

Review Article

<https://doi.org/10.20546/ijcmas.2026.1507.003>

High-Risk Oncogenic Human Papillomavirus Infection among Women in the Republic of the Congo: A Systematic Review

Dimitry Moudiongou Mboungou Malanda^{1,2,3}, Paola Candyse Tsimba Lemba^{1,3},
Bredin Roch Bissala Nkounkou^{1,3}, Archange Michel Emmanuel Mboungou Malonga¹
and Anicet Luc Magloire Boumba^{1,3}

¹Faculty of Health Sciences, Marien Ngouabi University, Brazzaville, Republic of the Congo;

²Department of Anatomy and Cytopathology, Brazzaville University Hospital, Brazzaville, Republic of the Congo;

³Research Unit in Virology and Molecular Biology at the FSSA, Brazzaville, Republic of the Congo

**Corresponding author*

ABSTRACT

Keywords

Human papillomavirus; hrHPV; genotypes; cervical cancer; Republic of Congo.

Article Info

Received:

05 May 2026

Accepted:

26 June 2026

Available Online:

10 July 2026

Infection with high-risk human papillomaviruses (hrHPV) is a major risk factor for cervical cancer in sub-Saharan Africa. In the Congo, epidemiological data remain limited and fragmented. This study aimed to synthesize available data on the prevalence of hrHPV infections, the distribution of circulating genotypes, and their implications for vaccination, screening, and prevention of cervical cancer in the Republic of the Congo. A systematic literature review was conducted using international databases (PubMed, ScienceDirect, AJOL, and SpringerLink). Studies on HPV prevalence, genotyping, and prevention were included according to predefined criteria. A total of 25 articles were selected based on methodological quality and contextual relevance. The analyzed data reveal high circulation of hrHPV among Congolese women, with an overall high prevalence ranging from 23% to 52%, depending on the population studied. The most frequently identified genotypes are HPV 16, HPV 35, and HPV 52, often associated with multiple infections. HPV 16 remains the predominant genotype in precancerous lesions and invasive cervical cancer. The observed genotypic diversity also includes several types not systematically covered by available vaccines. The high prevalence and diversity of human papillomaviruses (hrHPV) underscore the need to strengthen vaccination, molecular screening, and epidemiological surveillance to optimize cervical cancer prevention in the Republic of Congo.

Introduction

Human papillomavirus (HPV) infection is currently the most widespread sexually transmitted infection worldwide and represents a major public health concern

due to its direct involvement in cervical carcinogenesis (1, 2). Among the more than 200 identified genotypes, approximately fifteen are classified as high-risk oncogenic (hrHPV) due to their ability to induce precancerous and invasive lesions, primarily through the

expression of viral oncoproteins E6 and E7, which disrupt cell cycle regulation mechanisms and cause genomic instability (3). Globally, HPV genotypes 16 and 18 are responsible for approximately 70% of cervical cancers (4). The distribution of other oncogenic types (HPV 31, 33, 35, 45, 52, and 58) varies significantly across geographic regions (4). This genotypic heterogeneity has major implications for the effectiveness of primary prevention strategies through vaccination and secondary prevention strategies through molecular screening. The performance of these interventions depends directly on the match between the targeted vaccine genotypes and those circulating in local populations (3, 4). In sub-Saharan Africa, the prevalence rates of human HPV infections are among the highest in the world, often exceeding 30% in sexually active women. This situation is accompanied by high genotypic diversity and significant circulation of types not included in some first-generation vaccines (5). In this context, the Republic of Congo is an area of particular epidemiological interest, where cervical cancer represents a major public health problem. Data on the distribution of HPV genotypes remain limited and fragmented (6). Studies available in the Republic of Congo nevertheless suggest high circulation of hrHPV in the female population (6). A population-based study reported a high overall prevalence of HPV, with a significant proportion of high-risk types. The most frequently identified genotypes are HPV 16, HPV 35, and HPV 52, with a notable frequency of multiple infections (5, 6). Other studies have confirmed a strong involvement of oncogenic genotypes in cervical lesions, with a very high proportion of hrHPV in precancerous lesions and a persistent dominance of HPV 16 in invasive forms. However, these data are still derived from isolated, often hospital-based studies, which do not allow for the establishment of a representative national epidemiological profile (5, 6, 7). Furthermore, the genetic diversity of human HPV (hrHPV) in the Republic of Congo appears significant, with the co-circulation of several genotypes not systematically covered by current vaccines, notably HPV 35, HPV 52, and HPV 58 (5, 6, 7). This diversity could limit the effectiveness of standard vaccination strategies and underscores the need for a better understanding of the local genotypic structure to optimize prevention programs (5, 6, 7). In this context, it is suggested that the Congolese female population has a high frequency of HPV infections with high oncogenic risk, associated with a particular genotypic distribution that may differ from that observed in other regions of the world and influence the effectiveness of vaccination and

