



Oncogenic human papillomavirus infection and genotypes characterization among sexually active women in Tenkodogo at Burkina Faso, West Africa



Rogomenoma Alice Ouedraogo^{a,b}, Théodora Mahoukèdè Zohoncon^{a,b,e}, Sindimalgdé Patricia Guigma^{a,e}, Ina Marie Angèle Traore^{a,b}, Abdoul Karim Ouattara^{a,b}, Marie Ouedraogo^{b,e}, Florencia Wendkuuni Djigma^{a,b}, Dorcas Obiri-Yeboah^f, Charlemagne Ouedraogo^{a,c,d}, Jacques Simpore^{a,b,e,*}

^a Laboratory of Molecular Biology and Genetics (LABIOGENE), University of Ouagadougou, 03 BP 7021 Ouagadougou 03, Burkina Faso

^b Pietro Annigoni Biomolecular Research Centre (CERBA), 01 BP 364 Ouagadougou 01, Burkina Faso

^c Obstetrician-gynecologist, UFR/SDS, University of Ouagadougou, 03 BP 7021 Ouagadougou 03

^d Yalgado Ouedraogo University Hospital (CHU/YO), 03 BP 7022, Burkina Faso

^e Faculty of Medicine, University Saint Thomas d'Aquin, 06 BP 10212 Ouagadougou 01, Burkina Faso

^f University of Cape Coast, School of Medical Sciences, Department of Microbiology and Immunology, University Post Office, Ghana

ARTICLE INFO

Keywords:

High-risk HPV
Real time PCR
Genotypes
Multiple infections
Tenkodogo

ABSTRACT

Objective: This study was conducted to determine the prevalence and distribution of high-risk human papillomavirus (HR-HPV) genotypes among sexually active women in Tenkodogo, Burkina Faso.

Methods: Among 131 sexually active women attending the Tenkodogo Urban Medical Center, endocervical samples were collected prior to screening for precancerous lesions. After viral DNA extraction, fourteen HR-HPV genotypes were characterized by real-time multiplex PCR in these cervical samples.

Results: The mean age was 35.5 ± 9.5 years. Of the 131 women, 45 were infected with at least one HR-HPV genotype. The prevalence of HR-HPV infection among these women was 34.4%. Among the 45 oncogenic HPV-infected women, single HR-HPV genotype was found in 55.6% while 44.4% were infected with more than one HR-HPV genotype. The most frequent genotypes were HPV56 (36.5%), HPV66 (36.5%).

Conclusion: Tenkodogo women included in this study had a higher prevalence of HPV 56, HPV 66. A larger study with a more representative sample would therefore be needed to determine predominant oncogenic genotypes in the subregion and especially in cancer cases.

1. Introduction

Oncogenic HPV (HR-HPV) genotypes are closely related to malignant tumors. Sexually active women are at risk of genital HPV infection and the persistence of this infection is necessary for the development of cervical cancer [1,2], which is the leading cause of cancer death among women in Sub-Saharan Africa. HPV-related cancers in women remain a real public health problem. Currently available bivalent and quadrivalent vaccines target only HPV 16 and 18, leading causes of cervical cancer worldwide. The nonavalent vaccine detects additional high-risk genotypes (HPV16, 18, 31, 33, 45, 52, and 58). HPV studies in Burkina Faso were limited to two regions (central and the high basins) and had shown diverse predominance of HR-HPV genotypes in women by region [3–8]. Other genotypes would be more common in the African population, particularly in Burkina Faso [4,7,8], hence it is of importance to

characterize HR-HPV circulating in order to consider adequate prevention strategies. The objective of this study was therefore to contribute to the identification of HR-HPV circulating in the health region of eastern central Burkina Faso, particularly in the city of Tenkodogo.

2. Methods

2.1. Site and study population

This cross-sectional study was conducted in Tenkodogo (located in the health region of eastern central Burkina Faso). From April to May 2016, at the Urban Medical Center of Tenkodogo (CMU), 131 sexually active and consenting women provided cervical samples for this study. Only non-pregnant sexually active women in non-menstrual period who had not undergone a total hysterectomy were included in the study.

* Correspondence to: Jacques Simpore, Pietro Annigoni Biomolecular Research Centre (CERBA)/LABIOGENE, University of Ouagadougou, 01 BP 364 Ouagadougou 01, Burkina Faso.

