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RESEARCH ARTICLE

MSPI POLYMORPHISMS (RS4646903) OF THE CYP1A1 GENE IN WOMEN INFECTED WITH HUMAN PAPILLOMAVIRUS IN WEST AFRICA

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ABSTRACT

Background: Human papillomavirus (HPV) infection alone is not enough to induce malignant transformation due to viral clearance. Genetic factors could play a very important role in the progression. Genes such as cytochrome P450 1A1 (CYP1A1) could affect the progression of cervical lesions. The objectives of this study were to screen HPV-positive women and to determine CYP1A1 gene polymorphisms in human papillomavirus infections in West African women. **Methods:** For this study, a total of 1215 samples taken from endocervical cells, were collected. The women came from 5 countries in West Africa. These samples were analyzed by multiplexed real-time PCR to search for fourteen high-risk HPV genotypes (HR-HPV). The mono-nucleotide polymorphism MspI was determined using the RFLP PCR technique. **Results:** Of the 1215 swab specimens, 367 (30.28 %) were HPV positive and 848 (69.80 %) HPV negative. High-risk characterized genotypes were in descending order: HPV 66 (12.81 %), HPV 59 (10.85 %), HPV 52 (10.68 %), HPV 51 (9.25 %), HPV 45 (7.83 %), HPV 39 (6.94 %), HPV 56 (6.05 %), HPV 35 (5.87 %), HPV 58 (5.87 %), HPV 18 (5.52 %) , HPV 31 (3.38 %), HPV 33 (2.49 %), HPV 16 (2.14 %). This study also revealed the presence of the CYP1A1 genotype in women with HR-HPV infection. It also showed a decrease in the frequency of CC genotype in women with HR-HPV infection (3.45 %) compared with controls (7.28 %). **Conclusion**: This study showed a high prevalence of HPV 66, HPV 35 and HPV 59. The prevalence of HPV 18 and HPV 16 are among the lowest in this study. However, the results of the research demonstrated that the rs4646903 mutation of the CYP1A1 gene was not statistically related to a risk of acquiring HPV infection in the West African population.

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INTRODUCTION

Human papillomavirus (HPV) infection is one of the most common sexually transmitted infections (STIs) in the world (Dunne *et al.*, 2007, Bruni *et al.*, 2010). More than half of adults are infected during their lifetime. HPVs are involved in many cancers namely cervical cancer, anogenital and oropharyngeal cancers, benign lesions such as condyloma acuminata and warts (Monsonego, 2006). Cervical cancer is the second most important cancer among women worldwide, causing an estimated 266,000 deaths a year (Ding *et al.*, 2018). In sub-Saharan Africa, there are more than 75,000 new cases of invasive cervical cancer and more than 50,000 deaths per year (Ferlay *et al.*, 2010). In Burkina Faso, the prevalence of HR-HPV infection was 52.56 % (Salambanga *et al.*, 2019) ; this rate is really high and poses a real public health problem. In addition, the circulating high-risk genotypes that can cause precancerous lesions and then cancer, are not mainly composed of the HPV 16 and HPV 18 genotypes, but rather genotypes which are not taken into account by the currently available vaccines (Sagna *et al.*, 2010, Djigma *et al.*, 2011, Ouedraogo *et al.*, 2011, Zohoncon *et al.*, 2013, Ouedraogo *et al.*, 2015, Traore *et al.*, 2016a, Traore *et al.*, 2016b, Zohoncon *et al.*, 2016, Ouedraogo *et al.*, 2018, Salambanga *et al.*, 2019). Fortunately, most HPV infections regress spontaneously. Only 1 % of HPV infections cause invasive cancer (Barbisan *et al.*, 2011).

