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Association of TP53 Codon 72 Polymorphism with HPV-related Cervical Lesions in Iraqi Women

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Abstract

It is one of the major impacts of human papillomavirus (HPV) infection on the pathogenesis of the cervix. The present paper provides a comparison between the connection between TP53 gene codon 72 (Arg72Pro) and the likelihood of developing genital warts and HPV-related cervical cancer in Iraqi women. PCR-RFLP was used to genotype 116 women (60 controls, 30 genital wart patients and 26 cervical cancer patients). TP53 C/C (Pro/Pro) was far more common in genital warts than controls (43.3 vs. 18.3, p=0.024), indicating that it was more prone to alliteration, whereas G/G (Arg/Arg) was preventative. Associations with the INK4a 540C T polymorphism were not discernible. These data indicate that TP53 polymorphism is associated with benign HPV-related lesions, whereas INK4a does not emerge as a strong predictor.

Keywords: HPV, TP53, polymorphism, cervical cancer, genital warts, Iraqi women, RFLP

الملخص

هدفت هذه الدراسة إلى تقييم العلاقة بين تعدد الأشكال الجيني للكودون 72 في جين TP53 (Arg72Pro) وخطر الإصابة بالآفات المرتبطة بفيروس الورم الحليمي البشري (HPV) لدى النساء العراقيات، إضافة إلى دراسة تعدد الأشكال في جين.INK4a

أظهرت النتائج وجود ارتباط معنوي بين النمط الجيني C/C (Pro/Pro) في جين TP53 وزيادة خطر الإصابة بالثآليل التناسلية المرتبطة بفيروسHPV ، حيث كان هذا النمط أكثر شيوعًا لدى مريضات الثآليل مقارنة بمجموعة السيطرة. في المقابل، ظهر النمط G/G (Arg/Arg) بتأثير وقائي ملحوظ ضد الإصابة بالثآليل.

أما بالنسبة لسرطان عنق الرحم، فلم تُظهر النتائج وجود علاقة معنوية بين تعدد الأشكال في جين TP53 وخطر الإصابة بالمرض، كما لم يُسجل أي ارتباط إحصائي مهم لتعدد الأشكال في جين INK4a مع كل من الثآليل التناسلية أو سرطان عنق الرحم.

تشير هذه النتائج إلى أن التباين الجيني في TP53 قد يلعب دورًا في القابلية للإصابة بالآفات الحميدة المرتبطة بفيروس HPV، بينما يبدو أن تطور سرطان عنق الرحم يعتمد على عوامل وراثية وبيئية أخرى أكثر تعقيدًا. وتؤكد الدراسة أهمية التوسع في برامج التحري المبكر والتطعيم ضد فيروس HPV في العراق للحد من عبء المرض.

الكلمات المفتاحية: فيروس الورم الحليمي البشري، TP53، التباين الجيني، سرطان عنق الرحم، الثآليل التناسلية، النساء العراقيات، RFLP

Introduction

Still on the list of the most important health issues facing the world is cervical cancer, which is disproportionately high in women and causes high morbidity and mortality rates in low or middle-income countries. Human papillomavirus (HPV) infection, which is among the most prevalent sexually transmitted infection around the world, has been determined to be the primary etiological agent in the development of cervical cancer progression, with continued infection by highly susceptible HPV strains—especially types 16 and 18—accounting for around 70% of all incidents of cervical cancer [1]. Even though there is a clear link between HPV and cancer of the cervical cavity, more research is needed to fully understand the complex pathways involving immune system dysregulation and genetic alterations that propel the disease's progression.

As of 2009, the P53 gene, which had been found to have polymorphisms that altered the suppression of tumors, was crucial and could be used to estimate the risk of acquiring cancer. These alterations in the DNA sequence of the cell may be used as indicators for early screening and identifying areas that have a high

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risk of developing into advanced cancer [2]

Often described as the "guardian of the genome," TP53 is the primary regulator of stress-related responses, such as DNA repair, cell cycle arrest, and apoptosis. However, the HPV E6 protein impairs TP53's tumour-suppressive effects by targeting it for disintegration via the ubiquitin-proteasome road to learning. E6 binds to TP53 and attracts the E6-associated protein (E6AP), a type of ubiquitin which causes TP53 the polyubiquitination and degradation via proteasomal processes. This prevents TP53 from activating genes that control cell cycle arrest and apoptosis, and allows HPV-infected cells to survive despite genomic damage. The mutations that have been well studied in cervical carcinoma are TP53 mutations, including the variation of Arg72Pro. As previous studies by Laprano, [3] suggest, the Pro allele suppresses apoptotic cell activity compared to the Arg allele, which may compromise TP53-mediated apoptosis and predispose to the development of cervical cancer caused by the HPV. Other novel molecules, such as RITA (The Reactivation of p53 and Stimulation of malignant cell Apoptosis), are undergoing preclinical disease models as a possible treatment option. A study by Pillai et al. [4] found that RITA successfully restored TP53 activity in the presence of HPV in cancerous carcinoma of the cervical gland cells, causing the death of cancer cells and preventing tumour growth.

