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Evaluation of the Polymorphic Locus *rs1695* of the *GSTP1* Gene Association with HPV Infection in Women in Burkina Faso

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Abstract

Introduction: Genetic polymorphisms of certain classes of glutathione S-transferase (GST), the enzyme responsible for the biotransformation of drugs and xenobiotics, have been associated with the risk of several cancers such as cervical cancer. The objective of this study was to characterize the impact of the rs1695 polymorphism of *GSTP1* in women infected by high-risk human papillomavirus (HR-HPV) in Ouagadougou, Burkina Faso. **Methods:** Genotyping of *GSTP1* rs1695 polymorphisms was performed in 55 women with high-risk HPV infection, and 89 healthy controls using the PCR-RFLP method. Conventional PCR was used for DNA amplification and the enzymes Alw26I or BsmA1 were used for enzymatic digestion. **Results:** The prevalence of *GSTP1* rs1695 AA, AG and GG genotypes was respectively 27.8%, 45.8% and 26.4% in the study population with a mutation rate of 49.31%. However, the frequency of AA, AG and GG genotypic was respec-

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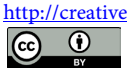

tively 30.3%, 45%, 24.7% in controls and 23.6%, 47.3%, 29.1% in cases. **Conclusion:** Our study allowed us to characterize the frequencies of *GSTP1* rs1695 genotypes in the study population, in cases and controls. From our analysis, none of the three genotypes of *GSTP1* rs1695 weren't associated with a risk or a protective factor for HR-HPV infection in women in Burkina Faso.

Keywords

Polymorphism, *GSTP1*, Women, HPV, Burkina Faso

1. Introduction

Cervical cancer is a pathology of the cervical mucosa due to viral infections caused by Human Papillomavirus (HPV). It is a major public health problem worldwide. Cervical cancer, the third most common cancer in women worldwide after breast and lung cancer and the second most common cancer in Africa after breast cancer, is a leading cause of cancer deaths [1]. According to the World Health Organization (WHO), cervical cancer is a common malignancy in women worldwide, with nearly 604,000 new cases and 342,000 deaths in 2020, with approximately 90% of cases occurring in developing countries [2]. Women in sub-Saharan Africa have the highest prevalence, followed by women in Eastern Europe and Latin America [3]. According to WHO, by 2030, cervical cancer will kill more than 443,000 women per year worldwide, with almost 90% of these deaths occurring in sub-Saharan Africa. According to GLOBOCAN in 2020, cervical cancer is the second cancer in terms of incidence and mortality in Burkina Faso. Human papillomavirus (HPV) infection is recognized as a major factor in the development of cervical cancer [4]. In approximately 99% of cervical cancer cases, women have been exposed to HPV [5]. Several studies have shown that HPV genotypes 16, 18, 45, 31, 33, 52, 58, 35 are the most common in women with cervical cancer [6]. Other exogenous risk factors such as sexual experience with several partners and/or at an early age and smoking have also been the subject of several studies. However, the genetic factors associated with the risk of cervical cancer are still poorly understood and the results are mixed.

Glutathione S-transferase (GST), which is present in almost all eukaryotes, is a phase II multigene superfamily metabolic enzyme [7] [8]. It catalyzes the conjugation of reduced glutathione with a variety of endogenous proteins, exogenous compounds, chemotherapeutic agents and drugs, thereby promoting their elimination from the body. Eight classes of the *GSTs genes* have been identified in mammals [9]. GSTs have well characterized polymorphisms in human populations. The *GSTP1 gene*, which encodes the *GSTP1* enzyme, plays a central role in the inactivation of toxic and carcinogenic electrophiles [9]. Two polymorphic sites in the DNA encoding the *GSTP1* gene (Ile105-Val105 and Ala114-Val114) have been identified [7] [9] [10]. The GSTP1 polymorphism in smoking women has

been associated with a higher risk of developing cervical cancer [11] and may influence cervical cancer susceptibility in northeastern Thai women, either as an independent factor or in combination with high-risk HPV infection [8]. The G allele and AG genotype are specific for the risk of HPV infection in the study population [12] and the G (Val) allele and AG genotype of the GSPTP1 gene have been identified as a risk factor for HPV infection, increasing the risk of infection by approximately four times [12]. Human papillomavirus-16 E7 interacts with glutathione S-transferase P1 and enhances its role in cell survival [13].