HPV infection alone is not enough to induce malignant transformation (Brestovac *et al.*, 2013). As a result, there are other potential environmental, epigenetic or genetic risk factors (McCann *et al.*, 1992, Goodman *et al.*, 2001). Cytochrome P450 1A1 (CYP1A1) is a key enzyme in the CYP1 family linked to the metabolism of many endogenous and pro-carcinogenic environmental substrates. CYP1A1 may contribute to the formation of highly reactive intermediate metabolites and these metabolites may form adducts of deoxyribonucleic acid (DNA) which, if clogged, could initiate or promote oncogenesis (Ding *et al.*, 2018). A mono-nucleotide polymorphism (SNP) of CYP1A1 at position 3801 in the 3' non-coding region causes the change from base T to C (Spurr *et al.*, 1987, Tan *et al.*, 2016).

Several mono-nucleotide polymorphisms have been identified in the CYP1A1 gene, all located on chromosome 15q22. Such polymorphisms have been considered to play an important role in determining individual susceptibility to many cancers, including cervical cancer (Ding *et al.*, 2018). This polymorphism has already been studied in many cases of malignant tumors (Kim *et al.*, 2000, Goodman *et al.*, 2001, Joseph *et al.*, 2006, Juarez-Cedillo *et al.*, 2007, Gutman *et al.*, 2009, Sergentanis *et al.*, 2012, Zhuo *et al.*, 2012, Xia *et al.*, 2013, Tan *et al.*, 2016, Ding *et al.*, 2018). The involvement of the MspI polymorphism with cervical cancer has been studied in some populations with mixed results. However, there is still no work on the MspI polymorphism of the CYP1A1 gene correlated with HPV infection in the West African population. It therefore seems important to study the association of HPV infection with polymorphisms of the CYP1A1 gene in our region. The objectives of this study were to screen HPV-positive women, to determine polymorphisms of the CYP1A1 gene in women infected with human papillomavirus in West Africa.

MATERIALS AND METHODS

Site, type and population of the study: The study population consisted of 1215 randomly selected samples from 2133 endocervical samples collected from the cervix of women from the general population. Samples came from four West African countries: Benin, Burkina Faso, Côte d'Ivoire and Niger. HPV sampling and genotyping was conducted in 2017 as part of a previous study funded by the Agence Universitaire de la Francophonie (AUF). In these four countries, five cities were selected as study frameworks according to their importance in terms of population density and geographical location. The distribution of the number of samples per city was 220 on average: This was a cross-sectional study, a control case. High-risk HPV-positive samples were considered high-risk HPV cases and samples as controls. So, we had a total of 367 cases and 848 control.

Collection of cervical specimens and screening for precancerous lesions: Sampling was done by endocervical swabbing of the uterus with a single-use speculum. The samples obtained were immersed in a transport medium of the DNA-Sorb-A kit (Sacace Biotechnologies, Como, Italy) and stored at -20 °C in the laboratory of the various sites, then at the Pietro Annigoni Biomolecular Research Center (CERBA), Ouagadougou, DNA extraction and characterization of high-risk HPV genotypes was performed. Screening for precancerous lesions was performed in women by visual inspection with acetic acid and Lugol (VIA / VIL) immediately after swabbing.

DNA extraction: For HPV genotyping, fresh cell sample DNA was extracted using the commercial kit called "DNA-Sorb-A" from sacace biotechnologies® according to the manufacturer's protocol. For genotyping CYP1A1, human DNA was extracted using the RIDA Biotrim® Xtract Art Kit. No. PGZ001 (R-Biopharm AC, AN deren BergstralƷe 17 6297 Darmstadt, Germany). The DNAs thus obtained were stored at a temperature of -20 ° C. for PCR amplifications.

