical practice include deferasirox, deferiprone, and deferoxamine. By reducing systemic iron burden, chelation therapy partially attenuates iron-induced oxidative tissue damage. However, the use of deferasirox in particular can be associated with adverse renal effects, such as transient or persistent elevations in serum creatinine and proteinuria. Consequently, routine and rigorous monitoring of renal function is mandatory for all patients undergoing iron chelation therapy [1], [3], [10], [11].

4. Conclusion

Analysis of clinical data from 177 patients with \beta-thalassemia major receiving chronic transfusion therapy between 2025 and 2026 demonstrates a high prevalence of renal and urinary tract complications. Ultrasonographic evaluation revealed microlithiasis as the most frequent renal abnormality, accounting for 68.93% of all detected renal pathologies. The prevalence of renal involvement exhibited a clear age-dependent trajectory: 8.47% in the 0–10 age group, increasing to 23.16% in the 19–30 age group, and remaining elevated at 19.77% in the 31–50 age group. These findings highlight that advancing age, disease duration, and cumulative transfusion duration are critical risk factors for the development of renal pathology.

The underlying pathophysiology of renal injury in chronically transfused thalassemic patients is multifactorial, primarily driven by chronic anemia, tissue hypoxia, iron overload, hemolysis-induced oxidative stress, tubular dysfunction, and the nephrotoxic potential of iron chelation therapy. These mechanisms are distinct from those observed in sickle cell anemia (SCA), where renal injury is predominantly characterized by erythrocyte sickling and microvascular vaso-occlusion within the renal medulla, hyposthenuria, hematuria, renal papillary necrosis, and progressive glomerular alterations, including focal segmental glomerulosclerosis (FSGS).

The results of this study emphasize that evaluating renal function in thalassemic patients should not be restricted solely to serum creatinine measurement. A comprehensive diagnostic approach—incorporating estimated glomerular filtration rate (eGFR), urinary albumin-to-protein ratios, urinary calcium, phosphate, and uric acid parameters alongside renal ultrasonography—is essential for clinical management. Early identification of subclinical renal changes and individualization of therapeutic strategies are crucial for preserving long-term renal function. Overall, renal complications in patients with beta-thalassemia represent a distinct entity requiring dedicated clinical surveillance.

Acknowledgement

The authors express their sincere gratitude to the Institute of Molecular Biology and Biotechnologies of the Ministry of Science and Education of the Republic of Azerbaijan, and the National Center of Hematology and Transfusion of the Ministry of Health of the Republic of Azerbaijan for their essential scientific, technical, and organizational support during the preparation and implementation of this research. Special thanks are extended to the Chief Physician of the Thalassemia Division, as well as the leadership and staff of the Departments of Biostatistics and Nephrology, for their invaluable contributions and collaboration.

Author Contributions

The sole author is responsible for conceptualizing, collecting, and analyzing data, and writing and editing the manuscript.

Conflict of Interest

The authors declare no conflicts of interest.

Funding

This research received no external funding.

References

[1] Musallam, K. M., & Taher, A. T. (2012). Mechanisms of renal disease in β-thalassemia. *Journal of the American Society of Nephrology*, *23*(8), 1299-1302. https://doi.org/10.1681/ASN.2011111070

[2] Quinn, C. T., Johnson, V. L., Kim, H. Y., Trachtenberg, F., Vogiatzi, M. G., Kwiatkowski, J. L., ... & Thalassemia Clinical Research Network. (2011). Renal dysfunction in patients with thalassaemia. *British journal of haematology*, *153*(1), 111-117. https://doi.org/10.1111/j.1365-2141.2010.08477.x

[3] Demosthenous, C., Vlachaki, E., Apostolou, C., Eleftheriou, P., Kotsiafti, A., Vetsiou, E., & Sarafidis, P. (2019). Beta-thalassemia: renal complications and mechanisms: a narrative review. *Hematology*, *24*(1), 426-438. https://doi.org/10.1080/16078454.2019.1599096