This genetic modification may be a biomarker for cancer risk in these individuals. In different populations, these results are inconclusive for cervical cancer [10] by 2030.

Considering the morbidity and mortality caused by HR-HPV induced cervical cancer, the aim of this study was to characterize the rs1695 polymorphisms of *GSTP1* in a subpopulation of HPV-infected women in Ouagadougou, Burkina Faso.

2. Material and Methods

2.1. Study Subjects

The subjects in case-control study were recruited from a large previous study of women in West Africa (UEMOA). The study samples consisted of 144 samples of endocervical cells from sexually active women [6] [14].

2.2. Genotyping of HPV and *GSTP1*

Endocervical specimens were collected from women using a disposable speculum and a sterile swab. The collected samples were frozen in a transport medium at −20˚C at the Pietro Annigoni Biomolecular Research Center (CERBA) in Ouagadougou for molecular analysis.

Genomic DNA extraction from endocervical specimens was performed by using the DNA-Sorb-A extraction kit (Sacace Biotechnologies, Como, Italy) according to the manufacturer's protocol.

The characterization of HR-HPV genotypes was performed using the Sacycler-96 Real Time PCR v.7.3 (SACACE Biotechnologies®, Como, Italy) and the "HPV Genotypes 14 Real-TM Quant" kit (Sacace Biotechnologies, Como, Italy) according to the manufacturer's protocol.

The polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP) method was used to determine the polymorphism of the *GSTP1* gene. The sequences of the primer pairs were: 5'-ACC CCA GGG CTC TAT GGG AA-3' and 5'-TGA GGG CAC AAG AAG CCC CT 3' [7].

The reaction mixture for each sample consisted of 5 µL of DNA, 10 µL of EmilaldAmp PCR Mix; 0.5 µL of each of the primers diluted 1/10 and 9 µL of distilled water to make up a volume of 25 µL.

Amplification of the *GSTP1* gene on the 9700 GeneAmp PCR system was per-

formed according to the following program: the initial denaturation was carried out at 95˚C for 5 min, followed by 50 cycles of 94˚C for 30 s, 55˚C for 30 sec, 72˚C for 30 sec. A final polymerization step at 72˚C for 5 min was performed to complete the process. The PCR product was subjected to 2% agarose gel electrophoresis, a 176 bp fragment was digested with BsmA1 (NEW ENGLAND BioLabs R0529S) according to the manufacturer's protocol and subjected to 4% agarose gel electrophoresis. The gel was visualized using ethidium bromide (BET) staining. BsmAI only recognizes the GTCTC sequence in the G allele to generate a 91 bp fragment and an 85 bp fragment, leaving the A allele to remain uncut (**Figure 1**).

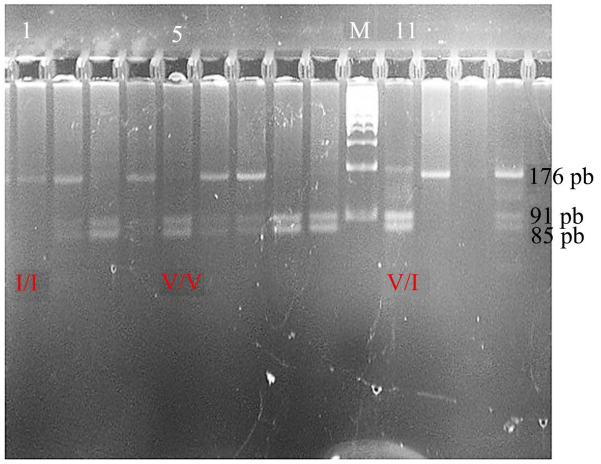

Figure 1. Gel electrophoresis of digestion products. I/I: AA genotype; V/V: Genotype GG; V/I: AG genotype; M: Marker.