HR-HPV detection: HR-HPV detection was performed with the Real-TMQuant HPV 14 genotype kit (SACACE Biotechnologies®, Italy) by real-time multiplex PCR. This kit allowed the detection of 14 HPV genotypes at high risk (HPV-16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, 66 et 68). For PCR amplification each sample was entitled to 4 tubes. Each tube contained 15 µL of the reaction mixture and 10 µL of the DNA to be amplified. The four 15 µL tubes consisted of a mixture of Hot Start DNA, PCR-buffer-FRT and respectively the E6 and E7 primers of the 3 to 4 HR-HPV target regions (PCR-mix-1 16, 18, 31, IC, PCR-mix-1 39, 45, 59, IC, PCR-mix-1 33, 35, 56, 68, PCR-mix-1 51, 52, 58, and 66). The amplification program was 1 cycle of 95 °C for 15 minutes, followed by a cycle of 95 °C for 5 sec, 60 °C for 20 sec and 72 °C for 15 sec and finally a cycle of 95 °C for 5 sec, 60 °C for 30 sec and 72 °C for 15 sec. The interpretation of results was done with the Microsoft Excel program named HPV Genotypes 14 Real-TM.xls. (SACCE Biotechnologies®, Italy) according to the manufacturer's protocol.

Genotyping of the CYP1A1 gene: For the CYP1A1 gene polymorphism study, 322 samples were selected from the 1215. Of these 116 were HR-HPV positive (cases) and 206 were HR-HPV negative (controls). Mononucleotide polymorphism was determined using the RFLP (restriction

fragment length polymorphism) PCR technique. We followed the procedure described by Tan (Tan *et al.*, 2016). The DNA was amplified using the following primers: CYP-F (forward): 5'-GAG GAA GAA GAG GAG GTA GCA G-3 '; and CYP-R (reverse): 5'-TGA GGT GGG AGA ATC GTG TGA-3'. The DNA to be used for this mixture must be of very good quality. The reaction mixture for a sample to be analyzed was obtained in the following manner using the Invitrogen kit: a 25 µL master mix containing DNA (with a variable volume as a function of its concentration) consists of 5 µL of DNA, 2.5 µL of 10 X PCR Buffer, 0.75 µL of MgCl 2, 0.5 µL of dNTPs, 0.1 µL of Platinum Taq Polymerase, distilled water up to a volume of 25 µL. Amplification program on the 9700 PCR System (Applied Biosystem USA) was done according to the following schedule: 94 °C for 5 minutes; 94 °C/30s, 55 °C/ 30s, 72 °C/45s all in 35 cycles and a final step of 72 °C/5mn. The resulting amplicons of the PCR were analyzed by gel electrophoresis using a 3 % gel stained with ethidium bromide on a UV transilluminator. MspI digestion produces bands of 24 bp and 155 bp for the homozygous CC variant, bands of 24 bp, 155 bp and 179 bp for the heterozygous TC variant, while the wild type TT variant remains undigested leaving a single 179 bp band.

Data analysis: Statistical analysis and data interpretation were performed using SPSS (Statistical Package for Social Sciences) software version 20.0, Epi Info 7 and Microsoft Office Excel 2013. The Chi square test was used for comparisons. proportions. The difference was significant for $p < 0.05$. Differences in CYP1A1 genotypic and allelic distribution between cases and controls were assessed.

Ethical consideration: This study received the approval of the Ethics Committee for Health Research of Burkina Faso (CERS) (Deliberation no 2018-01-012). Respect for confidentiality and anonymity in relation to the information provided was appropriate.

RESULTS

Socio-demographic characteristics: Table 1 provides information on the socio-demographic characteristics of the women who took part in the study. Of the 1215 participants, 367 (30.20 %) were positive for HPV and 848 had no HPV infection (69.80 %). Most women were between 25 and 45 years old, i.e. 69.21 % for those infected with HPV and 76.3 % for non-infected with HPV. The city of Ouagadougou had the highest prevalence of HPV infection (33.5 %), followed by Abidjan (23.6 %), Parakou (22.07 %), Bobo-Dioulasso (13.08 %) and the lowest prevalence was observed in Niamey (8.17 %). Most women in the study were married and 70.05 % were infected with HPV. It should also be noted that many of these women were illiterate and only 42 out of 367 (11.44 %) women infected with HPV had reached university level. The majority reported having only one sexual partner 82.83 % among infected women compared to 85.14 % for uninfected women. The IVA/IVL values were negative in more than 90 % of cases.