[4] Aliberti, L., Gagliardi, I., Gamberini, M. R., Ziggiotto, A., Verrienti, M., Carnevale, A., & Ambrosio, M. R. (2022). Beta-thalassaemia major: Prevalence, risk factors and clinical consequences of hypercalciuria. *British journal of haematology*, *198*(5), 903-911. https://doi.org/10.1111/bjh.18345

[5] Wong, P., Milat, F., Fuller, P. J., Kerr, P. G., Doery, J. C., Oh, D. H., & Lau, K. K. (2017). Urolithiasis is prevalent and associated with reduced bone mineral density in β-thalassaemia major. *Internal Medicine Journal*, *47*(9), 1064-1067. https://doi.org/10.1111/imj.13533

[6] Wong, P., Polkinghorne, K., Kerr, P. G., Doery, J. C., Gillespie, M. T., Larmour, I., & Milat, F. (2016). Deferasirox at therapeutic doses is associated with dose-dependent hypercalciuria. *Bone*, *85*, 55-58. https://doi.org/10.1016/j.bone.2016.01.011

[7] Ricchi, P., Ammirabile, M., Costantini, S., Di Matola, T., Spasiano, A., Genna, M. L., & Prossomariti, L. (2012). Splenectomy is a risk factor for developing hyperuricemia and nephrolithiasis in patients with thalassemia intermedia: a retrospective study. *Blood Cells, Molecules, and Diseases*, *49*(3-4), 133-135. https://doi.org/10.1016/j.bcmd.2012.05.012

[8] Capolongo, G., Zacchia, M., Beneduci, A., Costantini, S., Cinque, P., Spasiano, A., & Filosa, A. (2020). Urinary metabolic profile of patients with transfusion-dependent β-thalassemia major undergoing deferasirox therapy. *Kidney and Blood Pressure Research*, *45*(3), 455-466. https://doi.org/10.1159/000507369

[9] 9 Naderi, M., Sadeghi-Bojd, S., Valeshabad, A. K., Jahantigh, A., Alizadeh, S., Dorgalaleh, A., & Bamedi, T. (2013). A prospective study of tubular dysfunction in pediatric patients with Beta thalassemia major receiving deferasirox. *Pediatric hematology and oncology*, *30*(8), 748-754. https://doi.org/10.3109/08880018.2013.823470

[10] Scoglio, M., Cappellini, M. D., D'Angelo, E., Bianchetti, M. G., Lava, S. A., Agostoni, C., & Milani, G. P. (2021). Kidney tubular damage secondary to deferasirox: systematic literature review. *Children*, *8*(12), 1104. https://doi.org/10.3390/children8121104

[11] Badeli, H., Baghersalimi, A., Eslami, S., Saadat, F., Rad, A. H., Basavand, R., & Peluso, I. (2019). Early Kidney Damage Markers after Deferasirox Treatment in Patients with Thalassemia Major: A Case-Control Study. *Oxidative Medicine and Cellular Longevity*, *2019*(1), 5461617. https://doi.org/10.1155/2019/5461617

[12] 12 Tanous, O., Azulay, Y., Halevy, R., Dujovny, T., Swartz, N., Colodner, R., & Levin, C. (2021). Renal function in β-thalassemia major patients treated with two different iron-chelation regimes. *BMC nephrology*, *22*(1), 418. https://doi.org/10.1186/s12882-021-02630-5

[13] 13 Falahati, V., Ghasemi, A., Safari, M. R., Ghaffari, K., Yousefichaijan, P., & Zamanian, M. (2022). Renal function in patients with thalassemia major receiving Exjade® dispersible tablets and a new film-coated tablet formulation of deferasirox (Nanojade®). *Advanced Biomedical Research*, *11*, 84. https://doi.org/10.4103/abr.abr_89_21

[14] Akbarnataj, H., Eghbali, A., Ashayeri, N., Padooiy Nooshabadi, M., & Hosseini Shamsabadi, R. (2025). Evaluating Renal Glomerular Function in Beta-Thalassemia Patients Receiving Deferasirox Using Serum Cystatin-C and Creatinine: A Cross-Sectional Study. *Hemoglobin*, *49*(3), 178-186. https://doi.org/10.1080/03630269.2025.2497856