2.3. Statistical Analyzes

Genotype and allele frequencies between case and control were compared using Epi-info 7 Chi-squared test. Associations between selected variables were calculated using SPSS V21 for univariate and multivariate regression analyzes with odds ratio (OR) 95% CI. Differences were considered statistically significant if the p-value was <0.05.

2.4. Ethical Considerations

The protocol has been approved by the National Ethics Committee for Health Research (CERS) under consultation number 2018-01-012. The confidentiality and anonymity of the information obtained in the different patient registries will be maintained.

3. Results

This study included 144 samples, of which 89 (62%) were HPV-negative samples and 55 (38%) were HPV-positive samples. The age of the population ranged from 18 to 57 years with a mean of 35.3 ± 9.32 years. The age group of 20 to 40 years

represented approximately 70% of the study population. Approximately 75% of the women were married and had at least a primary education.

3.1. Distribution of HPV-HR Genotypes of Infected Women

Out of the 55 infected women, 24 were positive for at most one HPV genotype, or 47.27%. The genotypes found were HPV 16, HPV 18, HPV 31, HPV 33, HPV 35, HPV 39, HPV 45, HPV51, HPV 52, HPV 56, HPV 58, HPV 59, HPV 66, HPV 68. The prevalence of infection with HPV 59 is the highest with a frequency of 30.9%. We note the absence of HPV 33 and HPV 66 genotypes (Figure 2).

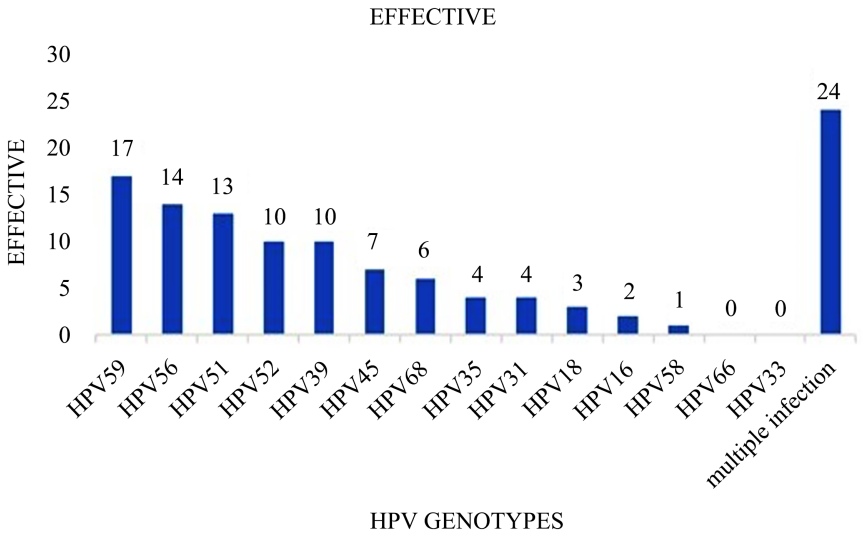

Figure 2. Frequency of HR-HPV genotypes in infected women.

3.2. Genotype and Allele Frequencies (*GSTP1* rs1695) in the Study Population

The frequencies of the AA, AG and GG genotypes in the study population were 27.8% (40/144), 45.8% (66/144) and 26.4% (38/144) respectively. The frequencies of the A and G alleles were 50.7% and 49.3% respectively (Table 1).

Table 1. Genotype and allele frequencies of *GSPT1* rs1695 in the study population.

	Genotype *GSTP1* (rs1695)				Alleles frequencies	
		Effective	Frequency (%)	Cumulative Frequency (%)	alleles	Frequency (%)
Genotypes	AA	40	27.8	27.8	A	50.69
	AG	66	45.8	73.6		
	GG	38	26.4	100.0	G	49.31
	Total	144	100.0			

The allele frequencies of the A and G alleles were 47.27% and 52.73% in the cases, 52.8% and 47.2% in the controls (Table 2) respectively.

