pagepress

Sahelian Journal of Responsible One Health
Sahelian Journal of Responsible One Health 2026; 2:616
doi:10.4081/sjroh.616

Molecular profiling of human papillomavirus genotypes, cytomorphological abnormalities, and their predictors in Cameroon: a multi-regional analysis

Keriane Diane Kambou Kountchou,[1-3] Michel Carlos Tommo Tchouaket,[4] Bernadette Fokou Bomgning,[2,4,5] Larissa Gaelle Moko Fotso,[4] Semengue Ezechiel Ngoufack Jagni,[4] Simon Manga,[6] Alistair Dumps,[7] George Enow-Orock,[3] Joseph Fokam,[4,8,9] Jules-Roger Kuiate[2,10]

[1]Department of Experimental Medicine, University of Rome "Tor Vergata", Italy; [2]Faculty of Science and Technologies, Evangelical University Institute of Cameroon, Bandjoun, Cameroon; [3]Department of Anatomy and Cytopathology, Bafoussam Regional Hospital, Cameroon; [4]Chantal Biya International Reference Centre, Yaoundè, Cameroon; [5]Department of Immunology, Molecular Medicine, and Applied Biotechnology, University of Rome "Tor Vergata", Italy; [6]Department of Obstetrics and Gynecology, University of Alabama, Birmingham, USA; [7]Department of Gynaecology and Obstetrics, Geneva University Hospitals, Switzerland; [8]National Aids Control Committee, Ministry of Public Health, Cameroon; [9]Department of Biochemistry, Faculty of Science, University of Dschang, Cameroon; [10]Faculty of Medicine and Biomedical Sciences, University of Yaoundè 1, Cameroon

Abstract

Cervical cancer-related mortality remains high in Cameroon, yet data on high-risk human papillomavirus (HR-HPV) genotypes are sparse. This study characterized HR-HPV profiles and evaluated associations with cytomorphological abnormalities and clinical predictors. We enrolled 555 women (≥25 years) from four regions in Cameroon. Cervical samples underwent cytological evaluation and HR-HPV genotyping using polymerase chain reaction (PCR)-based assays (Abbott Laboratories, USA; Sacace Biotechnologies, Italy). Questionnaires were used to collect sociodemographic/clinical data, and associations with cytological abnormalities and clinical factors were assessed using multivariable logistic regression. HR-HPV prevalence was 26.5%, dominated by HPV16 (21.1%), HPV68 (19.4%), and HPV45 (14.3%). Multiple infections (38.8%) significantly increased risks for high-grade squamous intraepithelial lesions (HSIL; odds ratio [OR]=1.59, p=0.002) and squamous cell carcinoma (SCC; OR=2.81, p<0.001). HPV16 showed the strongest association with SCC (OR=23.74, p<0.001) while HPV45 (OR=2.91, p=0.039), HPV31 (OR=4.15, p=0.018), HPV33 (OR=5.47, p=0.039) and HPV52 (OR=6.60, p=0.023) showed significant associations with HSIL and SCC. Significant clinical predictors (p<0.001) included age, parity, tobacco, and HIV-positive status. Our study confirms the diverse HR-HPV genotype profile in Cameroon, with notable frequencies of non-16/18 genotypes and multiple infections. These findings underscore the need for broader-spectrum vaccines and risk-based screening in Cameroon.

Key words: human papillomavirus; high-risk HPV genotypes; cervical neoplasia; Cameroon.

Correspondence: Keriane Diane Kambou Kountchou, Faculty of Science and Technologies, Evangelical University Institute of Cameroon, Bandjoun, Cameroon. E-mail: kamkerianek@gmail.com

Introduction

Cervical cancer is a significant global health burden, with over 90% of related deaths occurring in low- and middle-income countries, particularly in Sub-Saharan Africa.[1] In Cameroon, it is the second leading cause of cancer-related mortality among women, reflecting critical gaps in prevention and early intervention.[2] The disease is mostly caused by persistent infection with high-risk human papillomavirus (HR-HPV), which drives carcinogenesis through viral oncoproteins E6 and E7.[3] While HPV genotypes 16 and 18 cause approximately 70% of cases globally, the remaining 30% are linked to at least 12 other HR-HPV genotypes (31, 33, 35, 39, 45, 51, 52, 56, 58, 59, 66, 68) whose oncogenic potential varies by geography.[4] HR-HPV infection induces a spectrum of cytomorphological changes, classified by the Bethesda System from low-grade squamous intraepithelial lesions (LSIL) to invasive carcinoma.[5] However, the link between specific genotypes and clinical severity is not uniform; certain types exhibit higher oncogenic potential and faster progression.[6] Understanding these genotype-specific signatures is essential for refining risk stratification. In high-incidence settings like Cameroon, the landscape is further complicated by a high prevalence of multiple concurrent infections.[7] Such co-infections may modulate disease progression through viral interference or synergistic oncogenic activity, yet their significance remains inadequately characterized in many African populations.[8] Despite the need for context-specific data, comprehensive molecular epidemiological studies in Cameroon are sparse. Existing reports are often limited by small sample sizes