[15] Nath, K. A., & Hebbel, R. P. (2015). Sickle cell disease: renal manifestations and mechanisms. *Nature Reviews Nephrology*, *11*(3), 161-171. https://doi.org/10.1038/nrneph.2015.8

[16] Zahr, R. S., & Saraf, S. L. (2024). Sickle cell disease and CKD: an update. *American journal of nephrology*, *55*(1), 56-71. https://doi.org/10.1159/000534865

[17] Sumboonnanonda, A., Malasit, P., Tanphaichitr, V. S., Ong–ajyooth, S., Petrarat, S., & Vongjirad, A. (2003). Renal tubular dysfunction in α-thalassemia. *Pediatric Nephrology*, *18*(3), 257-260. https://doi.org/10.1007/s00467-003-1067-7

[18] Ali, D., Mehran, K., & Moghaddam, A. G. (2008). Comparative evaluation of renal findings in Beta-thalassemia major and intermedia. *Saudi Journal of Kidney Diseases and Transplantation*, *19*(2), 206-209.

Journal of Molecular Biosciences and Engineering

Review Article

Forest Plant Diseases and Pests: Assessing Biological Risks in Tourism Areas

Gadir Mammadov [1] ✉ **and Najaf Mammadzada** [2] ✉

[1] Department of Tourism and Hotel Management, Western Caspian University, Baku, AZ1072, Azerbaijan

[2] Department of Cybersecurity and Computer Engineering, Baku Engineering University, Baku, AZ0101, Azerbaijan

Received: 20.10.2026 Accepted: 28.10.2026 Published: 30.10.2026

https://doi.org/10.54414/BMSL7977

Copyright: © 2026 by the authors. Licensee: Journal of Molecular Biosciences and Engineering, Western Caspian University, Baku, Azerbaijan. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution 4.0 International License (CC BY 4.0).

Abstract

Forest health nears from a network of connections: how a tree hacks its own body, how a disease disseminates through its life, how insect numbers serve up and down, and what kind of weather and location mount these things. This review examines how these relationships hint at disease and pestilence problems, and counts recreational forests as a place where people are open, rather than a different biological environment. Colonization by fungi and oomycetes, bacterial infection, viral replication, defoliation, and bark provisioning each bust a different part of how a tree functions, such as how it grows carbon, moves water, or soaks nutrients. Trees exercise barriers they construct ahead of time, drive chemical defenses only when beset, and even exercise the natural enemies of their attackers. However, how effective these strategies are rides on the health of the tree and how severe the attack is. Studies on sudden oak death denote that the pathogen can ride on shoes and that epidemic it from one place to another is biologically possible. Research on firewood shows that live insects can travel out of wood that has been displaced from one location to another. Neither alone can arbitrate what portion of outbreaks should be chalked up to tourism. The presence of perturbed soil and broke roots brings additional openings for vulnerability, but they are also impressed by the environment. Protection relies on regular checks in the field, careful attention to diagnostic results, vigilance on cleanliness in specific areas where problems might arise, and exercising a combined approach to hack pests effectively. More research is essential to link the movement of propagules with real infections, to estimate how disturbances and climate factors intercourse, and to estimate interventions predicated on their biological effects and ecological impacts.

Keywords: forest pathology; forest entomology; tree physiology; host–pathogen interactions; biological control; recreational biosecurity.

1. Introduction

What makes a forest diverse is not just the trees that are there. Roots compound with microbes, the animals that partake of plant parts, the organisms that break down the plant material, and the environments that form as trees grow old and eventually cease. All these elements pitch in in their own way. Native pathogens and insects are part of this scene, and their presence in a stand does not denote that the stand has been broken. Problems startle when the damage goes too far for the host population to handle, making it hard for them to ride and copycats, which usually happens after an invasion by another species or a change in the environment. The presence of different species also influences how easily an infection can disseminate: when animals that are easy to infect are close together, it's simpler for the infection to hand over from one to another. However, if nearby animals cannot serve as hosts, they may lay over the infection to spread further [1], [2].