Table 2. Frequencies of rs1695 *GSTP1* alleles in cases and controls.

SNP	alleles	Cases (frequency)	Control (frequency)	p-value
rs1695	**A**	52 (47.27)	94 (52.8)	Ref
	G	58 (52.73)	84 (47.2)	0.39

3.3. Genotype and Allele Frequencies of *GSTP1* of Cases and Controls

Table 3 shows the distribution of the different genotypes according to the cases and the controls. There was no significant correlation between the *GSTP1* gene genotypes and HPV infection. None of the *GSTP1* genotypes seems to be a risk or protective factor for HR-HPV infection.

Table 3. Genotype and allele frequencies of *GSTP1* in cases and controls.

Genotype	Control n (%)	Cases n (%)	OR (95% CI)	p-value	Total n (%)
	N = 89	N = 55			
AA	27 (67.5)	13 (32.5)	ref.	ref.	40 (100)
GA	40 (60.6)	26 (39.4)	1.35 (0.59 - 3.09)	0.5	66 (100)
GG	22 (57.9)	16 (42.1)	1.5 (0.6 - 3.86)	0.48	38 (100)
AG/GG	62 (59.6)	42 (40.4)	1.4 (0.65 - 3.03)	0.44	104 (100)
allele					
A	47 (52.81)	26 (47.27)	ref.	ref.	
G	42 (47.19)	29 (52.73)	1.24 (0.77 - 2.00)	0.3	

3.4. Genotype Frequencies of *GSTP1* According to HPV Genotypes

Table 4 shows the distribution of the different *GSTP1* genotypes according to the HPV genotypes. There is no significant correlation between *GSTP1* gene genotypes and HPV infection according to HPV genotypes. *GSTP1* genotypes do not appear to be a risk or protective factor for HR-HPV infection.

Table 4. Genotype and allele frequencies of *GSTP1* and HPV.

HPV genotype		*GSTP1* genotype			p-value	Total
		AA	**AG**	**GG**		
HPV 16 & 18	Yes	0	3	1	**0.38**	4
	No	40	63	37		140
HPV 30'S	Yes	6	4	6	**0.20**	16
	No	34	62	32		128
HPV 50'S	Yes	11	20	12	**0.85**	43
	No	29	46	26		101
HPV 60'S	Yes	1	3	2	**0.81**	6
	No	39	63	36		138

Legend: HPV 30'S: HPV (30 - 39); HPV 50'S: HPV (50 - 59); HPV 60'S: HPV (60 - 69).

4. Discussion

The aim of this study was to characterize the genotypes of the GSTP1 gene (rs1695) for the first time in a population of women in Burkina Faso.

The most common genotype in this study among HPV-infected women was HPV59. However, other studies conducted in Burkina Faso have shown genotypes other than HPV 59. Ouédraogo *et al.* in 2018 in Tenkodogo and in 2020 in Garango in a general population found the HPV genotype 56 to be the most common [15] [16]. Zohoncon *et al.*, in 2013 in Ouagadougou and in 2016 in Parakou had found HPV 35 and 39 genotypes to be the most common [17] [18]. These discrepancies could be explained by regional differences or biases related to the nature of the samples. A large national study could clarify the issue and determine whether this reflects true emergence or an artifact related to the characteristics of the cohort.

In this study, more than 50% of HPV infections cases have the 50'S genotype. This confirms the results of Djigma *et al.* in 2011 in a population of HIV-positive women in Ouagadougou [19]. Multiple infection was detected in 47.27% of this study population. These results are comparable to those of Ouédraogo *et al.* who found 42.2% in a population of sexually active adolescents in Ouagadougou in 2015 [20].