or assays that fail to discriminate between individual non-16/18 genotypes.[9] Consequently, the circulating genotype profile, patterns of multiple infections, and their association with cytological outcomes remain poorly defined at a national level. This gap impedes the development of tailored screening protocols and the assessment of vaccine impact. To address these gaps, this study provides a detailed molecular and cytopathological characterization of HR-HPV in a multi-regional Cameroonian cohort. We aimed to: i) determine the distribution of 14 HR-HPV genotypes and cervical lesions across four diverse regions; ii) assess patterns of multiple HR-HPV infections; iii) evaluate the association between specific genotypes and cervical abnormalities; and iv) identify sociodemographic and clinical predictors significantly associated with cervical lesions.

Materials and Methods

Study design and population

A cross-sectional study was conducted from March 2024 to April 2025 across four regions of Cameroon representing diverse ecological and sociodemographic profiles: Centre (Yaoundé), West (Bafoussam), North (Garoua), and South (Sangmelima). These regions encompass both urban and semi-rural populations, providing a comprehensive view of the national epidemiological landscape. We enrolled 555 women aged ≥25 years attending routine cervical cancer screening or gynecological consultations. We included non-pregnant women aged ≥25 years willing to sign an informed consent form. Women with a history of invasive cervical cancer, cervical excision, total hysterectomy, pregnancy, or active menstruation at the time of sampling were excluded (Figure 1).

Sample size calculation

The minimum sample size was calculated with an absolute precision of ±4% at a 95% confidence level. Accounting for a 10% rate of invalid results or non-response, the required sample size was estimated at 540 participants. A total of 555 women were ultimately recruited, fully meeting the predetermined sample size requirements. The study was approved by the National Committee for Ethics in Human Health Research (No. 2020/06/1249/CE/CNERSH/SP). Administrative authorization was obtained from all participating hospitals and research centers. Written informed consent was obtained from each participant in either French or English prior to enrollment, and they were free to withdraw from the study at any stage. All data were anonymized using unique identification codes, and participants with abnormal findings were referred to a gynecologist for appropriate clinical management.

Sample collection and preservation

Cervical samples were collected by trained healthcare providers following standardized clinical procedures. Two parallel specimens were obtained from each participant: one for conventional cytology and one for liquid-based cytology. Conventional smears were collected using an Ayre spatula and cytobrush, spread on labeled glass slides, fixed with cytofixative spray, and immedi-