Disease and half-pint damage often delay the growth of a tree long before it communicates clear signs of

morbidity. Once parts like leaves, roots, bark, or the tree's internal transport system are injured, the tree changes how it allocates and deploys its resources, and this influences its ability to cope and regrow. The fact that drought and insect attack can work through the same physiological pathways implies that there is rarely a single explanation for decline [3].

Forests that people visit for fun need care, because visitors and the work of fettle trails in shape can convert spores to plants that might not have come into contact with them otherwise. Nonetheless, it would be erroneous to assume that recreational access accounts for all infections occurring near a path. This review constructs the biological reasons, how they spread, and the effects on the environment when forests pick up sick or are beset by pests. It also brackets the impact of tourism, like how people move around and displace the area, with the strength of the evidence confirming these issues. It printed research instead of divulging new findings.

2. Biological Causes of Forest Plant Diseases and Pest Infestations

Forest pathogens are not all the same kind of organism. They contrast in the way they partake, the way they copycat, and the extent to which they depend on functional host cells. Among the fungi, there are those that bring leaf diseases, some that reside in the plant's water transport system, others that bring cankers, and some that bring root decay; certain types grip nutrients from functional cells, while others decimate the tissue first before exercising it. Spores can come from the wind or from water that sloshes around, and some fungi that influence roots disseminate through the roots and through contact between them. Bacteria can pierce the body through cuts or natural openings and then culture in the tissues they detect as suitable for propagating. Viruses replicate inside living cells and disseminate through connections between cells and vascular tissue, riding on vectors or alloyed materials to cover longer distances. Finding a virus does not automatically establish that it causes disease, and unless there is specific evidence from the infected system, viruses should not be categorized as the main causes of tree death [4].

Getting into a host and then culturing inside it are two different challenges. Some pathogens deliver enzymes that assist in disintegrating parts of the cell wall, while others bring a way to menu from cells that they observe alive. Whether colonization chances ride on how the host IDs the invader and on how much the pathogen can bust the host's defenses. Once the virus copycats on the infected tissue, it determines the amount of virus present for the next stage of the epidemic. So, how often an infection occurs, how fast a lesion cultures, and how much chance of sporulation are separate things. They aren’t the same way to measure how aggressive a pathogen is [4].

Oomycetes like *Phytophthora* come across like fungi, but they are part of a different group below the stramenopiles. Water frolics play a key role in their biology. The sporangia can release zoospores that swim around, and special structures assist the organism in surviving tough conditions. *Phytophthora ramorum* shows that being susceptible and having a significant impact in an outbreak should not be the same thing. In a study from California, infected bay laurel leaves were able to deliver inoculum, but the cankers institution on coast live oak served not. A tree might be seriously sick, but not the main reason the disease is epidemic, and neighbors that come across less affected could be assisting the outbreak to endure [5], [6].

Insects can bring various types of injuries predicated on the way they feed. Leaf-eating caterpillars partake of the green parts of plants that lack food, and bark beetles deposit their eggs under the bark, which detrimentally affects the plant's food transport system, often carrying harmful fungi with them. The European spruce bark beetle, *Ips typographus*, communicates that the impact of an attack rides heavily on numbers: when they assemble and bring together, they can pacify defenses that would have been strong enough to lay over just a few of them. The outcome of an outbreak is influenced by factors like the available breeding material, the health of the host, the development stage of the insect, the presence of conspecific microbes, and natural enemies, rather than just the number of individuals present [7].

Colonization occurs only when there are chances of a suitable host, enough exposure, and the right conditions are present. Tree age marks the structure of the bark, how roots form, the amount of damage that has accumulated over time, and the type of tissue available, but this doesn't mean older trees are always more vulnerable than young seedlings. Temperature influences how plants form and copycats, and moisture plays a key role in assisting spores startle to culture, impactful zoospores, and fettle the host plant's water levels in check. Drought does not

influence all areas the same way: how plants react can contrast depending on what is edible and whether the damage comes from main pests or secondary ones. Also, even as water stress adds to it, it doesn't jack up every pest or disease worse [4], [8].