By performing a thorough genetic analysis of cervical cancer patients along with HPV infection, including looking for specific mutations in P53 genetic material and its relationships in the framework of the illness's progression, this study aims to shed light on new insights into the genetic components of the development of cervical cancer.

Materials and Methods

Patients and controls sampling

150 participants took part in the study between November 20 and May 20. The case class was (Cervical cancer=50 and Genital warts =50), whereas the comparison group included 50) individuals who are obviously in good health, have no history of sickness, and are clinically defined as healthy controls. This study enrolled adult women aged 18 years and above who were able to provide informed consent. For the first group, participants had to have a confirmed diagnosis of cervical cancer based on histopathological examination. For the second group, participants needed to have genital warts confirmed by clinical examination and cytopathological analysis, but without cervical cancer. For the third group, participants were healthy women with no history of cervical abnormalities, genital warts, or cancer. All participants had to be willing to provide blood and tissue samples for the study.

Preparation of Primers

The manufacturer's instructions were followed for preparing the primer. Lyophilized transcripts have been submerged in TE (Tris-EDTA) buffer in order to attain an initial solution with a concentration of 100 pmole/mL. Following centrifugation, this stock solution was diluted with TE buffer using the volume-concentration formula C1V1=C2V2, and The pH was adjusted to 8 and the solution was stored in the refrigerator. For electrophoresis, a 1X working solution was prepared by diluting 10 ml of 10X TBE stock with 90 ml of distilled water. Consequently, each primer's final concentration in the tested solution was adjusted to 20 pmole/mL. Different primer sets were used to detect various HPV types and to amplify genes of interest. For HPV detection, primers targeting HPV types 16, 18, 33, 35, 45, and 51

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were used, each producing amplicons of specific sizes. For host gene analysis, primers for p53 (producing a 421 bp product) and p16INK4a (producing a 143 bp product) were used. The PCR products were analyzed by agarose gel electrophoresis to confirm successful amplification and correct product size.

Table 1 Primes sets used in this study with their references.

Primer Name	Sequences 5'-'3	TM	PCR product	Source of primer
p53 RFLP	F TTCTGGTAAGGACAAGGGT	63°C	421bp	wong etal.,2000
	R AGGCATTGAAGTCTCATGGA			
p16(INK4a) RFLP	F GCCTGTTTTCTTTCTGCCCTCTG	60.4°C	143bp	Vargas-Torres etal., 2014
	R CGAAAGCGGGGTGGGTTGT			

DNA Extraction and Purification

A molecular test kit intended for viral DNA/RNA extraction was used to extract DNA. The kit purified viral DNA/RNA from a variety of biological samples using magnetic beads based on silica. The working buffers were ready before the extraction process began. Binding Buffer 46 (BB46) received 8 ml of isopropanol, whereas Clean Buffer 46 (CB46) and Wash Buffer 46 (WB46) received 15 ml and 48 ml of 100% ethanol, respectively.

DNA Quality Assessment

Agarose gel electrophoresis was used to evaluate the isolated DNA's purity. Trisborate-EDTA (TBE) buffer was first made. 3.8 grams of Tris-HCl, 2.7 grams of boric acid, and 2 milliliters of 0.5 M EDTA solution in 50 milliliters of distilled water made up the 10X stock solution. The solution was kept in the refrigerator once the pH was brought to 8. Ten milliliters of 10X TBE stock were diluted with ninety milliliters of distilled water to create a 1X working solution for electrophoresis.

One gram of agarose was added to 100 milliliters of 1X TBE buffer in a flask to create a 1% agarose gel. A microwave oven was used to heat the mixture until it began to boil. After swirling the flask, it was put back in the microwave to continue boiling. Two microliters of ethidium bromide dye were added once the mixture had cooled to between fifty and sixty degrees Celsius. After pouring the gel solution into a casting tray with a comb, it was allowed to set for fifteen minutes at room temperature.

Polymerase Chain Reaction (PCR)

To find HPV DNA and amplify particular gene snippets for examination, PCR amplification was used. TransGen Biotech's EasyTaq® PCR SuperMix was used to create the PCR master mix. Taq DNA was present in this ready-to-use combination. dNTPs,

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polymerase, and a 2X concentration of tailored reaction buffer. A PCR tube was filled with 1 µl of 10 µM forward primer, 1 µl of 10 µM reverse primer, 25 µl of EasyTaq 2X Master Mix, varying amounts of template DNA, and nuclease-free water to make a total volume of 50 µl for each reaction. After validation, the primers used in this investigation (Table 3.4) were taken from previously published findings.

Results

The current study group comprised of 150 women; their socioeconomic characteristics and clinical features are provided in Table 1. There were significant variations between the groups in the age distribution ($p<0.001$). In particular, the genital warts group had the youngest age profile (27.9 ± 5.7 years), while the women with cervical cancer group had the highest mean age (45.3 ± 11.0 years), compared to the healthy group (33.4 ± 10.3 years). None of the control subjects tested positive (0.0%), according to the HPV quick test results. whereas 68.0% of the warts in the genitals population and 60.0% of the cervical cancer group tested negative for HPV. Family history of cancer exhibited a varied pattern among the groups ($p<0.001$). The prevalence climbed progressively from 18.0% in the healthy patients' group to 32.0% in the genital warts category and reached 64.0% in the cervical cancer group. This stepwise increase shows a potential connection between family malignancy history as well as HPV-related diseases. Additionally, among the cervical carcinoma individuals, the majority (72.0%) presented with intermediate level disease (stages 2A, 2B, 3A, and 3B), although 16.0% had early-stage disease (stages 1A and 1B), and 12.0% had advanced levels of illness (stages 4A and 4B). According to this staging distribution, the majority of patients received their diagnosis after their illness had proceeded past its early stages but had not yet reached its advanced phases.