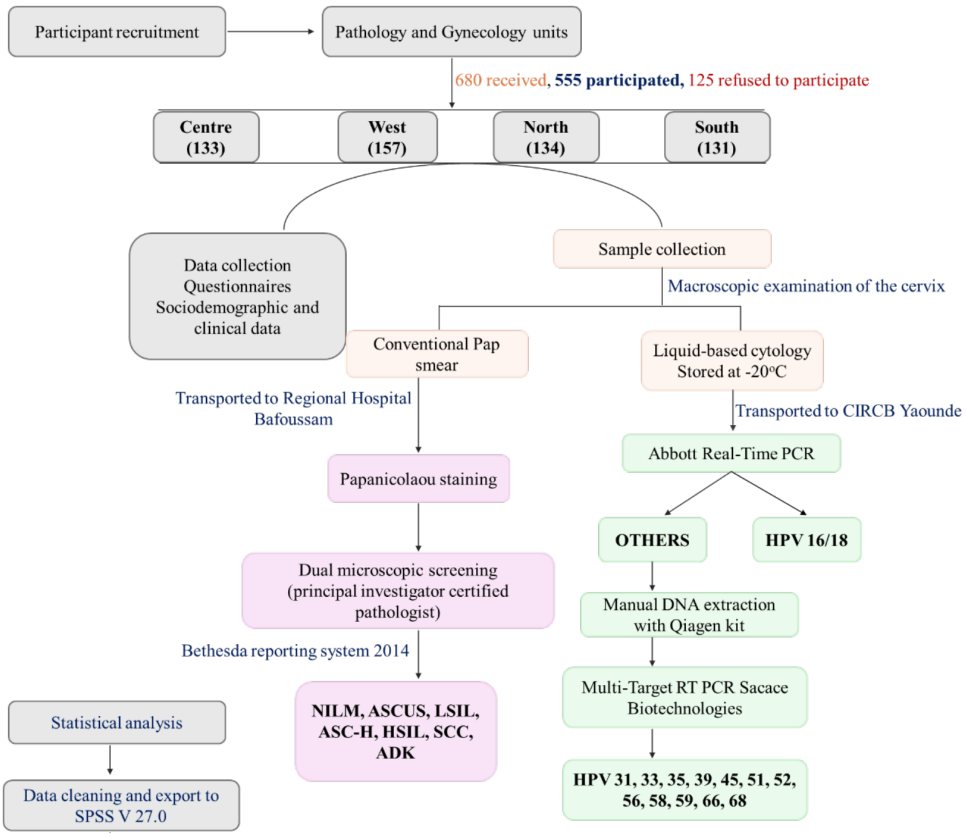

Figure 1. Flow diagram of the study methodology.

ately transported to the pathology laboratory of the Bafoussam Regional Hospital for staining and evaluation. Liquid-based samples were stored at −20°C and transported on ice to the Chantal Biya International Reference Centre (CIRCB) in Yaoundé for HR-HPV molecular analysis. Demographic and clinical information, including HIV status, parity, and tobacco use, was collected using structured questionnaires.

Cytomorphological analysis

Conventional smears were stained using the standardized Papanicolaou method and mounted with EUKITT® mounting medium. They were screened by a trained cytopathologist and reviewed by a certified pathologist blinded to the initial results. Discrepant cases were resolved through consensus review. Cytological diagnoses were reported according to the Bethesda System 2014,[5] with the following categories: negative for intraepithelial lesion or malignancy (NILM); atypical squamous cells of undetermined significance (ASC-US); atypical squamous cells cannot exclude HSIL (ASC-H); low-grade squamous intraepithelial lesion (LSIL); high-grade squamous intraepithelial lesion (HSIL); squamous cell carcinoma (SCC); and adenocarcinoma (ADK).

Molecular detection and genotyping of HR-HPV

Total DNA was extracted from cervical suspensions using either the Abbott m2000sp automated system (Abbott Park, Illinois, USA) or manual silica-column methods, according to manufacturer protocols. Samples were initially screened for 14 HR-HPV types (16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, 66, and 68) using the Abbott RealTime HR-HPV assay. This assay provides specific detection of HPV16 and HPV18 while reporting the remaining 12 types as a pooled "Other HR-HPV" result. Samples positive for the "Other HR-HPV" pool were further characterized using the Sacace HPV Genotypes 14 Real-TM Quant multiplex real-time polymerase chain reaction (PCR; Sacace Biotechnologies, Como, Italy). This facilitated individual identification of the 12 non-16/18 genotypes. Amplification was performed on the Applied Biosystems 7500 Fast platform. A cycle threshold (Ct) <33.0 was considered positive for high viral load, while 33.0-37.0 indicated a low load. All molecular assays included negative and positive controls in each run. The Abbott assays were performed under the manufacturer's quality standards, while the Sacace genotyping was conducted in an ISO 15189-accredited laboratory at CIRCB.

Statistical analysis

Data was analyzed using SPSS version 27.0. Descriptive statistics summarized participant characteristics and HPV prevalence. The chi-square test compared categorical variables across regions and cytological grades. Multivariable logistic regression identified independent clinical predictors of abnormal cytology, with results expressed as odds ratios (OR) and 95% confidence intervals (CI). A p-value <0.05 was considered statistically significant.

Results

Sociodemographic characteristics

The study included 555 women with a median age of 41 years (interquartile range [IQR]: 33-51). Participants were distributed across four regions: Centre (n=124), West (n=123), North (n=183), and South (n=125). Most participants had attained secondary (44.1%) or university-level (33.5%) education, and housewives constituted the largest occupational group (31.7%). The majority were married (56.8%) (Table 1).

Table 1. Sociodemographic and clinical characteristics of the study population (n=555).