screening strategies (5, 6, 7). The focus of this work is to characterize the prevalence and distribution of HPV genotypes with high oncogenic risk in women in the Republic of Congo. The objective of this study is to conduct a systematic review of available data on the prevalence and distribution of high-risk oncogenic HPV genotypes in women in the Republic of Congo, in order to assess their implications for molecular screening, vaccination and cervical cancer prevention strategies.

Methodology

Type, sources and search engines

A structured literature review was conducted in accordance with PRISMA guidelines, using PubMed, ScienceDirect, Google Scholar, AJOL, and SpringerLink databases. Additional grey literature was identified through ResearchGate and institutional reports. This search strategy aimed to maximize the comprehensiveness of the available literature, particularly in African contexts where some publications are less frequently indexed in international databases.

The keywords used, combined with the Boolean operators “AND” and “OR,” were:

- “Human Papillomavirus” OR “HPV infection”;
- “High-risk HPV” OR “hrHPV”;
- “Genotype distribution” OR “prevalence”;
- “Cervical cancer” OR “oncogenic HPV”;
- “Republic of Congo” OR “Central Africa”.

The search strategy was tailored to each database to optimize sensitivity and specificity of the results.

Inclusion criteria

The following were included:

- Studies published up to May 2026;
- Studies conducted in the Republic of the Congo or including country-specific data;
- Studies addressing:
 - HPV prevalence;
 - Typing of high-risk oncogenic genotypes;
 - Cervical cancer screening and prevention programs;
- Original articles, systematic reviews, and institutional reports.

These criteria ensured contextual relevance and scientific quality of the data included in the analysis.

Exclusion criteria

The following were excluded:

- Studies outside the targeted geographic context;
- Insufficient or unusable data;
- Articles not available in full text;
- Studies focusing solely on other types of viruses or infections unrelated to HPV.

This rigorous selection process aimed to limit biases related to data quality and ensure consistency of the synthesis.

Target population

This systematic review examined published studies on HPV infections among women living in the Republic of the Congo, particularly those attending urban and semi-urban health centers. The study also drew on secondary data from the scientific literature. Special attention was paid to sexually active women and those eligible for HPV vaccination, screening, and follow-up programs.

Selection and analysis steps

A total of 45 records were identified during the literature review. After title and abstract screening, 20 records were excluded. The remaining 25 articles and institutional reports underwent full-text assessment, and all met the predefined eligibility criteria for inclusion in the review and were retained for the final synthesis.

Figure 1 illustrates the flowchart describing the various stages of identification, screening, eligibility, and inclusion of studies in the systematic review.

This systematic review was conducted in accordance with the recommendations of the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) statement. However, the review protocol was not prospectively registered in a dedicated database such as PROSPERO or the Open Science Framework, which constitutes a methodological limitation. No post-hoc changes to the inclusion or exclusion criteria were made during the study selection process.

The risk of bias assessment was not based on a standardized quantitative tool. Nevertheless, a qualitative assessment was performed during the selection and analysis of studies. The predefined criteria included the type of study design, case definition, the use of validated diagnostic methods for the detection and genotyping of human papillomavirus (including molecular biology techniques such as PCR), sample size, and the clarity and transparency of the methods and results reported.

Studies with insufficiently described methodologies, incomplete data, or non-standardized diagnostic procedures were excluded. This approach allowed for the inclusion of studies demonstrating an acceptable level of scientific rigor and contextual relevance. However, the absence of a formal critical appraisal tool, such as ROBIS or the Joanna Briggs Institute (JBI) assessment grids, is a limitation that could introduce some variability in the interpretation of the reported results and estimates.