Table 1. Baseline demographic and clinical characteristics by study group (N= 150).

Characteristic	Control (n=50)	Genital warts (n=50)	Cervical cancer (n=50)	*P*-value
Age (years) ***Mean ± SD*** ***Median [IQR]*** ***Range***	33.4 ± 10.3 33.5 [26.0-38.0] 21-61	27.9 ± 5.7 26.0 [24.0-31.0] 19-45	45.3 ± 11.0 43.5 [37.0-53.8] 25-70	
HPV rapid test positive, n (%)	0 (0.0%)	34 (68.0%)	30 (60.0%)	-
Family history of cancer, n (%)	9 (18.0%)	16 (32.0%)	32 (64.0%)	<0.001[b]
Cancer stage, n (%) ***Early (1A, 1B)*** ***Intermediate (2A, 2B, 3A, 3B)*** ***Advanced (4A, 4B)***	N/A	N/A	8 (16.0%) 36 (72.0%) 6 (12.0%)	-

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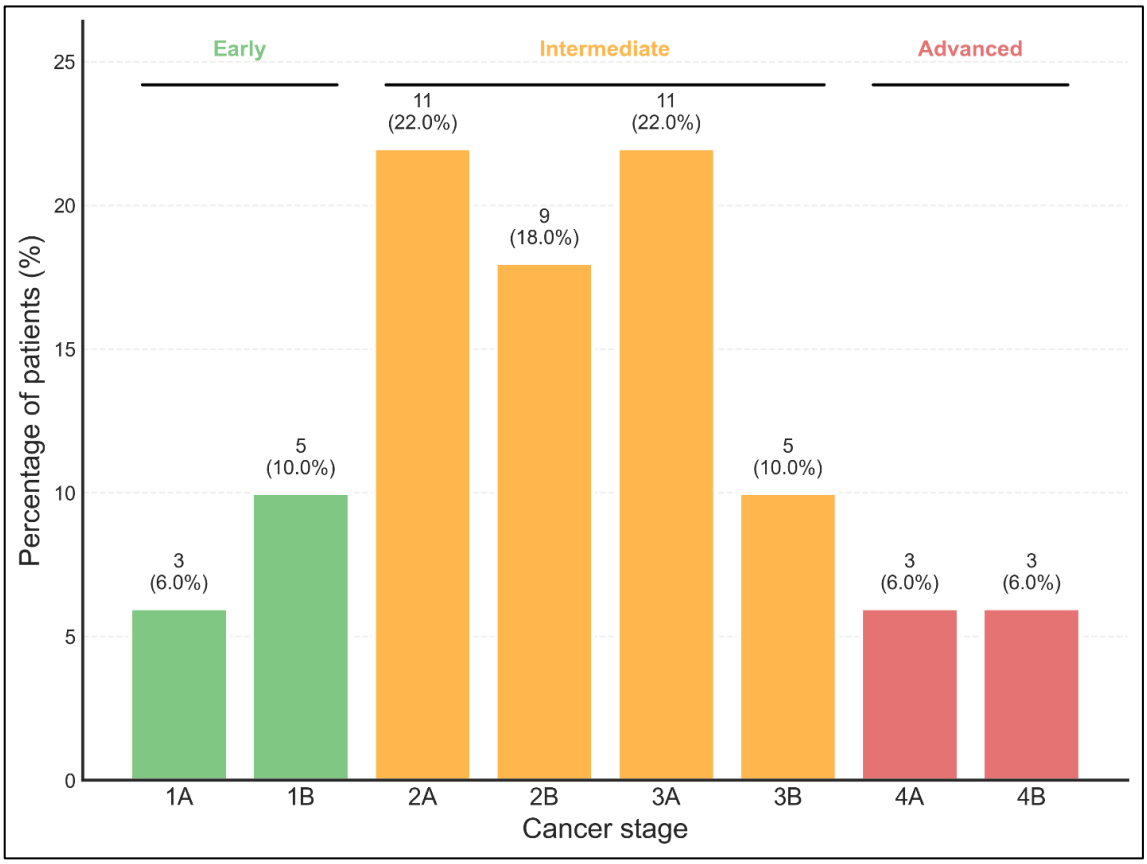

Figure 2. The percentage of individuals with cervical cancer who fall into the early (stages 1A, 1B; green), intermediary (stages 2A, 2B, 3A, 3B; orange), and progressed (stages 4A, 4B; red) categories of the disease is displayed in this bar chart. Both the percentage and the total number of patients are shown by the numbers above the bars. The majority of individuals are presented with stages of intermediate disease. Total sample size was 50 cervical cancer patients with accessible stage information.