<https://doi.org/10.1016/j.pvr.2018.09.001>

Received 22 May 2018; Received in revised form 13 September 2018; Accepted 16 September 2018

Available online 19 September 2018

2405-8521/ © 2018 The Authors. Published by Elsevier B.V. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

Each woman answered to a questionnaire that collected information on socio-demographic, behavioral and sexual characteristics, level of knowledge on HPV and cervical cancer.

2.2. Endocervical samples collection

Samples have been taken through endocervical swabbing of the uterus using a sterile cotton swab and a single use speculum. The samples thus obtained have been immersed in a transport medium which was provided with DNA-Sorb-A kit (Sacace Biotechnologies, Como, Italy) and kept at -20°C in the medical analysis laboratory of Regional Hospital (CHR) of Tenkodogo, then at the Pietro Annigoni Biomolecular Research Center (CERBA) of Ouagadougou until the DNA extraction.

2.3. Screening for precancerous lesions

Immediately after sampling, screening for precancerous lesions was done for the women by visual inspection with acetic acid and Lugol's iodine (VIA/VIL).

2.4. Ethics approval and consent to participate

This study was approved by the Ethics Committee for Health Research of Burkina Faso (Deliberation number 2016–3-026) as well as that of the Regional Office of Health (DRS) of the Central-East Health Region. All participants gave their free and written consent to participate in the study. Confidentiality and anonymity with respect to the information collected were required.

2.5. HPV DNA extraction

The viral DNA was extracted using the DNA-Sorb-A kit (Sacace Biotechnologies, Como, Italy) following the protocol supplied by the manufacturer.

2.6. Determination of HPV genotypes by real time PCR

Characterization of HR-HPV genotypes, was performed using 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), which allowed us to detect fourteen genotypes of HR-HPV (HPV 16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, 66, 68) using multiplex real time-PCR for each sample with the β -globin gene as internal control. The PCR program used was as follows: 1 cycle of 95°C for 15 min; 5 cycles of 95°C for 05 s, 60°C for 20 s, 72°C for 15 s; 40 cycles of 95°C for 05 s, 60°C for 30 s, and 72°C for 15 s.

2.7. Data analyses

Data were recorded and analyzed using IBM SPSS V.21 (SPSS, Chicago, Illinois, USA) and Epi Info 6. The Chi-square test was used for comparisons with a significant difference for $p < 0.05$.

3. Results

During the enrollment period in the Department of obstetrics and gynecology, 135 sexually active women gave their consent to participate in this study. The main motivation for consultation was to take advantage of the cervical cancer screening, symptoms of leucorrhea and pruritus, abdominal pelvic pain, dysmenorrheal, menstrual cycle disorders, requirement of contraception and fertility.

3.1. Sociodemographic characteristics of the study population

The mean age of women in our study was 35.5 ± 9.5 years

Table 1
Sociodemographic, sexual and behavioral characteristics of study population.

Characteristics	Number (n = 131)	Percentage (%)
Age group in years		
20–24	14	10.7
25–29	29	22.1
30–34	18	13.8
≥ 35	70	53.4
Level of education		
Illiterates	72	55.0
Primary	28	21.3
High school	31	23.7
Marital status		
Married or lives with a partner	114	87.0
Single	5	3.8
Widow	9	6.9
Divorced	3	2.3
Number of sexual partner		
1	130	99.2
≥ 2	1	0.8
Occupation		
Housewives	42	32.1
Pupils	7	5.3
Civil servants	9	6.9
Informal sector	73	55.7
Use of contraception		
No/Natural method	48	36.6
Yes	83	63.4
Use of condom		
Yes	27	20.6
No	104	79.4
Age at the first sexual intercourse		
14–17	38	29.0
18–24	92	70.2
≥ 25	1	0.8
Number of pregnancies		
0	5	3.8
1–5	85	64.9
5–11	41	31.3
Knowledge on HPV and cervical cancer		
Yes	25	19.1
No	106	80.9

(20–60) and the median was 35 years old. Women older than 35 years were significantly more represented (53.4% or 70/131). Married women or lived with a partner were 87.0% (114/131); 55.0% were illiterate (72/131), 55.7% (73/131) were working in the informal sector and only 12.2% (16/131) were students or civil servants. The age at first intercourse ranged from 14 to 30 years with an average of 18.8 ± 2.1 . Only one woman reported having more than one sexual partner; the others had only one. The majority of women (96.2%) had been pregnant at least once in their lifetime and 43.5% had at least 5 children. More than half of women (63.4%) used a contraceptive method. In addition, 79.4% had never used a condom during intercourse and 80.9% had no knowledge of cervical cancer and Human Papillomavirus infection at the time of inclusion in this study (Table 1).