Epidemiology of high-risk oncogenic human papillomaviruses

Overall prevalence and infection burden

The prevalence of high-risk oncogenic human papillomavirus (hrHPV) infections in the Republic of Congo is generally high, with variability ranging from 23% to 52% depending on the populations studied in different surveys (general population, hospital settings, and women with precancerous lesions). However, some local studies report higher prevalences, notably a study conducted in Brazzaville and the Plateaux department, where the overall prevalence of HPV DNA reached 64.4%, of which 80.9% were high-risk oncogenic infections (8), reflecting possible geographical and methodological heterogeneity. The most frequently identified genotypes were HPV 16 (15.8%), HPV 35 (15.3%), and HPV 52 (15.3%), with a significant proportion of multiple infections reaching 60.6% (8). Human HPV infection was associated with several risk factors, including young age and HIV-positive status, promoting the persistence and replication of viral infections (9). Furthermore, the genotypes included in the nonavalent vaccine (Gardasil 9®) represented only about 42.6% of infections detected in this population, highlighting the diversity of circulating genotypes and the need to adapt vaccination and screening strategies to the national epidemiological context (8). Studies

conducted in other regions of the country, particularly in the Niari and Bouenza departments and in Pointe-Noire, also confirm this high circulation of human HPV, with prevalence rates ranging from 37.5% to 44.1% depending on the geographic area and population studied (10). This situation reflects a significant burden of hrHPV infections in the Republic of Congo and reinforces the need for expanded molecular screening and continuous monitoring of the distribution of circulating genotypes (8).

HPV and cervical lesions

In precancerous lesions and invasive cervical cancer, the proportion of hrHPV infections is even higher, exceeding 85%. HPV 16 appears to be the dominant genotype, followed by HPV 18, HPV 33, HPV 31, and HPV 35 (6, 7, 11). At the Brazzaville University Hospital Center, a high prevalence of hrHPV was observed in precancerous lesions, with a marked dominance of HPV 16, followed by HPV 18 and other rare types (6, 7, 11). In cervical cancer, HPV 16 is found in more than 80% of cases, confirming its central role in cervical carcinogenesis in Congo (6, 7, 8, 11).

Genetic diversity and genotype distribution

The diversity of circulating hrHPV in the Republic of the Congo is notable. In addition to HPV 16 and HPV 18, genotypes such as HPV 35, HPV 52, HPV 58, HPV 45, HPV 33, and HPV 31 are frequently detected, particularly in multiple infections (7, 8, 12). Some studies even show a significant contribution from types not included in current vaccines, notably HPV 35, HPV 56, and HPV 68. This genotypic distribution differs partially from the profiles observed in Europe and America, where HPV 16 and HPV 18 are largely dominant. These observations suggest a regional African specificity, with a broader spectrum of oncogenic genotypes involved in cervical infections (5, 11, 12).

Comparative data and regional trends

At the regional and global levels, HPV is the most widespread sexually transmitted infection, with an estimated prevalence of 11-12% among women aged 15-49 (5, 12). In sub-Saharan Africa, the prevalence of human HPV (hrHPV) can exceed 30%, reflecting a particularly high burden. The most common oncogenic types worldwide include HPV 16, 18, 31, 33, 45, 52, and

58 (5, 6, 7, 12). However, in Central Africa, genotypes less common elsewhere, such as HPV 35, show a significant presence, highlighting the importance of local studies to adapt prevention policies (5, 6, 7, 12).

Genetic diversity and strain variability

The genetic diversity of HPV results from point mutations and variations in the L1 and E6/E7 regions, leading to the emergence of lineages and sub-lineages. HPV 16, for example, has several lineages (A, B, C, D), some of which are associated with a higher oncogenic risk (5, 13, 14). Regional studies show that African variants of HPV 16, HPV 35, and HPV 52 exhibit greater diversity than those observed elsewhere. This variability could influence viral persistence, transmissibility, and potentially the immune response (13, 14, 15).

Molecular evolution and strain dynamics

HPV evolves slowly, primarily through point mutations and not through massive recombination. Immune pressure, vaccination, and host-virus interactions influence their circulation (14, 15, 16). Phylogenetic studies in Central Africa suggest a geographical structuring of variants, with African lineages distinct from those observed elsewhere. Certain mutations in the L1 and LCR regions can modulate the expression of E6/E7 oncogenes and influence immune neutralization (15, 16, 17).

Implications for public health

The high prevalence of high-risk oncogenic HPV (hrHPV), genotypic diversity, and the high frequency of multiple infections in the Republic of the Congo constitute major public health challenges (2, 5, 8, 9, 10). Currently available vaccines, while effective against HPV genotypes 16 and 18, cover only a portion of the circulating types in the population (4, 11, 12, 15).