The initial molecular research demonstrated effective amplification of the target gene sequences by PCR (Figure 1A and 1B). Agarose gel electrophoresis of PCR products produced from the P53 gene using primers suited to a 421 bp area revealed unambiguous and consistent amplification across all analyzed samples. The distinct DNA samples shown in lanes 1–42 in Figure 1A and lanes 1–30 in Figure 1B had separate bands at the anticipated product size of 421 base pairs. The consistency and brightness of the bands indicate good quality of DNA templates and optimal working conditions of the PCR experiment whereas the negative control lanes (N) did not amplify, which proves the specificity of the primers and lack of contamination. The DNA size markers (L) between the 400 to 1500 bp provided were precise size markers that would detect bands. Such effects were indicative that the P53 gene constituent was efficiently increased in warts induced by HPV, as well as malignant tissues.

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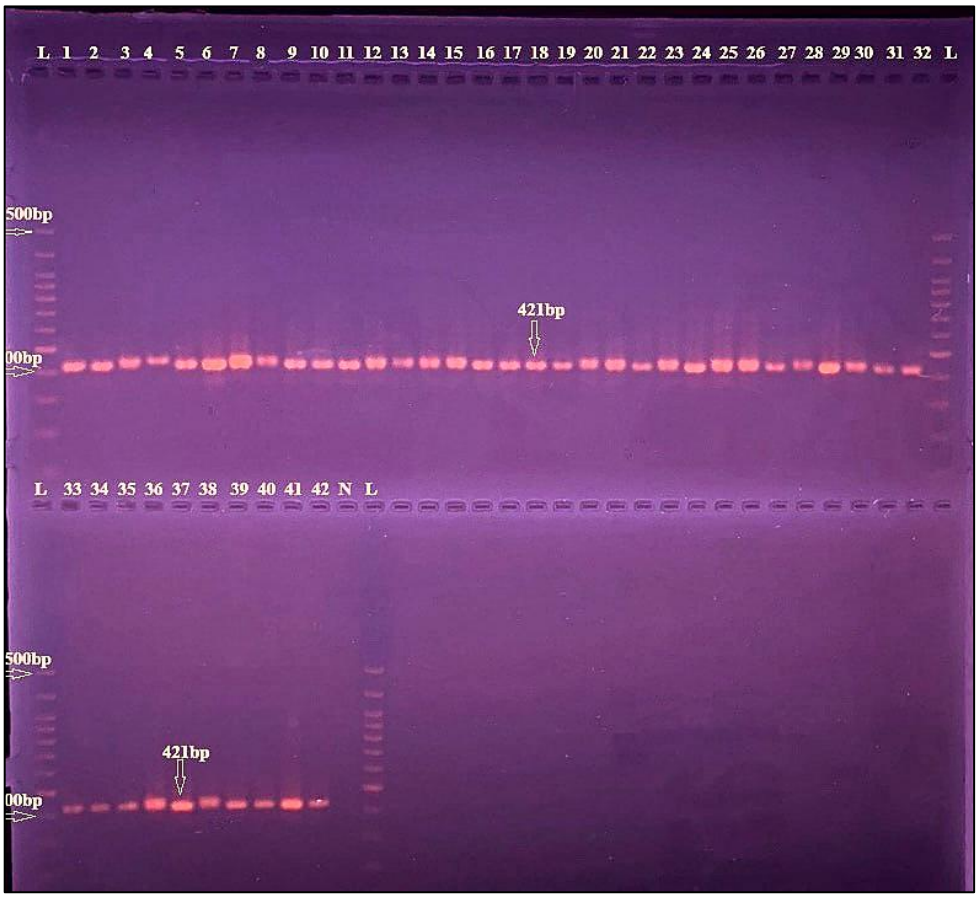

Figure 13A. PCR products of P53 gene with primers that identify a sequence of 421 bp were inserted into an agarose gel and run via electrophoresis. Lanes 1-42 are individual samples of DNA and unique amplified product of the desired size is located. Lane L represents the marker of size that is 400 to 1500 bp in size and lane N is the negative control (non-amplification). Amplification reaction of P53 is efficient and specific depending on its results.