3.2. Prevalence of HR-HPV infection and genotypes characterization in women in Tenkodogo

The amplification kit used allowed us to identify fourteen HR-HPV genotypes (HPV 16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, 66 and 68) among which we found 12 genotypes in women in Tenkodogo. HPV 39 and HPV 59 were not found. Among 131 women, 45 were infected with at least one HR-HPV genotype. The prevalence of HR-HPV infection among these women was 34.4% (CI = 26.4–43.2). The total number of genotypes per infected woman ranged from 1 to 5. Considering multiple infections, we counted a total of 74 HR-HPV genotypes. Of these genotypes, the most frequent were in descending order HPV56 (36.4%), HPV66 (36.4%) followed by HPV68 (5.4%) and HPV51 (5.4%). The frequency of other genotypes ranged from 1.3% to

Table 2
Prevalence and distribution of 14 HR-HPV genotypes in single and multiple infections among women in Tenkodogo.

HR-HPV genotypes	Prevalence n (%)	Confident interval 95%	HR-HPV genotypes associated with multiples and single infection	Prevalence n (%)
			Single Infections	
HPV56	27(36.4)	25.8–48.5	HPV 18	1 (2.2)
HPV66	27(36.4)	25.8–48.5	HPV 31	2 (4.4)
HPV68	4 (5.4)	1.7–13.9	HPV 51	1 (2.2)
HPV51	4 (5.4)	1.7–13.9	HPV 56	10 (22.2)
HPV18	3 (4.0)	1.1–12.1	HPV 58	1 (2.2)
HPV31	2 (2.7)	0.4–10.3	HPV 66	10 (22.2)
HPV35	2 (2.7)	0.4–10.3	Total 1	25 (55.6)
HPV52	1 (1.4)	0.1–8.3	Multiples infections	
			HPV 35/51	1 (2.2)
HPV58	1 (1.4)	0.1–8.3	HPV 56/66	12 (26.7)
HPV45	1 (1.4)	0.1–8.3	HPV 66/68	1 (2.2)
HPV16	1 (1.4)	0.1–8.3	HPV 33/35/56	1 (2.2)
HPV59	–	0.0–6.1	HPV 56/66/68	1 (2.2)
HPV39	–	0.0–6.1	HPV 51/56/66	1 (2.2)
			HPV 18/45/52	1 (2.2)
			HPV18/56/66/68	1 (2.2)
			HPV16/51/56/66/68	1 (2.2)
Total of HR-HPV genotype	74 (100)		Total 2	20 (44.4)
			Total	45

4.1% as shown in Table 2. Among the 45 women infected by the oncogenic HPV, 55.6% (25/45) were infected by only one genotype from HR-HPV and 44.4% (20/45) infected by more than one HR-HPV genotype. The number of HR-HPV genotypes in the 09 multiple infection cases was 49 (66.2%) versus 25 (33.8%) involved in single infections (Table 2). Considering the genotypes covered by the HPV vaccines, 3% (4/131) women had HPV 16 and/or 18, or 9% (4/45) of those infected with at least one HR-HPV genotype; 7.6% (10/131) had one or more of 16/18/31/33/45/52/58 genotypes or 22.2% (10/45) of those infected.

3.3. Potential risk factors associated with the carrying of HPV

In Table 3, we analyzed association between the HR-HPV infection and age, age of first sexual intercourse, level of education, marital status, number of sexual partner, frequency of sexual intercourse, occupation, use of condom, parity, use of contraception, knowledge on HPV and cervical cancer, and VIA/VILI result. Women older than 55 years and those between age 40 – 44 years were less infected (2.2%), the highest carrier rate of HR-HPV (24.4%) was found among the 25–29 age group (Table 3). Visual screening for precancerous lesions showed that 2 of the 131 women or 1.5% were positive for VIA/VILI. Of these two women, one was carrier of HPV as shown in Table 3. This woman was infected with HPV66. Among the 45 women infected with at least one HR-HPV genotype, 88.9% (40/45) had more than two sexual intercourse per month against 6.7% (3/45) with a sexual intercourse per month, while 4.2% (2/45) had two sexual intercourse per month. This study found no statistical association between HPV infection and the risk factors analyzed.