These data support the need for the adoption of broad-spectrum vaccines, particularly the nonavalent vaccine, the strengthening of multi-genotype molecular screening, and the implementation of continuous genomic surveillance of circulating strains (5, 8, 16, 17). Therefore, an adaptation of national prevention strategies is essential to improve the effectiveness of the fight against cervical cancer in the Congolese context (2, 11, 14).

Routes of transmission

High-risk human papillomaviruses (hrHPV) are primarily transmitted through direct contact between infected mucous membranes or skin and a susceptible individual (2, 5, 8). Sexual transmission is the predominant route of infection, particularly during vaginal, anal, or oral-genital contact with an infected partner (2, 11, 12). The virus is mainly transmitted through skin-to-skin contact in the genital and anogenital regions, even without complete penetration (2, 5, 8, 18). In women, this route is the primary mode of hrHPV transmission (8, 10, 18). Vertical or perinatal transmission can also occur when the newborn is exposed to an infected maternal genital tract during vaginal delivery (2, 12, 18). Although these neonatal infections are generally transient and cleared by the immune system, they constitute a recognized route of virus transmission (2, 12, 18). Less frequently, indirect transmission can occur through contaminated objects or surfaces (fomites), such as shared underwear, towels, or intimate accessories, when environmental conditions allow for the temporary survival of the virus. Iatrogenic transmission related to the use of insufficiently sterilized medical instruments also remains possible, although exceptional in facilities adhering to hygiene standards. The main routes of HPV transmission thus include sexual transmission (heterosexual, anogenital, and orogenital), mother-to-child transmission, and, in rare circumstances, certain forms of non-sexual transmission (2, 5, 12, 18). These mechanisms underscore the importance of early HPV vaccination, sexual health education, regular screening, and strengthening perinatal prevention measures to reduce viral circulation and the incidence of precancerous lesions and cancers associated with hrHPV (2, 4, 12, 15). Fig. 2.

This illustration presents the main routes of transmission of human papillomavirus (HPV) and the viral genotypes associated with benign and cancerous lesions. In Part A, the image highlights horizontal transmission, which is the most frequent route of infection. HPV can be transmitted during heterosexual intercourse through genital-genital, anogenital, or orogenital contact.

The illustration also shows the possibility of autoinoculation, that is, the transfer of the virus from one area of the body to another via contaminated fingers, clothing, towels, utensils, or medical instruments. Part B distinguishes HPV genotypes according to their pathogenic potential. High-risk oncogenic HPV types, including types 16, 18, 31, 33, 42, 51, and 59, are

associated with the development of precancerous and cancerous lesions. Conversely, low-risk HPV types, such as types 6, 11, 2, 4, and 57, are primarily responsible for benign lesions, including warts and genital warts. Part C illustrates other, less common routes of transmission. Perinatal transmission can occur from mother to child during childbirth or, more rarely, during breastfeeding. The image also shows oral-oral transmission, which can result from close oral contact facilitating the spread of the virus within the oral cavity. Overall, this figure highlights that HPV transmission relies primarily on direct contact between infected mucous membranes or skin, while certain indirect or vertical routes can also contribute to the spread of the virus. It also highlights the importance of high-risk genotypes, particularly HPV 16 and 18, in the occurrence of cancers linked to HPV infection.

Determinants of high-risk HPV infections in women

In the female population, infection with high-risk oncogenic human papillomaviruses (hrHPV) results from a complex interaction between demographic, behavioral, biological, and geographic factors (5, 8, 12). Demographically, the prevalence of hrHPV infections is particularly high in young women under 30 years of age, a period generally corresponding to the onset of sexual activity and increased exposure related to multiple sexual partners (5, 8, 12).

A second peak in prevalence is sometimes observed in older women, particularly after age 60, probably due to the persistence or reactivation of latent infections associated with the progressive weakening of immunity. Women living with HIV have a higher prevalence of hrHPV infections, a reduced capacity for viral clearance, and an increased risk of developing precancerous or cancerous cervical lesions (5, 9, 11). In the Congolese context, the coexistence of HIV and HPV epidemics significantly contributes to the burden of cervical cancer (2, 9).