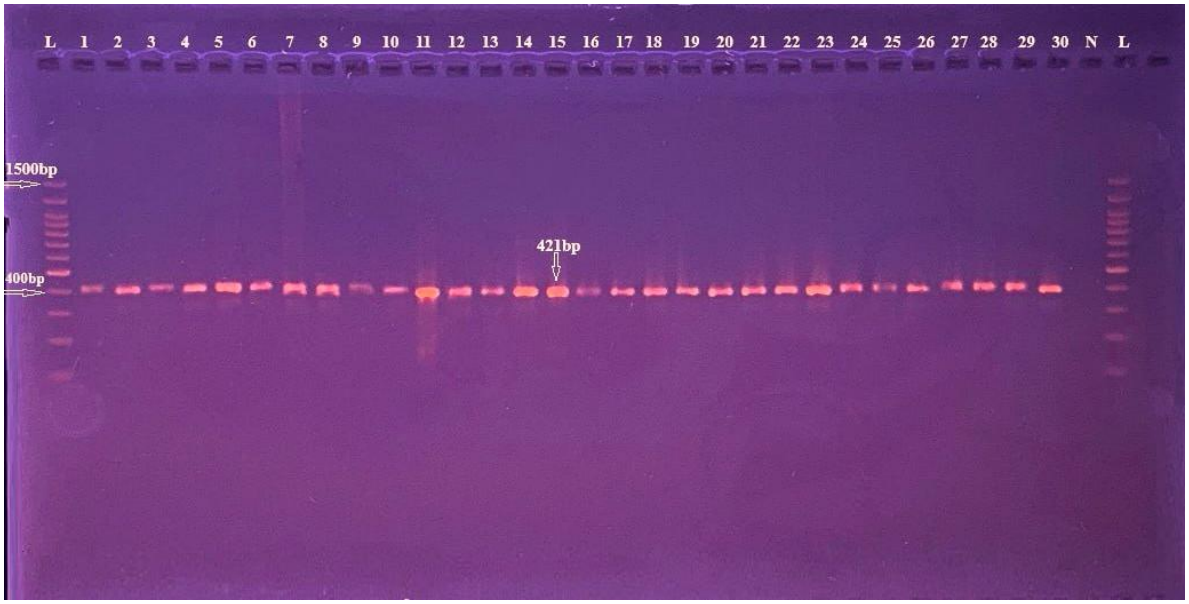

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Figure 13B. PCR products of P53 gene using primers that were unique to a 421bP region were electrophoresed on agarose gel. The samples in the lanes 1-30 represent individual samples of DNA and the amplification was observed to be clear at the anticipated product size. Lane L is the DNA size marker (400-1500 bp), whereas lane N is the negative control (no amplification).The results corroborate the selectivity and efficacy of the P53 amplified reaction.