Characteristics	Categories	n	%
Region	West	157	28.3
	North	134	24.1
	Centre	133	24.0
	South	131	23.6
Age (years)	25-35	148	26.7
	36-46	152	27.4
	47-57	167	30.1
	≥58	88	15.8
Mean ± SD	44.8±13.2		
Educational level	Uneducated/primary school	124	22.3
	Secondary	245	44.1
	University	186	33.5
Profession	Housewife	176	31.7
	Trader	93	16.8
	Professionals*	93	16.8
	Others#	193	34.8
Marital status	Married	315	56.8
	Single	127	22.9
	Widowed/divorced	81	14.6
	Cohabitation	32	5.8
HIV status	Negative	479	86.3
	Positive	76	13.7
Tobacco use	No	502	90.5
	Yes	53	9.5

*Professionals = teacher, healthcare personnel; #others = student, secretary, hairdresser, policewoman, tailor.

Prevalence and genotype distribution of HR-HPV

The overall prevalence of HR-HPV infection was 26.5% (147/555). Among HPV-positive women, HPV16 was the most frequently detected genotype (21.1%), followed by HPV68 (19.4%), HPV45 (14.3%), and HPV58 (14.3%). HPV18 was identified in only 8.8% of positive cases (Figure 2).

The overall prevalence of cytological abnormalities was 33.0% (183/555). The distribution of abnormalities was as follows: ASC-US 8.2% (15/182), LSIL 41.2% (75/182), HSIL 37.4% (68/182), and SCC 7.1% (13/182). Notably, high-grade lesions (HSIL/SCC) were most frequent in the North region (Figure 3).

Multiple HPV infections were detected in 38.8% (57/147) of HPV-positive women and were significantly associated with cervical lesion severity. Compared to single infections, women with multiple infections had higher odds of HSIL (OR=1.59, 1.19-2.14, p=0.002) and SCC (OR=2.81, 1.72-4.59, p<0.001) (Table 2).

Association between cervical precancerous/cancerous lesions and HPV genotypes

In genotype-specific unadjusted logistic regression analyses, HPV16 demonstrated the strongest association with high-grade lesions and cancer. Women infected with HPV16 had significantly increased odds of HSIL (OR=4.22, 1.73-10.32, p=0.002) and SCC (OR=23.74, 6.99-80.62, p<0.001) compared to women with NILM (Table 3). Several non-16/18 genotypes also showed significant associations with cytological severity: HPV45 with HSIL (OR=2.91, 1.05-8.04, p=0.039) and SCC (OR=5.47, 1.09-27.44, p=0.039), HPV31 with HSIL (OR=4.15, 1.28-13.48, p=0.018), and HPV52 with SCC (OR=6.60, 1.29-33.77, p=0.023) (Table 3).

Association between cervical precancerous/cancerous lesions and clinical/behavioral covariates

Adjusted multivariate logistic regression models revealed significant associations between some clinical/sociodemographic factors and cervical lesions. Age was a strong predictor: women aged 36-46 years (OR=1.74, 1.03-2.94, p=0.038) and 47-57 years (OR=3.00, 1.82-4.95, p<0.001) had significantly higher odds of cervical lesions compared to those aged 25-35 years. Tobacco use was associated with a 3.87-fold increased odds of cervical lesions (2.15-6.97, p<0.001), while HIV-positive status was associated with a 3.36-fold increase (2.05-5.53, p<0.001) (Table 4).

Discussion

This study provides a comprehensive characterization of the HR-HPV landscape in Cameroon, revealing a complex interplay between viral genotypes, cytomorphological outcomes, and regional sociodemographic factors. It revealed a high prevalence of high-risk HPV infection (26.5%, 147/555), which is consistent with studies across Sub-Saharan Africa, where pooled estimates

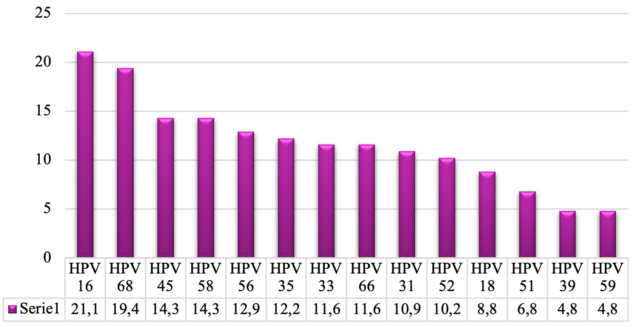

Figure 2. Distribution of specific HR-HPV genotypes among HPV-positive women (n=147). Percentages reflect the proportion of each genotype among all HPV-positive cases and exceed 100% due to multiple HPV infections (see the HPV co-infection matrix in *Appendix*).