Sexual behaviors also play a major role in the transmission of human HPV (hrHPV). Early onset of sexual activity, multiple sexual partners, and repeated exposure to infected partners considerably increase the risk of acquiring and transmitting oncogenic genotypes (8, 10, 12). Reproductive factors, particularly multiparity, have also been associated with an increased risk of viral persistence and progression to cervical lesions, possibly

due to hormonal changes and anatomical alterations of the cervix occurring during repeated pregnancies (8, 11). Furthermore, low levels of education and disadvantaged socioeconomic status often limit access to prevention, screening, and vaccination services, thus contributing to late diagnosis and disease progression (2, 8). Other individual factors, such as smoking and prolonged use of oral contraceptives, are recognized as cofactors that can promote the malignant transformation of cells infected with hrHPV.

Geographically, sub-Saharan Africa remains one of the regions of the world most affected by hrHPV infections and associated cancers (2, 5, 11, 12). This situation is characterized by a high prevalence of infection, significant genetic diversity of viral genotypes, and a high burden of morbidity related to cervical cancer (1, 2, 5, 11, 12). HPV16 and HPV18 genotypes remain the most frequently associated with precancerous and cancerous lesions, but other high-risk genotypes, such as HPV35, HPV52, and HPV68, also circulate at significant frequencies (1, 5, 7, 11, 12).

The presence of certain genotypes less well covered by available vaccines underscores the need for ongoing epidemiological surveillance and adaptation of vaccination strategies to local realities (1, 15, 17).

Disparities between urban and rural areas also influence the dynamics of infection (8). While urban environments generally offer better access to health services, they may be associated with higher transmission rates due to population density and more extensive sexual networks. Conversely, rural populations often face difficulties accessing screening, vaccination, and specialized care, leading to later diagnosis of cervical lesions (2, 8). Finally, several cultural and structural factors, such as early marriage, gender inequalities, sociocultural norms limiting access to sexual health information, and taboos surrounding sexuality, indirectly contribute to increasing women's vulnerability to hrHPV infections (2).

Overall, the high prevalence of hrHPV infections and the significant incidence of cervical cancer are explained by the interaction of multiple individual, social, and geographic factors (2, 5, 8, 12). This reality underscores the need to strengthen HPV vaccination programs, improve screening coverage, promote sexual health education, and develop prevention strategies tailored to the country's epidemiological and sociocultural context (2, 5, 8, 11). The main risk factors for high-risk HPV

infections in women, according to demographic and geographic dimensions, are presented in the table below.

Diagnostic methods

In the female population, the diagnosis of high-risk oncogenic HPV infections is part of a profound and structuring evolution in public health strategies, marking the gradual shift from screening primarily based on the observation of lesions to a high-precision molecular approach geared towards the early detection of infection and its transforming potential. In so-called traditional approaches, screening relied mainly on indirect methods aimed at identifying already established cellular or tissue abnormalities (2, 19, 20). The Pap smear, or Pap test, was the central tool, allowing for cytological analysis of cervical cells to detect precancerous changes. In addition to this method, visual inspection with acetic acid (VIA), sometimes combined with iodine (VILI), was widely adopted in resource-limited settings due to its operational simplicity and low cost. Colposcopy combined with biopsy was also used as the diagnostic reference to confirm suspicious lesions (2, 19, 20). However, these approaches, while historically crucial, remained limited by late lesion detection, significant inter-observer variability, a strong reliance on clinical expertise, and insufficient population coverage, particularly in rural areas of Central Africa (2, 19, 20).

Currently, the diagnostic paradigm has undergone profound changes driven by advances in molecular biology and international recommendations, notably those of the WHO, which now position HPV testing as the first-line method. Viral DNA detection tests are now the gold standard due to their ability to directly identify oncogenic genotypes with significantly higher sensitivity than conventional cytology. These tests rely on technologies such as PCR, Hybrid Capture 2, and automated platforms like Cobas and Onclarity, enabling the detection of persistent infections even before the appearance of precancerous lesions (5, 19, 21). These tools can be used alone or integrated into co-testing strategies combining HPV testing and cytology, thus enhancing diagnostic performance and individual risk stratification (8, 10, 17). In this context, vaginal self-sampling stands out as a major innovation, as it gives women greater diagnostic autonomy while reducing the sociocultural, geographical, and organizational barriers that still hinder access to screening in many African contexts, all while maintaining reliability comparable to samples collected in clinical settings (2, 22).