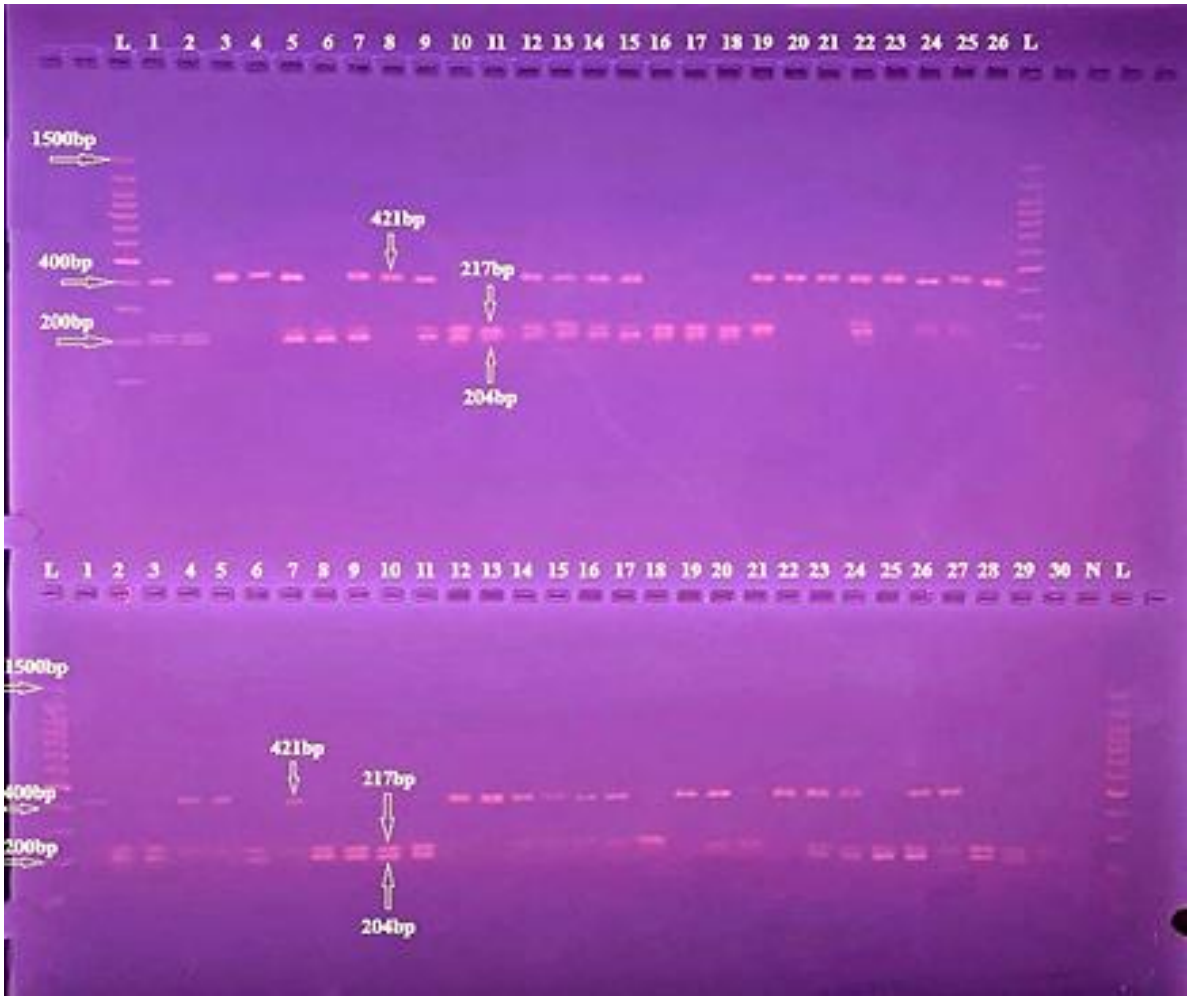

Figure 14. A gel electrophoresis photograph showing RFLP analysis for the P53 gene (421 bp PCR product) digested with BstFN I enzyme. HPV-positive cancer samples are shown in the top panel (lanes 1–26), whereas HPV-positive wart tissues are shown in the bottom panel (lanes 1–30). The genotypes were determined as GG (421 bp band), GC (421, 217, and 204 bp bands) and CC (217 and 204 bp bands). The lanes labeled L are the DNA ladders and the negative control was lane N. The banding patterns represent the distribution of TP53 polymorphism in HPV-related lesions.

The analysis of the p53 codon 72 (Arg72Pro) polymorphism showed large differences in the research groups (Table 9). G/G genotype was predominant in healthy controls (46.7%), whereas the C/C genotype provided high population spike in genital warts (43.3% as compared to only 18.3% in healthy controls) which indicates strong relationship between C/C and HPV induced warts. This was further corroborated by the protective odds ratio

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of 0.21 (95% CI: 0.07-0.67), which means that individuals with the G/G genotype are at a lower risk of getting warts. The allelic frequency test revealed that, the C allele (Pro) was highly improved in wart sample (60.0%) as compared to controls (35.8%), whereas the G allele (Arg) was also common in controls (64.2%). Surprisingly, the intermediate frequencies of the samples of cervical cancer did not indicate significant correlation ($p>0.05$) but presented equal allelic distribution (50% of G and C alleles). The following genotype and allelic distribution features are illustrated in Figures 15 and 16 where specific genetic profiles across the three groups of research and specific association between the C/C genotype are displayed. and genital warts.

Table 9: *P53* codon 72 (Arg72Pro) polymorphism (N= 116).

Parameter	Control (n=60)	Genital wart (n=30)	Cervical cancer (n=26)
Genotype distribution			
G/G (Arg/Arg)	28 (46.7%)	7 (23.3%)	7 (26.9%)
G/C (Arg/Pro)	21 (35.0%)	10 (33.3%)	12 (46.2%)
C/C (Pro/Pro)	11 (18.3%)	13 (43.3%)	7 (26.9%)
***P*-value**	–	**0.024***	0.227
Allelic frequencies			
G allele (Arg)	77 (64.2%)	24 (40.0%)	26 (50.0%)
C allele (Pro)	43 (35.8%)	36 (60.0%)	26 (50.0%)
Odds ratios (95% CI)			
G/C vs G/G	1.00 (Reference)	0.53 (0.17-1.61)	0.44 (0.15-1.30)
C/C vs G/G	1.00 (Reference)	**0.21 (0.07-0.67)***	0.39 (0.11-1.38)
Hardy-Weinberg Equilibrium			
HWE p-value	0.064	0.094	0.695
Important statistical findings			
Most frequent genotype	G/G (46.7%)	C/C (43.3%)	G/C (46.2%)

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Disease association	-	Significant*	Non-significant