4. Discussion

Voluntary inclusions have been a limitation of the study and therefore subject to selection bias. This may justify the small size of the study cohort and the high prevalence of the HPV 56 and 66 genotypes

Table 3
Potential risk factors associated with the carrying of HPV.

	HPV – n = 86	HPV+ n = 45	Total n = 131	P*
Age group, n(%)				0.07
20–24	8 (57.1)	6 (42.9)	14(10.7)	
25–29	18(62.1)	11(37.9)	29(22.1)	
30–34	10(55.6)	8(44.4)	18(13.7)	
35–39	20(69.0)	9(31.0)	29(22.1)	
40–44	18(94.7)	1(5.3)	19(14.5)	
45–49	3(33.3)	6(66.7)	9(6.9)	
50–54	6(66.7)	3(33.3)	9(6.9)	
≥ 55	3(75.0)	1(25.0)	4(3.1)	
Age of first sexual intercourse, n (%)				0.19
14–17	29 (76.3)	9(23.7)	38(29.0)	
18–24	56(60.9)	36(39.1)	92(70.2)	
≥ 25	1(100)	–	1(0.8)	
Level of education, n (%)				0.06
Illiterates	52 (72.2)	20(27.8)	72(55)	
Primary	19 (67.9)	9(32.1)	28(21.4)	
High school	15 (48.4)	16(51.6)	31(23.7)	
Marital status, n (%)				0.91
Married or lives with a partner	76 (66.7)	38(33.3)	114(87.0)	
Single	3(60.0)	2(40.0)	5(3.8)	
Widow	5(55.6)	4(44.4)	9(6.9)	
Divorced	2(66.7)	1(33.3)	3(2.3)	
Number of sexual partner, n (%)				0.34
1	86(66.2)	44(33.8)	130(99.2)	
≥ 2	–	1(100)	1(0.8)	
Frequency of sexual intercourse, n (%)				0.27
1/Month	14 (82.4)	3 (17.6)	17 (13.0)	
2/Month	5 (71.4)	2 (28.6)	7 (5.3)	
> 2/Month	67 (62.6)	40 (37.4)	107 (81.7)	
Occupation, n (%)				0.53
Housewives	28(66.7)	14 (33.3)	42(32.1)	
Pupils	3(42.9)	4(57.1)	7(5.3)	
Civil servants	7(77.8)	2(22.2)	9(6.9)	
Informal sector	48(65.8)	25(34.2)	73(55.7)	
Use of condom, n (%)				0.65
No	67(64.4)	37(35.6)	104(79.4)	
Yes	19(70.4)	8(29.6)	27(20.6)	
Parity, n (%)				0.96
Nulliparous	3(60.0)	2(40.0)	5(3.8)	
Primiparous	8(66.7)	4(33.3)	12(9.2)	
Multiparous	75(65.8)	39(34.2)	114(87.0)	
Use of contraception, n (%)				1
No/Natural method	32(66.7)	16(33.3)	48(36.6)	
Yes	54(65.1)	29(34.9)	83(63.4)	
Knowledge on HPV and cervical cancer, n (%)				0.49
No	68(64.2)	38(35.8)	106(80.9)	
Yes	18(72.0)	7(28.0)	25(19.1)	
VIA/VILI result, n (%)				1
VIA/VIL –	85(65.9)	44(34.1)	129(98.5)	
VIA/VIL +	1(50.0)	1(50.0)	2(1.5)	

* P: p value for difference in characteristic according to HR-HPV status.

in this subpopulation. The size of our sample does not allow us to generalize. Despite these methodological limitations, the identification of HR-HPV circulating in this region brings us not only complementary results to evaluate the HR-HPV infection level and give information on the distribution of HR-HPV genotypes in Burkina Faso.

4.1. HR-HPV carrying and genotypes characterization among women in Tenkodogo

In this study, the prevalence of HR-HPV infection is high among women in Tenkodogo. Indeed, the overall prevalence of 34.4% carriage of HR-HPV in these women was close to that of 38.3% in Burkina Faso [5], 35.0% in England [9], 33.8% in Costa Rica [10] and 33.2% in

Benin [11]. On the other hand, it was lower than those of 75.0% in Ghana [12], 74.0% in Tanzania [13] and 54.0% in South Africa [14], 41.5% in Burkina Faso [7] obtained from studies of younger populations with a mean age of less than 30 years while that of our study was 35.5 ± 9.5 years.