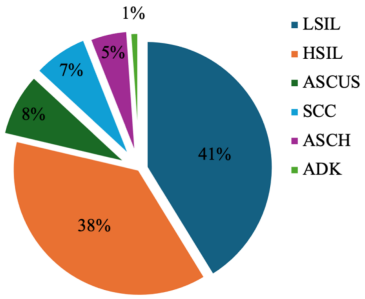

Figure 3. Overall distribution of the different types of cervical lesions in the study population. LSIL, low-grade squamous intraepithelial lesion; HSIL, high-grade squamous intraepithelial lesion; ASCUS, atypical squamous cells of undetermined significance; ASCH, atypical squamous cells not excluding high grade; SCC, squamous cell carcinoma; ADK, adenocarcinoma.

Table 2. Association between number of concurrent HR-HPV infections and lesion severity.

Cervical lesion	Infection counts (n)	OR (95% CI)	p
NILM	Negative: 293; single: 49; double: 21; >2: 10	1 (reference)	-
LSIL	Negative: 56; single: 39; double: 3; >2: 2	1.05 (0.74-1.49)	0.797
HSIL	Negative: 42; single: 14; double: 7; >2: 5	1.59 (1.19-2.14)	0.002
ASC-US	Negative: 11; single: 3; double: 0; >2: 1	1.16 (0.58-2.30)	0.675
ASC-H	Negative: 5; single: 1; double: 3; >2: 0	1.85 (0.96-3.56)	0.065
SCC	Negative: 2; single: 7; double: 2; >2: 2	2.81 (1.72-4.59)	<0.001

OR, odds ratio; CI, confidence interval; NILM, negative for intraepithelial lesion or malignancy; LSIL, low-grade squamous intraepithelial lesion; HSIL, high-grade squamous intraepithelial lesion; ASC-US, atypical squamous cells of undetermined significance; ASC-H, atypical squamous cells not excluding high grade; SCC, squamous cell carcinoma.

range between 20% and 30% in similar clinic-based populations.[10,17] It is, however, lower than 41.8% and 57.9% prevalence reported in recent Cameroonian studies by Ebong *et al.*,[18] Manga *et al.*,[11] and 33.2% reported in Benin by Piras *et al.*[19] This discrepancy may be explained by differences in the sensitivity of the genotyping assays used by Ebong *et al.* (Ampfire HPV DNA test), as well as variations in the study population and the small sample size of Manga *et al.*'s cohort (26 women who have sex with women).

Extended genotyping revealed a distinctive epidemiological profile (Figure 2). HPV16 was the most prevalent (21.1%), consistent with studies across Sub-Saharan Africa.[7,20] However, HPV68 emerged as the second most frequent genotype (19.4%), followed closely by HPV45 and HPV58 (14.3% each). HPV18 prevalence was comparatively low (8.8%). This hierarchy highlights significant circulation of non-16/18 high-risk types, aligning with other African studies.[18-20] Consequently, the protective efficacy of bivalent or quadrivalent vaccines may be reduced in our setting. Strategic introduction of the nonavalent HPV vaccine in Cameroon, which targets a broader spectrum of HR-HPV types, is strongly recommended.

The burden of cervical lesions is high (33.0%, 183/555), which exceeds the 19.05% reported by Manga *et al.*,[11] 12.2% by Wabo *et al.*,[12] 17.06% by Mengistie *et al.*,[13] and 8.8% by Kiros *et al*.[14] Several factors likely explain these disparities. Our hospital-based recruitment may have enriched for symptomatic or higher-risk women compared to community screening campaigns[12] or population-based systematic reviews.[13] Again, the specific focus on women who have sex with women[11] *vs.* our broader sexually active population explains the difference in prevalence. Among lesion-positive women, LSIL was detected in 41%, HSIL in 38%, SCC in 7.0%, and ADK in 1% (Figure 3). Our HSIL prevalence (38%) exceeds the 15.3% reported by Tagne *et al.* in Yaoundé,[15] while invasive cancer (7.0%) is markedly lower than the 29.1% found by Tufon *et al*.[16] This difference may reflect increased cervical cancer awareness and screening uptake between 2013 and 2025. This elevated lesion burden underscores the urgent need for scalable, effective triage strategies in Cameroon.

Table 3. Genotype-specific associations with cervical lesion severity (multivariable logistic regression, n=555).