Populations are observed under control by predators, parasitoids, entomopathogenic fungi, and microorganisms that either face off with the pests or directly attempt them. How important these agents are depends on how available the host is, whether their life cycle episodes at the same time, the type of habitat, and the conditions of the microclimate. Natural regulation can be less effective exactly when a pest is rapidly epidemic or the environment is irregular, and depending solely on natural enemies doesn't always keep pest levels below harmful thresholds [7], [9].

3. Effects of Diseases and Pests on Forest Plant Physiology and Ecosystem Function

The order in which the constraints show depends on where the injury is. Lesions on leaves and the loss of foliage degrade the area of the leaf that can perform photosynthesis, and when plants experience water stress, their stomata close, which limits how much carbon they can take in. Root necrosis dents the root's ability to take in water and nutrients, and when certain microbes settle in the plant's vascular system, the blockages they bring can slow the flow of water through the plant. Feeding on bark catches the flow of nutrients between the leaves and the roots. In living tissues, respiration might increase initially as the body brings resources to support and mend itself, but if too much tissue is misplaced, it can eventually slow down the overall metabolic activity of the entire tree. These responses occur in episodes one after another, and their importance makes overcoming the disease [3], [4].

Trees exercise the barriers they naturally hold, along with the responses they bring. The bark structure, resin systems, and phenolic compounds in conifers impede penetration or slow colonization. After an attack, chemicals build up in the area, traumatic resin ducts form, and the tissue can snare the invader in compartments. Defense also touches on how cells fetch and pack signals, not just being a passive structure. Even so, chemical toxicity, how resins run, and how plants construct their defensive tissues contrast between species and depend on their physical state, so knowledge gained from conifer bark can't be directly applicable to broadleaved plants [4], [10].

Damage can disseminate from one organ to another. A lower amount of water makes it harder for plants to perform photosynthesis, and when the movement of carbon is not functioning properly, it can influence the health of the roots, which in turn degrades their ability to take up water and nutrients. Repeated loss of forsakes can drain a tree exercise up its stored energy to foliage, which forsakes less energy for culturing and stuff new seeds. In the other direction, minor damage can be recovered if there are enough resources and enough time to recover. When both drought and bark beetles attack a tree, it might be too much for the tree to recover, but to elucidate the tree conked because of these factors, hypodermic needle proof of how and how strongly each stressor came in order [3].

The effects on reproduction can be evident prior to death. Less assimilation or weaker transport can condition how much a tree can dispose into flowers, cones and seeds, and damage to these reproductive parts directly degrades the output. Seedlings hold different challenges because their small root systems and limited stored energy make it hard for them to recover after mislaying roots or being denuded of leaves multiple times. Focusing only on the mature canopy may command changes in the population dynamics that arbitrate if a forest endures to live [3], [4].

At the population level, mislaying seedlings or reproductive trees repeatedly neuter how new plants are established and arbitrate where future host plants will grow. Death that affects certain species can modify the makeup of the tree layer, the amount of shade, and the resources available to other living things. Biological disturbance also leaves behind deadwood and brings spatial heterogeneity. To preserve the functioning of an ecosystem, it is not necessary to close out native pathogens, insects, or dead trees; rather, it is important to discern their ecological roles from impacts that are excessively severe or caused by an invader. The disease triangle, which contains a susceptible host, a strong pathogen, and a favorable environment, helps explain why just keeping a pathogen doesn't always mean the disease will be severe. Host density and identity also play a role: combining different plants can reduce the harm by specialized insects, but this effect depends on how varied the plant diet is and which species are common. Diversity functions as a resistance strategy that rides on the situation rather than being a

consistent obstacle. In a similar way, alternative hosts can sometimes assist in observing a pathogen alive, as exhibited by the difference between bay laurel and badly infected oaks [1], [4], [5].

4. Biological Risks in Forests Used for Tourism

A full transmission path claims picking up the pathogen, impacting it, uncorking it, riding, and encountering a vulnerable host in conditions that permit infection or the pathogen to gain a hold. Davidson et al. [5] recaptured *P. ramorum* disseminates through soil contaminated by hikers' shoes during the rainy season, and it was shown how the infection displaces from alloyed soil to green leaf litter and then to the leaves of young seedlings. These observations denote that their chances a possible way this could episode; they don't describe to us exactly how many existing infections were converted in by visitors. Cushman and Meentemeyer [11] found that bustling patterns compounded to trails that match how humans disseminate, but an observation-predicated study like that can't rule out all possible environmental influences or variations in how easy it is to hand over certain areas.