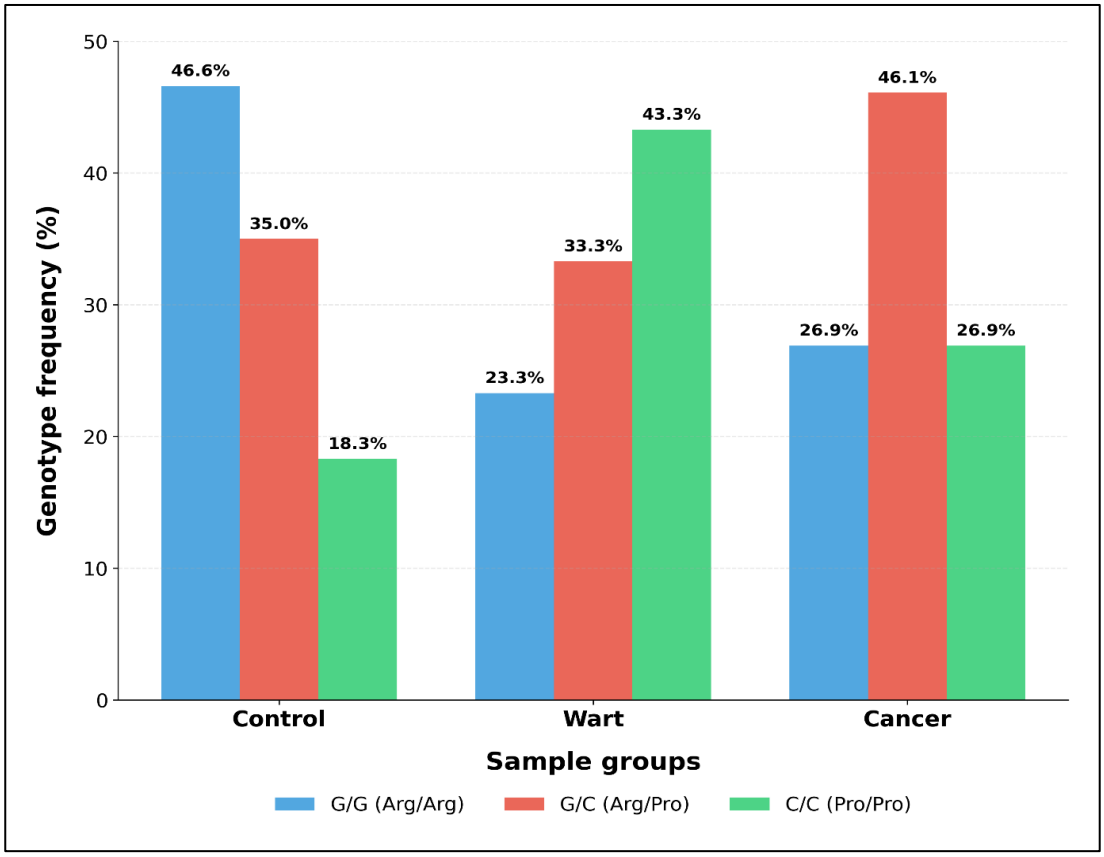

Figure 15. Genotype frequencies of the p53 codon 72 (Arg72Pro) in the cancer, wart and control samples. Bars indicate the percentage of each genotype (G/G, G/C, and C/C) of each sample group. The C/C genotype showed the greatest frequency in wart samples (43.3%), while G/C genotype is the most

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common in cancer specimens (46.1%). The G/G genotype is most prevalent in control samples (46.6%).

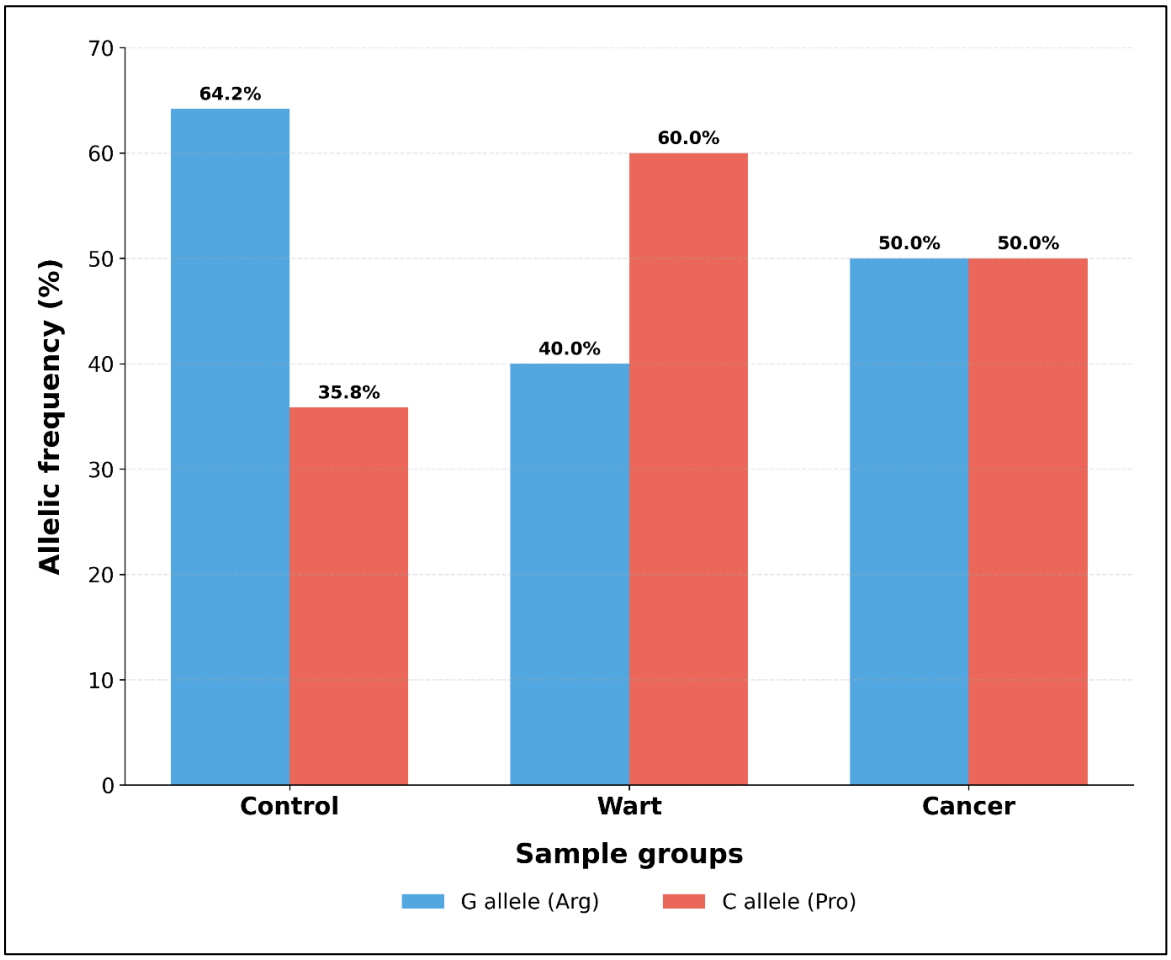

Figure 16. The frequency distribution of the alleles of the p53 codon 72 mutation in the samples of the control, wart, and cancer groups. Horizontal lines are taken to represent the percentage of the G allele (Arg) and C allele (Pro) in the various groups of the samples. The highest frequency of the G allele was in control samples (64.2%), and the wart samples have the highest frequency of the C allele (60.0%). The cancer samples have a balance allelic dispersion having an equal frequency (50.0 each).