Prevalence of HR-HPV genotypes: The search for 14 HR-HPV genotypes showed that 30.20 % (367/1215) of women were infected with HR-HPV. There were multiple cases of infection with co-infections of three HR-HPV genotypes. Figure 1 provides information on the distribution of high-risk genotypes. The highest prevalence was found at HPV 66 (12.81 %), followed by HPV 59 (10.85 %), HPV 52 (10.68 %). The lowest prevalence was in the HPV 16 genotype (2.14 %).

Frequency of distribution of CYP1A1 genotypes: Figure 2 shows the result of electrophoresis genotypes of the CYP1A1 gene (CC, TC, TT) after digestion with the restriction enzyme MspI. Table 2 shows the frequency of CYP1A1 genotype in women with HR-HPV infection compared with uninfected controls. In 332 samples, there was a decrease in the frequency of the CC variant genotype in women with HR-HPV infection (3.45 %) compared with controls (7.28 %). However, this decrease in the CC variant genotype in cases of infection was not statistically significant in the general study population (OR = 0.46, 95 % CI = 0.15 to 1, 46, p = 0.2). There was no significant difference between the TC heterozygous genotype and the TT wild type with respect to the risk of cervical cancer.

Frequency of distribution of the C and T alleles: The frequencies of CYP1A1 alleles in women with HR-HPV infection and controls are shown in Table 3. The frequency of different alleles is substantially the same in HPV cases as in controls in the study population.

DISCUSSION

The objectives of this study were to screen HPV positive women and to determine CYP1A1 gene polymorphisms in West African women infected with human papillomaviruses. This study concerned four West African countries and five cities: Benin (Parakou), Burkina Faso (Ouagadougou and Bobo-Dioulasso), Ivory Coast (Abidjan), Niger (Niamey). Molecular characterization gave the most prevalent genotypes HPV 66 (12.81%), HPV 59 (10.85%), HPV 52 (10.68%), followed by genotypes HPV 51 (9.25%), HPV 45 (7.83%), HPV 39 (6.94%), HPV 56 (6.05%), HPV (5.87%), HPV 58 (5.87%); finally, HPV 18 (5.52%), HPV 31 (3.38%), HPV 33 (2.49%), HPV 16 (2.14%). The HPV 16 and HPV 18 genotypes are among the lowest prevalence. Similar results have been found in previous studies in Burkina Faso and the subregion (Djigma *et al.*, 2011, Zohoncon *et al.*, 2013, Ouedraogo *et al.*, 2015, Traore *et al.*, 2016a, Traore *et al.*, 2016b, Ouedraogo *et al.*, 2018). The high prevalence of high-risk genotypes other than 16 and 18 was found in precancerous and cancerous lesions in Burkina Faso and Benin (Zohoncon *et al.*, 2016, Zohoncon, 2016).

In this study, the HR-HPV prevalence was 30.20 %. These results were higher than those found by Traore et al in Bobo-Dioulasso which was 25.40 % (Traore *et al.*, 2016a). While recently, Salambanga et *al.* (2019) found a high prevalence of HR-HPV of 52.56% in the city of Ouagadougou. Fortunately, not all HR-HPV infections consistently evolve into cervical cancer because of human factors that regulate clearance.