HPV genotype	Positive cases (n)	Cytological outcome	OR (95% CI)	p
HPV16	31	HSIL	4.22 (1.73-10.32)	0.002
HPV16	31	SCC	23.74 (6.99-80.62)	<0.001
HPV68	17	HSIL	1.31 (0.43-4.02)	0.638
HPV68	17	SCC	1.75 (0.21-14.21)	0.603
HPV45	12	HSIL	2.91 (1.05-8.04)	0.039
HPV45	12	SCC	5.47 (1.09-27.44)	0.039
HPV33	12	SCC	5.47 (1.09-27.44)	0.039
HPV31	7	HSIL	4.15 (1.28-13.48)	0.018
HPV52	10	SCC	6.60 (1.29-33.77)	0.023

OR, odds ratio; CI, confidence interval; HSIL, high-grade squamous intraepithelial lesion; SCC, squamous cell carcinoma.

Table 4. Multivariable logistic regression analysis of clinical/sociodemographic covariates associated with cervical lesions.

Risk factors	Cervical lesions		OR (95% CI)	p
	Negative	Positive		
Age range (years)				
25-35	117	31	1 (reference)	
36-46	104	48	1.74 (1.03-2.94)	0.038*
47-57	93	74	3.00 (1.82-4.95)	<0.001**
58-68	45	23	1.92 (1.10-3.66)	0.044*
≥69	13	7	2.03 (0.74-5.53)	0.165
Number of pregnancies				
0	46	8	1 (reference)	
1-3	129	62	2.76 (1.23-6.21)	0.014*
4-6	113	81	4.12 (1.85-9.20)	0.001***
7-9	71	27	2.19 (0.91-5.23)	0.079
≥10	13	5	2.21 (0.62-7.92)	0.223
Tobacco consumption				
No	352	150	1 (reference)	
Yes	20	33	3.87 (2.15-6.97)	<0.001**
HIV status				
Negative	340	139	1 (reference)	
Positive	32	44	3.36 (2.05-5.53)	<0.001**
Alcohol consumption				
No	157	60	1 (reference)	
Yes	215	123	1.50 (1.03-2.17)	0.033*

OR, odds ratio; CI, confidence interval; statistical significance *p<0.05; **p<0.01; ***p<0.001.

The strong association between HR-HPV and cervical lesions aligned with the well-established causal link reported globally.[21,22] Importantly, our data extended beyond this binary association to reveal a clear risk gradient determined by specific genotypes and infection patterns. First, HPV16 was the strongest predictor, with a 4-fold increase in the odds of HSIL (OR=4.22; 1.73-10.32, p=0.002) and a 24-fold increase in the odds of SCC (OR=23.74; 6.99-80.62, p<0.001). Second, non-16/18 genotypes, including HPV45, HPV31, and HPV52, showed significant associations with HSIL and/or SCC (OR=2.9-6.6) (Table 3). Third, the number of concurrent infections was itself a powerful predictor: each additional HR-HPV type increased HSIL odds by 59% (OR=1.59; 1.19-2.14, p=0.002) and SCC odds by 181% (OR=2.81; 1.72-4.59, p<0.001) (Table 2). This aligns with studies reporting synergistic effects of multiple infections, possibly through immune modulation or genomic instability.[22,23] These findings underscore that infection multiplicity must be captured in any effective risk assessment. Beyond viral factors, key sociodemographic and clinical variables were independently associated with cervical lesions. Age was a significant predictor: compared to women aged 25-35 years, those aged 47-57 years had 3-fold increased odds (OR=3.00; 1.82-4.95, p=0.038), and those over 69 years had a 2-fold increased odds (OR=2.03; 0.74-5.53) (Table 4), aligning with African cohorts reporting peak lesion prevalence in women over 40 years.[24] University education was protective (OR=0.23; 0.06-0.75, p=0.015), likely reflecting greater health literacy, access to cervical cancer preventive services, and potentially lower exposure to other risk factors. High parity (>4 pregnancies) conferred over 4-fold increased odds (OR=4.12; 1.85-9.20, p=0.001) (Table 4), consistent with studies identifying parity as an independent predictor,[25,26] though Okyere *et al.*[20] found no such association. Sexual behavior variables were not significant, contrasting with findings from Rwanda and Ethiopia;[27,28] this may reflect reporting bias or differing risk factor hierarchy post-infection. The strongest non-viral predictors were biological (Table 4): HIV-positive status (OR=3.36; 2.05-5.53, p<0.001) and tobacco use (OR=3.87; 2.15-6.97, p<0.001), both well-documented drivers of HPV persistence and progression.[29] Alcohol showed a weaker association (OR=1.50; 1.03-2.17, p=0.033), while contraceptive use was not significant (p=0.741), consistent with some contemporary studies.[30]