In the present study, the frequencies of the AA, AG and GG genotypes are 30.3%, 45% and 24.7% in HR-HPV negative women and 23.6%, 47.3% and 29.1% in HR-HPV infected women, respectively. The frequencies of the A and G alleles are 47.27% and 52.73% in cases and 52.8% and 47.2% in controls, respectively. Our results showed that the polymorphisms of GSTP1 rs1695 A and G alleles were not associated with HPV infection in our study population. These results are consistent with those of Zhao *et al.* in 2017 and Tian *et al.* in 2019.

The genotypic frequencies of GSTP1 rs1695AA, AG and GG were 27.8%, 45.8% and 26.4%, respectively. The prevalence of individuals with a double mutation was 26.4%. Our results are comparable to those observed in Mali by Kassogue *et al.* who worked on the genetic polymorphisms of GST in the general population of Mali, whose prevalence of genotypes AA, AG, GG were 27.73%, 49.03% and 25.24% respectively [21]. However, we have noticed that the frequency of the double mutation is 5 to 10 times higher than that observed by Saleh *et al.* in 2019 (1.7%) in a population of Yemeni, Cho *et al.* in 2005 (2.5%) in the population Korean; Palma *et al.*, in 2010 (3.6%) in an Italia population with cervical cancer; Sharma *et al.*, in 2014 (4.40%) in a New-Delhi population in India [9] [22]-[24]. We noticed a low mutation rate in white-skinned populations. This significant difference in the frequency of the double mutation could be explained by the effects of the different exposures of the climate, the way of life for the adaptation of the human species according to the countries and the socio-cultural traditions of each people [9].

GSTP1 plays a central role in the inactivation of toxic and carcinogenic electrophiles [9]. The enzyme activity of GSTP1 is significantly lower in individuals with

the 105Val allele due to the polymorphism of nucleotide 313 in the *GSTP1* gene, which causes a decrease in enzyme activity and reduces the ability to metabolize certain xenobiotics and carcinogens [10]. Biochemical studies have shown that the *GSTP1* AA genotype was 2 - 3 times less stable [25] and may be associated with gynecological cancer risk [26]. Possible gene-environment interactions or epigenetic factors may account for instability.

In northeastern Thailand, a study by Phuthong *et al.* in 2018 showed that heterozygous AG, high-risk HPV-infected women were associated with a reduced risk of cervical cancer [8]. The 2010 study by Palma *et al.* suggests that the homozygous AA genotype of GSTP1 appears to be associated with a 5.7-fold increased risk of developing cancerous lesions, independent of smoking [23].

On the other hand, a study conducted in Turkey by Kiran *et al.* in 2010 showed that *GSTP1* polymorphisms were not associated with cervical cancer [7]. Meta-analysis studies conducted by Zhao *et al.* in 2017 and Tian *et al.* in 2019 suggested that *GSTP1* rs1695 polymorphism is not associated with the development of CC [10] [26].

In view of these results, we note that the involvement of *GSTP1* rs1695 in HR-HPV infection towards the development of cervical cancer varies from region to region and also from person to person.

5. Conclusion

This study allowed us to characterize HR-HPV in cases of infected women and to characterize the rs1695 polymorphisms of the *GSTP1* gene in a subpopulation of HR-HPV infected and uninfected women. The HPV 59 genotype is the most frequent genotype in this study. The *GSTP1* rs1695 polymorphisms are similar in the population of HPV-infected and uninfected women. No association was found between the *GSTP1* rs1695 polymorphism and HR-HPV infection. A study on samples of precancerous lesions and large cervical cancer is needed to confirm these results.

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Informed Consent Statement

See ethical approval.

Data Availability

The datasets used and/or analyzed during the current study are available from the

corresponding author on reasonable request.

Authors' Contributions

Conceptualization, data curation, formal analysis, methodology, writing original draft, writing review and editing by MAS, PZ, ZD and FWD.

FWD, MAS, PZ, LT, ZD, TWCO, MAET, TL, RAO, TMZ, DOB, PAS, PB, AB, ATY, SDK, CO, OML and JS contribute to the samples analysis, validation, review and editing the manuscript.

FWD mobilized the funding for the study.

All authors read and approved the final version of the manuscript.

Conflicts of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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