Table 1. Sociodemographic characteristics

	HPV+		HPV-		OR	(95 % CI)	p-value
	N	%	N	%			
Age group							
< 25	57	15.53	89	10.5	Ref.		
25 - 45	254	69.21	647	76.3	0.61	0.42 - 0.88	0.01
> 45	56	15.26	112	13.2	0.78	0.49 - 1.23	0.3
Total	367	100	848	100			
Cities							
Abidjan	85	23.16	165	19.46			0.003
Bobo-Dioulasso	48	13.08	186	21.94			
Ouagadougou	123	33.52	111	13.09			
Niamey	30	8,17	220	25,94			
Parakou	81	22,07	166	19,57			
Total	367	100	848	100			
Marital status							
Single	100	27,25	112	13,21			0,5
Divorced	3	0,8	1	0,11			
Married	257	70,05	720	84,91			
Widow	7	1,9	15	1,77			
Total	367	100	848	100			
Educational level							
Illiterate	118	32.15	310	36.56			0.06
educated	41	11.17	106	12.5			
Primary	57	15.54	127	14.98			
Secondary	109	29.7	222	26.18			
University	42	11.44	83	9.78			
Total	367	100	848	100			
Number of sexual partners							
0	12	3.27	14	1.65			0.5
1	304	82.83	722	85.14			
2	18	4.9	44	5.19			
3	31	8.45	64	7.55			
No information	2	0,55	4	0,47			
Total	367	100	848	100			
VIA							
Positive	28	7,63	50	5,9	1,31	0,81 - 2,12	0,2
Negative	339	92,37	798	94,1			
Total	367	100	848	100			
VILI							
Positive	31	8,45	57	6,72	1,28	0,81 - 2,01	0,2
Negative	336	91,55	791	93.27			
Total	367	100	848	100			