Firewood is a different vehicle. Bark and wood furnish shelter for young insects as they are conveyed, permitting their development to endure once they hand over a new location. Jacobi et al. In 2012, they expressed live insects from firewood picked up at a store, which is more convincing proof of the wood conveying insects than just judgment holes in it. Recreational firewood can carry farther than usual local spread, but for an invasion to be successful, it also inevitably needs the right hosts, the ability to ride, and the chance to copycats. Research findings contrast riding on the region and the type of species considered, which implies that any risk assessment can only apply to a limited extent [12].

Soil attached to tires, bicycles, tools, or other equipment can be a routine means for the target organism to disseminate, wherever it is present in that material. Even so, evidence for one pathogen and one carrier should not be used to conclude all forest pests. Tourism should not be treated the same as the broader movement of nursery stock. The trade in live plants is separately registered as a significant way that pests or diseases can pierce new areas, and it can also introduce infected plants to public parks and recreational areas [6], [13].

Recreation can modify host conditions as well. Trampling and trails can jackleg the soil, disturb roots, harm young plants, and modify how water runs through the area. Trees that are vulnerable may be more susceptible to infection due to poor aeration and injured roots, and a lack of regeneration can hinder recovery, even in the absence of a pathogen. These links are scientifically reasonable, but keeping evidence of recreational use doesn't exhibit that disease cases are aggrandizing. Wet conditions might hint at the movement of dirty soil and amplify the activity of oomycetes, while drought can make certain stress–pathogen relationships more intense. The overall impact is inevitably to be assessed for each pathogen individually instead of being generalized, because not all types of disturbance lead to higher infection rates [8], [14][8].

5. Biological Monitoring, Prevention, and Forest Protection

Monitoring should discern between symptoms, which are the host's reaction, and the signs that indicate the organism is harmful. Crown thinning, chlorosis, wilting, resin bleeding, and slower growth all offer us some clues, but they often don't point to a specific problem. Fruiting bodies, distinct galleries, frass, and recognizable insects furnish more direct evidence, even though different agents often show together. Repeated surveys should document the host species, its size or age group, which organs are affected, how symptoms form over time, and any recent environmental stress factors. This assists in interpreting the results in a biological context rather than just predicated on what is described [4].

Diagnostic sampling must be concluded by checking where the possible cause of the problem is located. Different parts like the edges of the broken area, the infected fine roots, the sick leaves, the insects, and the environment all require different ways to collect samples. Microscopy and culture methods exhibit the organism's shape and whether it is still alive, but only if the organism can be grown in a lab. Targeted molecular tests can determine organisms with great accuracy, whereas metabarcoding aims at a wider range of species. In surveillance for airborne fungi [15], it was shown that it was able to detect a broader range of taxa, but specific quantitative PCR was developed to be more sensitive. Neither approach dispenses with the need for proper sampling, authenticated reference sequences, and checks for contamination; DNA by itself cannot establish that an organism is alive,

arbitrate how much it contributes to bringing infection, or show that it was the actual cause. Negative results can be due to hypodermic needle contamination too. An organism can be missed by means of patchy colonization, low inoculum abundance, sampling at the wrong time of year, or substances that hinder amplification. Creating copies of the sample, exercising proper controls, and additional testing methods can assist growers with this uncertainty. If a test observes an imminent back negative, it doesn't necessarily imply the infection isn't present [4], [15].

The use of a surveillance design links high-exposure sites with similar reference areas and habitual observations throughout the seasons. Linking lab results with the symptoms a host communicates later helps describe the difference between an organism that was just present without infection and one that has gripped hold in the body. Maps must expose the locations of susceptible hosts and the locations where pathogens have been bustling, and if this is not concluded, a change in detection frequency may be due to sampling effort, host distribution, or seasonal sporulation, rather than a change in risk [11].