Limitations of the study

This study has several limitations. First, hospital-based recruitment may introduce selection bias, potentially limiting the generalizability of prevalence estimates to the general population; thus, findings should be interpreted within the context of a consultation-seeking population. Second, the cross-sectional design precludes establishing causal relationships between HPV infection and lesion progression. Third, statistical power was limited for rare HPV genotypes and squamous cell carcinoma cases, resulting in wide confidence intervals for these subgroup analyses. Fourth, certain behavioral variables were based on self-report and may be subject to recall or social desirability bias. Despite these limitations, this study provides valuable epidemiological insights and underscores the need for larger, longitudinal, population-based studies to refine risk estimates and inform national cervical cancer prevention strategies.

Policy implications

The findings of this study have direct implications for cervical cancer prevention strategies in Cameroon. The high burden of cervical lesions and the predominance of non-16/18 HR-HPV genotypes underscore the urgent need to introduce the nonavalent HPV vaccine, which offers broader protection against circulating strains. Integration of cervical cancer screening with HIV care and tobacco cessation programs could further reduce risk in vulnerable populations. Collectively, these results support a "One Health" approach that combines biomedical, behavioral, and socio-environmental interventions to strengthen cervical cancer control in Cameroon.

Conclusions

In conclusion, this study provides a foundational molecular epidemiological profile of HR-HPV in Cameroon, highlighting the significant roles of genotype diversity and multiple infections in shaping cervical cancer risk. The strong correlation between multiple infections and severe cervical lesions underscores the oncogenic potential of co-infections and the need for continued monitoring. Independent predictors, including age, parity, tobacco use, and HIV status, illustrate the multifactorial nature of cervical carcinogenesis in this population.

These findings can inform national prevention strategies. The high prevalence of non-16/18 types strongly supports transitioning to the nonavalent HPV vaccine for broader protection. Integrating screening with HIV services and programs addressing behavioral risk factors may enhance effectiveness. Ultimately, this work reinforces the need for a comprehensive "One Health" approach combining vaccination, screening, and socio-environmental strategies to reduce cervical cancer morbidity and mortality in Cameroon.

References

1. Sung H, Ferlay J, Siegel RL, et al. Global cancer statistics 2020: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J Clin 2021;71:209-49.
2. Bruni L, Albero G, Rowley J, et al. Global and regional estimates of genital human papillomavirus prevalence among men: a systematic review and meta-analysis. Lancet Glob Health 2023;11:e1345-62.
3. Estêvão D, Costa NR, Gil da Costa RM, Medeiros R. Hallmarks of HPV carcinogenesis: The role of E6, E7 and E5 oncoproteins in cellular malignancy. Biochim Biophys Acta Gene Regul Mech 2019;1862:153-62.
4. Seyoum A, Assefa N, Gure T, et al. Prevalence and genotype distribution of high-risk human papillomavirus infection among sub-Saharan African women: A systematic review and meta-analysis. Front Public Health 2022;10:875833.
5. Nayar R, Wilbur DC. The Bethesda System for Reporting Cervical Cytology: A Historical Perspective. Acta Cytol 2017;61:359-72.
6. de Oliveira GG, de Oliveira JMSC, Eleutério RMN, et al. Atypical squamous cells: Cytopathological findings and correlation with HPV genotype and histopathology. Acta Cytol 2018;62:386-92.
7. Tagne Simo R, Djoko Nono AG, Fogang Dongmo HP, et al. Prevalence of precancerous cervical lesions and high-risk human papillomavirus types in Yaounde, Cameroon. J Infect Dev Ctries 2021;15:1339-45.
8. Na J, Li Y, Wang J, et al. The correlation between multiple HPV infections and the occurrence, development, and progno-