Although the prevalence of genital HPV infection varies by region around the world [15], all studies agree that it is particularly important in young women at the onset of sexual activity and decreases later when age increases because of both clearance and the acquisition of immunity. Our prevalence could be justified by the mean age of women which was higher than that of the study populations of the last three authors. However, our prevalence was higher than 25.4% in Burkina Faso [6], 24% [16], 15.9% in Great Britain [2] and 7.8% in Italy [17].

In this study, the most common genotypes among women in Tenkodogo were HPV 56 (36.5%) and HPV 66 (36.5%) followed by HPV 68 and HPV 51 with a prevalence of 5.4% for each. The least common genotypes in descending order were HPV 18, 35, 31, 58, 52, 45, 33, and 16. HPV 59 and 39 were not found. Similar studies on HPV conducted in Burkina Faso and Vietnam using the same amplification kit and the same device showed various and predominant genotypes other than 56 and 66 [5–7,18–20].

Contrary to our results, HPV 56 is not currently frequent in the literature but Kavanagh and *al.* (2013) [21] found a high frequency of HPV56 and 66 while these genotypes are not covered by the vaccine. The prevalence of 36.5% of HPV 66 found in our study was higher than that of 9.5% found by Traore and *al.*, (2016b) [5] in a similar study conducted in a population of sexually active women in Banfora.

Among women recruited in Tenkodogo, HPV genotypes 16 and 18 covered by currently vaccines available in Burkina Faso, accounted for 4.1% and 1.4% respectively. The prevalence of HPV18 obtained is lower than that of 5.5% in Great Britain [2], 10.6% in Ghana [12], 14.8% in Bobo-Dioulasso [6], 14.3% [8] and 5.1% [7] in Ouagadougou. The results of these studies thus show a regional difference in the distribution of HPV 18. The low prevalence of single HPV 16 (1.4%) infection would be contrary to the literature according to which genotype 16 is the most common in the world, especially in Europe. [15,22], in the United States [23] and in North Africa [24]. On the other hand, our results corroborate those of some studies that had already shown a low prevalence of HPV 16 in some African countries compared to other HR-HPV genotypes [4–8,25]. Although HPV 16 appears to be the most prevalent in the world and particularly in cases of cervical cancer, studies in the African subregion have shown the existence of other predominant genotypes. In Burkina Faso, the study by Zohoncon and *al.* (2016b) [19] on invasive cervical cancer had revealed a predominance of HPV 18, 31, 39, 16, 45, 35 and 58. In addition, two similar studies conducted in Benin and Burkina Faso in High-Grade Cervical Intraepithelial Neoplasia and in Cervical Cancer showed lack of HPV 16 and a predominance of oncogenic genotypes, 18, 45, 35, 52, 33, 51, 31, 58, 66, 68, 56, 59, 68 [18,19]. This classical difference in the distribution of genotypes in our study population should lead to a larger study with more representative sample, to determine if the frequency of HPV16 circulating in women is lower than other populations and to characterize the predominant oncogenic genotypes in the subregion and particularly in cervical cancer.

Among the infected women in our study, we found 44.4% of women who had two or more types of HR-HPV. In contrast, 55.6% (25/45) was infected with a single HR-HPV genotype. Our prevalence of 44.4% multiple infections in the Tenkodogo HR-HPV positive women population is similar to the 48.1% reported by Kavanagh and *al.* [21] but is higher than that of 12.3% reported by Ouedraogo and *al.* [8] and 15.2% found by Traore and *al.* [6]. The number of isolated HR-HPV genotypes among infected women ranged from 2 to 5 in multiple infections. This result is comparable to those of Traore and *al.* [6] and Ouedraogo and *al.* [7] who found a variation of 2–3 and 2–5 h-HPV genotypes respectively in multiple infections. We did not find HPV 16 and HPV 18 coinfection and our results corroborate those of Traore and *al.*,

Ouedraogo and *al.*, and Pannatto and *al.* [6,7,22].