Integrated pest management integrates the process of diagnosing the pest, estimating the level of the population or disease, and taking action that halves the severity of the problem. Keeping predators, parasitoids, and harmful microbes in check can assist in attacking the hosts and their environments more effectively. Microbial biological control can be achieved by facing off with pathogens, delivering substances that inhibit them, acting as parasites, or assisting the host plant to form resistance. However, even if these microbes suppress pathogens in lab tests, it doesn’t imply they will effectively control diseases in a whole forest. Any candidate agent inevitably to be evaluated for how specific it is to its host, how long it remains effective, how well it works, and how it influences other organisms that aren't the target. Similarly, aggrandizing the variety of native tree species should capitalize on selecting trees that work well together and align with local environmental objectives, instead of claiming that any mix of trees will automatically preclude all pests [1], [9].

Prevention should disrupt the cycle that disseminates the risk. Removing stuck soil before impacting anything, eviscerating exercising methods that bring against the pathogen, heading off the spread of infected plant parts, and picking up firewood that's from nearby or has been properly refined each cover a separate way to hack the issue. Cleaning should not touch contaminated soil or wash water near plants that are sensitive to contamination. When the case is strong enough, accommodating access during specific times of the year can lower exposure during periods that are most likely to disseminate the disease. The biology of the target organism and the habitat it furnishes should be estimated when, or before, breeding material. Success should be considered by factors like lower use of transport that isn't very effective, less spread of infections, or smaller harmful populations, rather than by the presence of hygiene facilities [5], [7], [12].

6. Conclusion

Biological risk in forests comes from how things intercourse with each other, not just from individual factors. How a pathogen picks up food and reproduces determines if it can grip onto tissue and how much of it can culture; how many insects there are and how many of them live influences the damage they bring; the protections and strength of the host determine if the damage stays in one place or leads to bigger problems. Environmental conditions influence each stage, and the impact on the ecosystem rides on which hosts are misplaced and if new ones can displace them in time. Keeping forests healthy means we use a hypodermic needle to capitalize on how plants interact and how they compound with each other in nature, not just on what we can feel [3], [4].

Tourism can hint at specific problems like more transport and disruption, but it doesn't cause forest diseases in all cases. The presence of active germs on shoes and resident bugs in firewood implies that efforts should capitalize on specific ways these things disseminate. However, the link institution along trails and the possible ways stress influences roots import we should be more careful and not leap to conclusions. The main goals of the research are to compound carrier contamination with infection or the spread of reproduction, to determine whether recreational activities are changes separate from what changes in hosts or because of climate changes, and to bill if biological control methods and hygiene practices bring in real-world settings. Longitudinal studies that cover both the health of the host, the identification of organisms, and the results in the ecosystem can assist in forecasting changes better, while ensuring they do not displace the natural processes of the forest [5], [8], [9]

Author Contributions

The sole author is responsible for conceptualizing, collecting, and analyzing data, and writing and editing the manuscript.

Conflict of Interest

The authors declare no conflicts of interest.

Funding

This research received no external funding.

References

[1] Jactel, H., Moreira, X., & Castagneyrol, B. (2021). Tree diversity and forest resistance to insect pests: patterns, mechanisms, and prospects. *Annual Review of Entomology*, *66*(1), 277-296. https://doi.org/10.1146/annurev-ento-041720-075234

[2] Keesing, F., & Ostfeld, R. S. (2024). The more, the healthier: Tree diversity reduces forest pests and pathogens. *PLoS Biology*, *22*(2), e3002525. https://doi.org/10.1371/journal.pbio.3002525

[3] Anderegg, W. R., Hicke, J. A., Fisher, R. A., Allen, C. D., Aukema, J., Bentz, B., & Zeppel, M. (2015). Tree mortality from drought, insects, and their interactions in a changing climate. *New Phytologist*, *208*(3), 674-683. https://doi.org/10.1111/nph.13477

[4] Agrios, G. N. (2005). *Plant pathology*. Elsevier.