sis of cervical cancer. Front Microbiol 2023;14:1220522.
9. World Health Organization. WHO guideline for screening and treatment of cervical pre-cancer lesions for cervical cancer prevention. 2nd ed. Geneva: World Health Organization; 2021.
10. Tommo Tchouaket MC, Fokam J, Sosso SM, et al. High genotypic diversity of human papillomavirus among women in Cameroon: Implications for vaccine effectiveness. IJID Reg 2022;5:130-6.
11. Manga SM, Lee GM, Ekoue N, et al. Prevalence of human papillomavirus and cervical neoplasia among women who have sex with women in Cameroon: A descriptive study. Afr J Reprod Health 2025;29.
12. Wabo B, Nsagha DS, Nana TN, Assob CJN. Prevalence and risk factors associated with precancerous cervical lesions among women in two cities in Cameroon. Pan Afr Med J 2022;41:276.
13. Mengistie BA, Aragaw GM, Anteneh TA, et al. Prevalence and determinants of precancerous cervical lesions among women screened for cervical cancer in Africa: A systematic review and meta-analysis. Plos One 2025;20:e0338484.
14. Kiros M, Belay DM, Getu S, et al. Prevalence and determinants of pre-cancerous cervical lesion and human papillomavirus among HIV-infected and HIV-uninfected women in North-West Ethiopia: A comparative retrospective cross-sectional study. HIV AIDS (Auckl) 2021;13:719-25.
15. Tagne Simo R, Mbock CV, Nwabo Kamdje AH, Telefo PB. Cervical precancerous lesions and associated factors among women screened in two hospitals in the city of Douala, Cameroon. Cureus 2023;15:e41993.
16. Tufon EN, Yuwun NB, Simeone E, Noel NN. Prevalence, associated risk factors and methods of diagnosing cervical cancer in two hospitals in Yaounde, Cameroon. Int J Adv Res Pharm Bio Sci 2013;2.
17. Tebeu PM, Ako A, Nguefack TC, et al. Épidémiologie de l'infection à HPV en région semi-urbaine du Cameroun: L'expérience du district de santé de Baham (Ouest-Cameroun). Health Sci Dis 2018;19.
18. Ebong CE, Nzang JL, Nyada SR, et al. Prevalence of high-risk human papillomavirus serotypes and confrontation with available vaccine antigens: The case of Cameroon. Health Sci Dis 2025;26:15.
19. Piras F, Piga M, De Montis A, et al. Prevalence of human papillomavirus infection in women in Benin, West Africa. Virol J 2011;8:514.
20. Okoye JO, Ofodile CA, Adeleke OK, Obioma O. Prevalence of high-risk HPV genotypes in sub-Saharan Africa according to HIV status: A 20-year systematic review. Epidemiol Health 2021;43:e2021039.
21. Ma X, Yang M. The correlation between high-risk HPV infection and precancerous lesions and cervical cancer. Am J Transl Res 2021;13:10830-6.
22. Luo Q, Zeng X, Luo H, et al. Epidemiologic characteristics of high-risk HPV and the correlation between multiple infections and cervical lesions. BMC Infect Dis 2023;23:667.
23. Liu Y, Zheng J, Zhang L, Wang Q. Clinical study on the pathogenic risks of different genotypes of high-risk HPV infection and multiple infections. Diagn Pathol 2025;20:81.
24. Eilu E, Aliero AA, Odoki M, et al. Prevalence of cervical intraepithelial neoplasia and its associated factors among women attending healthcare services in Eastern Uganda. J Cancer Res Exp Oncol 2020;12:1-12.
25. Tekalegn Y, Sahiledengle B, Woldeyohannes D, et al. High parity is associated with increased risk of cervical cancer: Systematic review and meta-analysis of case–control studies. Womens Health (Lond) 2022;18:17455065221075904.
26. Stensen S, Kjaer SK, Jensen SM, et al. Factors associated with type-specific persistence of high-risk human papillomavirus infection: A population-based study. Int J Cancer 2016;138:361-8.
27. Makuza JD, Nsanzimana S, Muhimpundu MA, et al. Prevalence and risk factors for cervical cancer and pre-cancerous lesions in Rwanda. Pan Afr Med J 2015;22:26.
28. Teklehaimanot DA, Mekuria AD, Dadi AF. Precancerous lesion determinants in women attending cervical cancer screening at public health facilities in North Shoa Zone, Amhara, Ethiopia: An unmatched case-control study. BMC Womens Health 2024;24:271.
29. Stelzle D, Tanaka LF, Lee KK, et al. Estimates of the global burden of cervical cancer associated with HIV. Lancet Glob Health 2021;9:e161-9.
30. Kashyap VK, Kenchappa DB, Singh AK, et al. Alcohol consumption and cervical carcinogenesis: Time to draw conclusions. Cells 2025;14:1639.

Online Supplementary Material:
Appendix. HPV co-infection matrix.

Received: 16 March 2026; Accepted: 17 March 2026.

Conflict of interest: the authors have no conflict of interest to declare.

Ethics approval and consent to participate: the study was approved by the National Committee for Ethics in Human Health Research (No. 2020/06/1249/CE/CNERSH/SP). Administrative authorization was obtained from all participating hospitals and research centers. Written informed consent was obtained from each participant in either French or English prior to enrollment.

Availability of data and materials: the datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.