4.2. HR-HPV carriage and risk factors associated with infection

In this study, the carrying of the HR-HPV was not associated with risk factors such as the frequency of sexual intercourse [26], the age, the age of first sexual intercourse, the level of education, the marital status, the number of sexual partner, the occupation, the use of condom, the parity, the use of contraception, the knowledge on HPV and cervical cancer, or the VIA/VILI result. This lack of statistically significant association could be explained by the small size of our study population. The prevalence of precancerous lesions detected by VIA/VIL was 1.5%. This value is lower than that of 6% and 4.2% obtained respectively for women in Ouagadougou and Bobo-Dioulasso [6,7]. HR-HPV infection was found in 50.0% of women with a positive VIA/VIL result.

Studies have shown a prevalence of HR-HPV infection of 73.5% and 41.5% in women aged 10–25 years [13] and 15–19 years old [7]. In this study, we found that HR-HPV infection was higher in women aged 25–29 years (24.4%). This high prevalence of HR-HPV infection could be explained by the fact that 99.2% of women had their first sexual intercourse between 14 and 24 years of age and 79.4% had never used a condom during sex. As pointed out by Gavillon and *al.*, 2010, this prevalence is inversely proportional to the age of the patient [27].

Among 45 women infected with at least one HR-HPV genotype, 88.9% (40/45) of women had more than two sexual intercourse per month. We did not find a statistically significant association between carriage of HR-HPV and frequency of sexual intercourse ($p < 0.26$). Our results corroborate those of Ouedraogo and *al.* [7] who did not find this correlation in teenage girls in Ouagadougou. However, our result differs from that of Figueroa and *al.* [26] who found a highly significant association between high prevalence of HPV and frequent sexual activity.

The results of this study could not be generalized to the entire population of Tenkodogo but they helped to know the HPV genotypes present in a population of women in this city. Our results show that it would be important to extend HR-HPV studies in all regions of Burkina Faso in more representative populations to provide data on the distribution of genotypes within populations.

5. Conclusion

Tenkodogo women included in this study had a higher prevalence of HPV 56, HPV 66, followed by oncogenic genotypes 68, 51, 18, 35, 31, 58, 52, 45, 33 and 16. HR-HPV other than HPV16 and HPV 18 were frequently found in studies in Burkina Faso and oncogenic genotypes 56 and 66 seem to be the most predominant in our study population. A larger study with a more representative sample would therefore be needed to determine predominant oncogenic genotypes in the sub-region and especially in cancer cases.

Acknowledgements

The authors wish to thank the Saint Camille Hospital in Ouagadougou (HOSCO) and the CERBA/LABIOGENE, University of Ouagadougou, the Regional Directorate of Health and the Medical Center with Surgical Antenna of the Central East Region, Burkina Faso. They express their deep gratitude to the Italian Episcopal Conference (IEC).

Competing interest

The authors declare that there is no conflict of interest regarding the publication of this article.

Funding

This work was supported by the Italian Episcopal Conference (CEI), CERBA/LABIOGENE of the University of Ouagadougou and Saint Camille Hospital of Ouagadougou for the collection, analysis, interpretation of the data and the writing of this article.