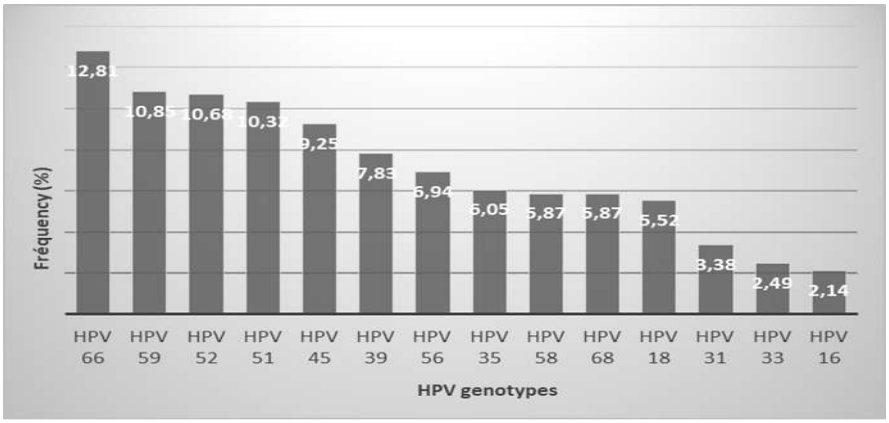


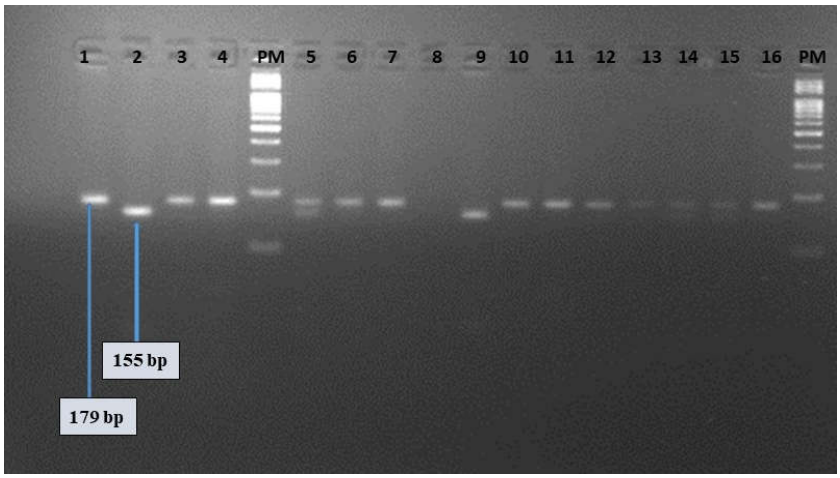
Figure 1. Frequency of HR-HPV genotypes

Table 2. Frequency of CYP1A1 genotype

Genotypes	Cases		Controls		OR	(95 % CI)	P-value
	N = 116	%	N = 206	%			
TT	71	61.2	125	60.68			Rf
TC	41	35.35	66	32.04	1.09	0.67 - 1.77	0.8
CC	4	3.45	15	7.28	0.46	0.15 - 1.46	0.2
Total	116	100	206	100			

Table 3. Frequency of alleles of the CYP1A1 TC polymorphism

Alleles	Cases		Controls		OR	(95 % CI)	P-value
	N = 116	%	N = 206	%			
T	112	78.88	191	76.7			Rf
C	45	21.12	81	23.3	1.05	0.68 - 1.62	0.8
Total	157	100	272	100			



Legend of Figure 2: Lanes 1, 3, 4, 6, 7, 10, 11, 12, 13, 16 represent an undigested 179 bp band in which the restriction enzyme site was not present and indicating that the samples were TT so homozygous wild type. Lanes 5, 14, 15 show the bands of 179 bp and 155 bp indicating that the samples are TC thus heterozygous. Lanes 2 and 9 show complete digestion of the amplicon, giving only the 155 bp band indicating that the samples have the homozygous CC variant.

Figure 2. Electrophoresis after digestion with the restriction enzyme MspI

At the polymorphism of the CYP1A1 gene, it was found that the CC genotype had a low prevalence in HR-HPV infected subjects (3.45%) compared to 7.28% in control subjects. This low prevalence has also been observed in some studies, particularly in Mexico, with 5 % in control and 21 % in cases (Juarez-Cedillo *et al.*, 2007). Results similar to this study were observed in Chhattisgarh, Turkey. Indeed, Jain et al. found in 200 subjects, including 100 cases and 100 controls, that the association of CYP1A1 polymorphism showed no significant relationship with cervical cancer patients (p = 0.23) (Jain *et al.*, 2017). Gutman *et al*. (2009) concluded that mutations in CYP1A1 and CYP2D6 were unrelated to an increased risk of cervical cancer in the Israeli Jewish population. For him, only smoking was the independent risk factor for cervical cancer (p = 0,0003) (Gutman *et al.*, 2009). In contrast, other studies have found that the CC genotype is associated with invasive carcinoma of the cervix, particularly with respect to the wild-type TT genotype. This is the case observed among women in a multiethnic population in Malaysia (Tan *et al.*, 2016). Similarly, in Mexico, in a population where smoking is common, a study was conducted in 310 women including 155 cervical cancer patients and 155 healthy controls. (Juarez-Cedillo *et al.*, 2007). The CC genotype presented an increased risk of cervical cancer. Also, in Mexico, women with TC or CC genotype have a 3 to 9-fold increase in cervical cancer, respectively, compared to women with wild-type TT. In Hawaii, the CC genotype has been shown to be a risk factor for carcinogenesis (Goodman *et al.*, 2001). In China, Li et al also found a significant association between CYP1A1 gene polymorphism and cervical cancer OR 1.45 (1.20 - 1.95) (Li *et al.*, 2016).

Conclusion

This study represents a first in West Africa. The HR-HPV genotypes found once again confirm the high prevalence of other types other than HPV 16 and HPV 18 that are both covered by vaccines. In addition, the study presents the CC homozygous genotype (with two mutated alleles) as a protective factor against HPV infection rather than risk. There is still limited research on the association effects between HR-HPV infection and CYP1A1 gene polymorphisms. It would therefore be necessary to conduct large-scale studies with a large sample size in order to clarify forever the role of the CYP1A1 gene in HPV-dependent carcinogenesis.

Conflict of interest: The authors state that there is no conflict of interest in the publication of this article.

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