[5] Davidson, J. M., Wickland, A. C., Patterson, H. A., Falk, K. R., & Rizzo, D. M. (2005). Transmission of Phytophthora ramorum in mixed-evergreen forest in California. *Phytopathology*, *95*(5), 587-596. https://doi.org/10.1094/PHYTO-95-0587

[6] Grünwald, N. J., Goss, E. M., & Press, C. M. (2008). Phytophthora ramorum: a pathogen with a remarkably wide host range causing sudden oak death on oaks and ramorum blight on woody ornamentals. *Molecular Plant Pathology*, *9*(6), 729-740. https://doi.org/10.1111/j.1364-3703.2008.00500.x

[7] Biedermann, P. H., Müller, J., Grégoire, J. C., Gruppe, A., Hagge, J., Hammerbacher, A., & Bässler, C. (2019). Bark beetle population dynamics in the Anthropocene: challenges and solutions. *Trends in ecology & evolution*, *34*(10), 914-924. https://doi.org/10.1016/j.tree.2019.06.002

[8] Jactel, H., Petit, J., Desprez-Loustau, M. L., Delzon, S., Piou, D., Battisti, A., & Koricheva, J. (2012). Drought effects on damage by forest insects and pathogens: a meta-analysis. *Global Change Biology*, *18*(1), 267-276. https://doi.org/10.1111/j.1365-2486.2011.02512.x

[9] Balla, A., Silini, A., Cherif-Silini, H., Chenari Bouket, A., Moser, W. K., Nowakowska, J. A., & Belbahri, L. (2021). The threat of pests and pathogens and the potential for biological control in forest ecosystems. *Forests*, *12*(11), 1579. https://doi.org/10.3390/f12111579

[10] Franceschi, V. R., Krokene, P., Christiansen, E., & Krekling, T. (2005). Anatomical and chemical defenses of conifer bark against bark beetles and other pests. *New Phytologist*, *167*(2), 353-376. https://doi.org/10.1111/j.1469-8137.2005.01436.x

[11] Cushman, J. H., & Meentemeyer, R. K. (2008). Multi-scale patterns of human activity and the incidence of an exotic forest pathogen. *Journal of Ecology*, *96*(4), 766-776. https://doi.org/10.1111/j.1365-2745.2008.01376.x

[12] Solano, A., Rodriguez, S. L., Greenwood, L., Dodds, K. J., & Coyle, D. R. (2021). Firewood transport as a vector of forest pest dispersal in North America: A scoping review. Journal of Economic Entomology, 114(1), 14-23. https://doi.org/10.1093/jee/toaa278

[13] Liebhold, A. M., Brockerhoff, E. G., Garrett, L. J., Parke, J. L., & Britton, K. O. (2012). Live plant imports: the major pathway for forest insect and pathogen invasions of the US. Frontiers in Ecology and the Environment, 10(3), 135-143. https://doi.org/10.1890/110198

[14] Pickering, C. M., Hill, W., Newsome, D., & Leung, Y. F. (2010). Comparing hiking, mountain biking and horse-riding impacts on vegetation and soils in Australia and the United States of America. Journal of Environmental Management, 91(3), 551-562. https://doi.org/10.1016/j.jenvman.2009.09.025

[15] Chandelier, A., Hulin, J., San Martin, G., Debode, F., & Massart, S. (2021). Comparison of qPCR and metabarcoding methods as tools for the detection of airborne inoculum of forest fungal pathogens. Phytopathology, 111(3), 570-581. https://doi.org/10.1094/PHYTO-02-20-0034

[16] Jacobi, W. R., Hardin, J. G., Goodrich, B. A., & Cleaver, C. M. (2012). Retail firewood can transport live tree pests. *Journal of Economic Entomology*, *105*(5), 1645-1658. https://doi.org/10.1603/EC12069

Western Caspian University

Journal of Molecular Biosciences and Engineering

Vol 1, No 3

October 2026

Editorial Office Address: 17 A, Ahmad Rajabli Street,

III Parallel, Baku, Azerbaijan

Phone: +994 12 565 39 77

Email: jmbe@wcu.edu.az

https://jmbe.wcu.edu.az/

Submitted for collection: 29.10.2026

Signed for printing: 29.10.2026

Paper format: 60x84 1/16 17.75