References

- [1] N. Munoz, X. Castellsague, A.B. de Gonzalez, L. Gissmann, Chapter 1: hpv in the etiology of human cancer, *Vaccine* 24 (Suppl 3) (2006) (S3/1-S3/10).
- [2] A.M. Johnson, C.H. Mercer, S. Beddows, N. de Silva, S. Desai, R. Howell-Jones, C. Carder, P. Sonnenberg, K.A. Fenton, C. Lowndes, et al., Epidemiology of and behavioural risk factors for, sexually transmitted human papillomavirus infection in men and women in Britain, *Sex. Transm. Infect.* 88 (3) (2012) 212–217.
- [3] F.W. Djigma, C. Ouedraogo, D.S. Karou, T. Sagna, C. Bisseye, M. Zeba, D. Ouermi, C. Gnoula, V. Pietra, N.W. Ghilat-Avoid-Belem, et al., Prevalence and genotype characterization of human papillomaviruses among HIV-seropositive in Ouagadougou, Burkina Faso, *Acta Trop.* 117 (3) (2011) 202–206.
- [4] T.M. Zohoncon, C. Bisseye, F.W. Djigma, A.T. Yonli, T.R. Compaore, T. Sagna, D. Ouermi, C.M. Ouedraogo, V. Pietra, J.B. Nikiema, et al., Prevalence of HPV high-risk genotypes in three cohorts of women in Ouagadougou (Burkina Faso), *Mediterr. J. Hematol. Infect. Dis.* 5 (1) (2013) e2013059.
- [5] I.M.A. Traore, T.M. Zohoncon, O. Ndo, F.W. Djigma, D. Obiri-Yeboah, T.R. Compaore, S.P. Guigma, A.T. Yonli, G. Traore, P. Ouedraogo, et al., Oncogenic human papillomavirus infection and genotype characterization among women in Orodara, Western Burkina Faso, *Pak. J. Biol. Sci. PJB* 19 (7) (2016) 306–311.
- [6] I.M. Traore, T.M. Zohoncon, A. Dembele, F.W. Djigma, D. Obiri-Yeboah, G. Traore, M. Bambara, C. Ouedraogo, Y. Traore, J. Simpoire, Molecular characterization of high-risk human papillomavirus in women in Bobo-Dioulasso, Burkina Faso, *Biomed. Res. Int.* 2016 (2016) 7092583.
- [7] C.M. Ouedraogo, R.M. Rahimy, T.M. Zohoncon, F.W. Djigma, A.T. Yonli, D. Ouermi, A. Sanni, J. Lankoande, J. Simpoire, Epidemiology and characterization of high-risk genotypes of human Papillomavirus in a population of sexually active adolescents in Ouagadougou, *J. De. Gynecol., Obstet. Biol. De. la Reprod.* 44 (8) (2015) 715–722.
- [8] C.M. Ouedraogo, F.W. Djigma, C. Bisseye, T. Sagna, M. Zeba, D. Ouermi, S.D. Karou, V. Pietra, F. Buelli, N.W. Ghilat-Avoid-Belem, et al., Epidemiology, characterization of genotypes of human papillomavirus in a population of women in Ouagadougou, *J. De. Gynecol., Obstet. Biol. De. la Reprod.* 40 (7) (2011) 633–638.
- [9] R. Howell-Jones, A. Bailey, S. Beddows, A. Sargent, N. de Silva, G. Wilson, J. Anton, T. Nichols, K. Soldan, H. Kitchener, et al., Multi-site study of HPV type-specific prevalence in women with cervical cancer, intraepithelial neoplasia and normal cytology, in England, *Br. J. Cancer* 103 (2) (2010) 209–216.
- [10] A.K. Chaturvedi, H.A. Katki, A. Hildesheim, A.C. Rodriguez, W. Quint, M. Schiffman, L.J. Van Doorn, C. Porras, S. Wacholder, P. Gonzalez, et al., Human papillomavirus infection with multiple types: pattern of coinfection and risk of cervical disease, *J. Infect. Dis.* 203 (7) (2011) 910–920.
- [11] F. Piras, M. Piga, A. De Montis, A.R. Zannou, L. Minerba, M.T. Perra, D. Murtagh, M. Atzori, M. Pittau, C. Maxia, et al., Prevalence of human papillomavirus infection in women in Benin, West Africa, *Virology* 438 (2) (2011) 514.
- [12] D. Obiri-Yeboah, P.K. Akakpo, M. Mutocheluh, E. Adjei-Danso, G. Allornuvor, D. Amoako-Sakyi, Y. Adu-Sarkodie, P. Mayaud, Epidemiology of cervical human papillomavirus (HPV) infection and squamous intraepithelial lesions (SIL) among a cohort of HIV-infected and uninfected Ghanaian women, *BMC Cancer* 17 (1) (2017) 688.
- [13] D. Watson-Jones, K. Baisley, J. Brown, B. Kavishe, A. Andreasen, J. Chagalucha, P. Mayaud, S. Kapiga, B. Gumodoka, R.J. Hayes, et al., High prevalence and incidence of human papillomavirus in a cohort of healthy young African female subjects, *Sex. Transm. Infect.* 89 (5) (2013) 358–365.