Discussion

The age distribution of each of the three groups in this study had significant differences because the age of people with cervical cancer was the oldest (45.3 years), then those with excellent health (33.4 years) and the youngest (27.9 years) was those with genital warts. This discrepancy can be by default because the disease caused by HPV has a natural onset, so younger women are most prone to infection and can take years before turning into cancer [5,6]. Similar age effects have been also observed in women of Iraq with genital warts predominantly affecting younger individuals and cervical cancer being common among older individuals [7,8]. The age of genital warts patients in the younger age group is in line with the onset of sexual activity and exposure to HPV, whereas the older age of cancer patients shows a considerable gap between infection and malignancies [9].

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HPV quick test showed positive percentages of 68 in genital wards sufferers and 60 in cervical cancer individuals, with no positive cases among healthy controls.These rates are much higher than the 17.96% HPV prevalence in Iraq's general population [8], although they are lower than the 84.6% HPV prevalence identified in genital wart patients in Mosul [7]. Since molecular approaches have shown low HPV prevalence in asymptomatic women, the lack of positive results in controls may suggest true negative status or test sensitivity limits [9,10].

The percentage of individuals with a family history of cancer differed dramatically between groups, ranging from 18% in controls to 32% in patients with genital warts and 64% in patients with cervical cancer. This suggests that genetic determinants may influence susceptibility to persistent HPV infection or development to cancer in Iraqi women[11,12]. However, this result might also be impacted by behavioral and environmental traits which households share [13].

The majority of patients with cervical cancer (72%) had intermediate-stage disease, while only 16% had early-stage illness. This delayed presentation may account for significant gaps in cervical cancer screening in Iraq [14,15,16]. Multiple investigations have shown that Iraqi women are not knowledgeable about HPV and cervical cancer testing, and there are presently no readily available systematic screening programs [9,17]. There is an urgent need for programs of screening and HPV vaccination that will allow early identification, as this delayed identification pattern has been consistently recorded throughout Iraq [18].

Significant differences between the p53 codon 72 and INK4a 540 C>T variants in their associations with HPV-related illnesses were shown by the genetic polymorphism study. In particular, the p53 Pro/Pro (C/C) genotype was significantly more prevalent in genital wart patients (43.3%) compared to controls who were healthy (18.3%, p=0.024), suggesting that this variant may increase the risk of benign HPV lesions. Furthermore, the Arg/Arg (G/G) genotype has a beneficial prevalence ratio of 0.21, meaning that individuals with this variant are 80% less likely to have genital warts. These findings are in line with studies that suggest p53 polymorphisms could impact vulnerability to HPV-associated lesions, however the specific associations differ depending on the general population and sickness type [19]. Surprisingly while some literature has reported increased cancer of the cervical cavity risk with the arginine genome variant due to increased susceptibility to HPV E6-mediated decomposition [20], our results demonstrated no significant association between p53 genetic variants and cancer of the cervical gland, which is consistent with several investigations from different communities including South India, Germany, and Argentina (Abba, 2003; Klaes et al., 1999; Pillai et al., 2002) [21,4].

Different pathogenic pathways between both malignant and benign HPV-associated illnesses are suggested by the unique relationship of the Pro/Pro genotypes with warts in the genital area but not cervical cancer. This pattern runs counter to some Asian research that linked the proline variant to a higher risk of cervical cancer [22]. However, the lack of correlation with cancer of the cervical cavity in our sample complements findings from a large, pooled study of 49 research, which identified no consistent relationship with p53 codon 72 polymorphisms and cervical cancer in studies with sound methods [23]. Furthermore, our cervical cancer patients' intermediate allelic frequencies (50% each for the Arg and Pro alleles) imply that other genetic and environmental variables may be more important in this population's transformation into carcinoma.

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The INK4a 540 C>T polymorphism did not significantly correlate with either genital warts or cervical cancer, in contrast to the p53 results. There was a small statistical T/T genotype difference in samples with wart (46.7% and 38.3% in controls, respectively), which was not significant (p=0.725). As well, the T allele was always predominant in all the groups (62.5-66.7%), which showed that the population frequencies were consistent despite the possessions of an illness problem. Since p16/INK4a is a common biomarker of lesions caused by HPV, this absence of correlation is of particular interest. No significant associations between INK4a polymorphism and cervical cancer have been established in the past [24], which implies that genetic variations in the INK4a gene might not have such a significant effect on disease susceptibility despite an increase in p16 protein expression in the infected cells of the HPV virus. Thus, these conflicting interactions between p53 and INK4a polymorphism reflect the complicated genetic environment of the HPV-related pathophysiology and suggest that the specific genetic variables might influence susceptibility to benign and malignant HPV-related illnesses.

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