- [14] D. Adler, F. Laher, M. Wallace, K. Grzesik, H. Jaspán, L.G. Bekker, G. Gray, Z. Valley-Omar, B. Allan, A.L. Williamson, High rate of multiple concurrent human papillomavirus infections among HIV-uninfected South African adolescents, *J. Immunol. Tech. Infect. Dis.* 2 (1) (2013) 1000106.
- [15] G.M. Clifford, M.A. Goncalves, S. Franceschi, Hpv, group HIVS: human papillomavirus types among women infected with HIV: a meta-analysis, *Aids* 20 (18) (2006) 2337–2344.
- [16] D. Forman, C. de Martel, C.J. Lacey, I. Soerjomataram, J. Lortet-Tieulent, L. Bruni, J. Vignat, J. Ferlay, F. Bray, M. Plummer, et al., Global burden of human papillomavirus and related diseases, *Vaccine* 30 (Suppl 5) (2012) F12–F23.
- [17] C. Campari, C. Fedato, A. Petrelli, M. Zorzi, C. Cogo, A. Caprioglio, F. Gallo, L. Giordano, S. Domenighini, L. Pasquale, et al., HPV prevalence and risk of pre-cancer and cancer in regular immigrants in Italy: results from HPV DNA test-based screening pilot programs, *Infect. Agents Cancer* 10 (2015) 14.
- [18] T.M. Zohoncon, T.C. Ouedraogo, L.V.C. Brun, D. Obiri-Yeboah, W.F. Djigma, S. Kabibou, S. Ouattara, M. Gomina, A.T. Yonli, V. Bazie, et al., Molecular epidemiology of high-risk human papillomavirus in high-grade cervical intraepithelial neoplasia and in cervical cancer in Parakou, Republic of Benin, *Pak. J. Biol. Sci. PJB* 19 (2) (2016) 49–56.
- [19] Théodora Mahoukèdè Zohoncon, Prosper Bado, Djénéba Ouermi, Esther M.A. Traoré, Florencia W. Djigma, Ina M.A. Traore, Albert, T. Yonli, Charlemagne Ouédraogo, Simon A. Akpona, Olga Lompo, et al., Human papillomavirus genotypes involved in invasive cervical cancer from formalin-fixed, paraffin embedded tissues in Ouagadougou, Burkina Faso, *Int. J. Curr. Res.* 8 (9) (2016) 39314–39318.
- [20] Song Nguyen Van, Minh Nguyen Khac, Jan Dimberg, Andreas Matussek, Anna J. Henningsson, Prevalence of cervical infection and genotype distribution of Human papilloma virus among females in Da Nang, Vietnam, *Anticancer Res.* 37 (2017) 1243–1248.
- [21] K. Kavanagh, K. Sinka, K. Cuschieri, J. Love, A. Potts, K.G. Pollock, H. Cubie, M. Donaghy, C. Robertson, Estimation of HPV prevalence in young women in Scotland; monitoring of future vaccine impact, *BMC Infect. Dis.* 13 (2013) 519.
- [22] D. Panatto, D. Amicizia, E. Tanzi, S. Bianchi, E.R. Frati, C.M. Zotti, P.L. Lai, A. Bechini, S. Rossi, R. Gasparini, Prevalence of human papillomavirus in young Italian women with normal cytology: how should we adapt the national vaccination policy? *BMC Infect. Dis.* 13 (2013) 575.
- [23] J. Monsonego, J.T. Cox, C. Behrens, M. Sandri, E.L. Franco, P.S. Yap, W. Huh, Prevalence of high-risk human papilloma virus genotypes and associated risk of cervical precancerous lesions in a large U.S. screening population: data from the ATHENA trial, *Gynecol. Oncol.* 137 (1) (2015) 47–54.
- [24] H. Guettiti, E. Ennaifer, L. Attia, D. Chelly, N.B. Alaya, R.B. Aissa, T. Laassili, S. Boubaker, Pre-vaccination prevalence and genotype distribution of human papillomavirus infection among women from urban Tunis: a cross-sectional study, *Asian Pac. J. Cancer Prev.* 15 (21) (2014) 9361–9365.
- [25] L. Bruni, M. Diaz, X. Castellsague, E. Ferrer, F.X. Bosch, S. de Sanjose, Cervical human papillomavirus prevalence in 5 continents: meta-analysis of 1 million women with normal cytological findings, *J. Infect. Dis.* 202 (12) (2010) 1789–1799.
- [26] J.P. Figueroa, E. Ward, T.E. Luthi, S.H. Vermund, A.R. Brathwaite, R.D. Burk, Prevalence of human papillomavirus among STD clinic attendees in Jamaica: association of younger age and increased sexual activity, *Sex. Transm. Dis.* 22 (2) (1995) 114–118.
- [27] N. Gavillon, H. Vervaet, E. Derniaux, P. Terrosi, O. Graesslin, C. Quereux, How did I contract human papillomavirus (HPV)? *Gynecol. Obstet. Fertil.* 38 (3) (2010) 199–204.