Table.1 Risk factors for high-risk HPV infections in women according to demographic and geographic dimensions

Category	Risk factors	Main impact
Demographic	Young age (<30 years)	High exposure due to early sexual activity and high incidence of transient infections
	Older age (>60 years)	Persistence or reactivation of latent HPV infection
Behavioral and reproductive	Early onset of sexual activity	Prolonged exposure to HPV and increased risk of infection
	Multiple sexual partners	Increased risk of acquiring high-risk HPV infections
	High parity	Promotes viral persistence and progression of cervical lesions
	Smoking	Cofactor promoting viral persistence and cervical carcinogenesis
	Long-term use of oral contraceptives	May increase the risk of progression to cervical lesions in infected women
Biological	HIV infection	Increased prevalence, persistence of high-risk HPV, and higher risk of progression to cervical cancer
Socio-economic	Low socio-economic status	Limited access to screening, vaccination, and healthcare services
Geographical and contextual	Sub-Saharan Africa	High prevalence of high-risk HPV
	Central Africa (Congo)	High genetic diversity of circulating genotypes and significant disease burden
	Urban settings	Potentially higher transmission due to population density and risk behaviors
	Rural settings	Limited access to screening, early diagnosis, and healthcare services
	Low healthcare coverage	Limited access to vaccination, screening, and treatment of precancerous lesions
	Cultural factors	Early marriage, gender inequality, and limited access to health information increase infection risk and persistence

Table.2 Evolution of diagnostic methods for HPV infections with high oncogenic risk

Period	Methods	Characteristics
Initial approach	Cervical smear test, VIA/VILI, colposcopy	Indirect approach, late detection, limited sensitivity
Current approach	HPV DNA tests, PCR, co-testing, self-sampling	Highly sensitive molecular methods, recommended by WHO
Future perspectives	Artificial intelligence, biomarkers, rapid E6/E7 tests, liquid biopsy	Early, personalized, and more accessible diagnosis

Figure.1 PRISMA flow diagram for the selection of studies

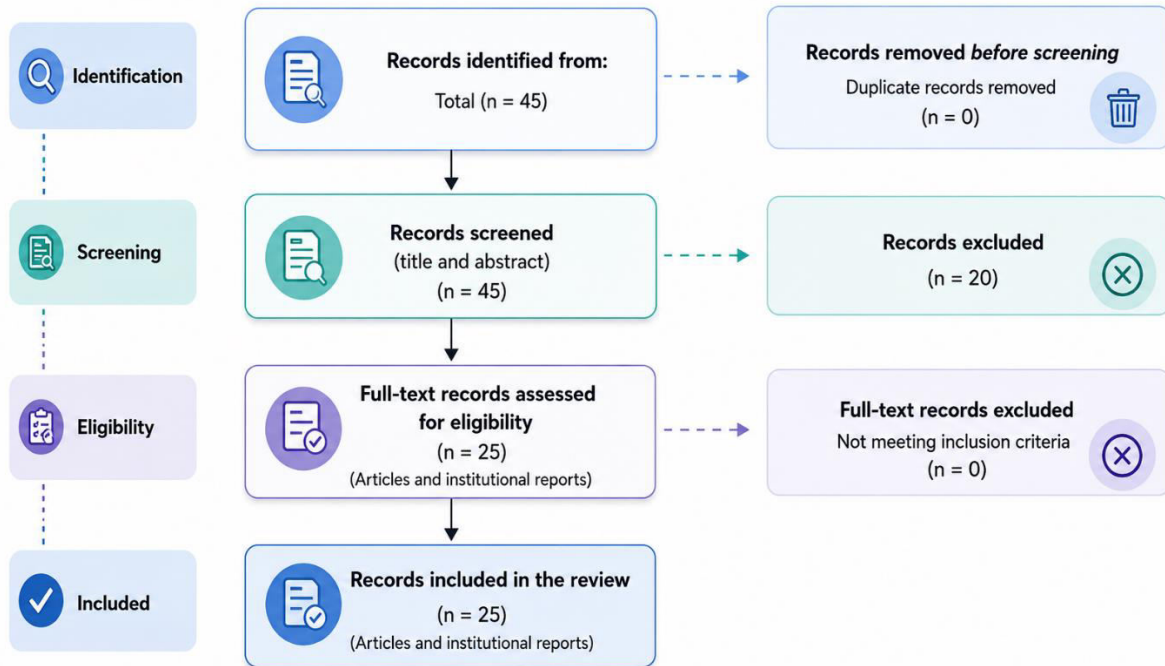
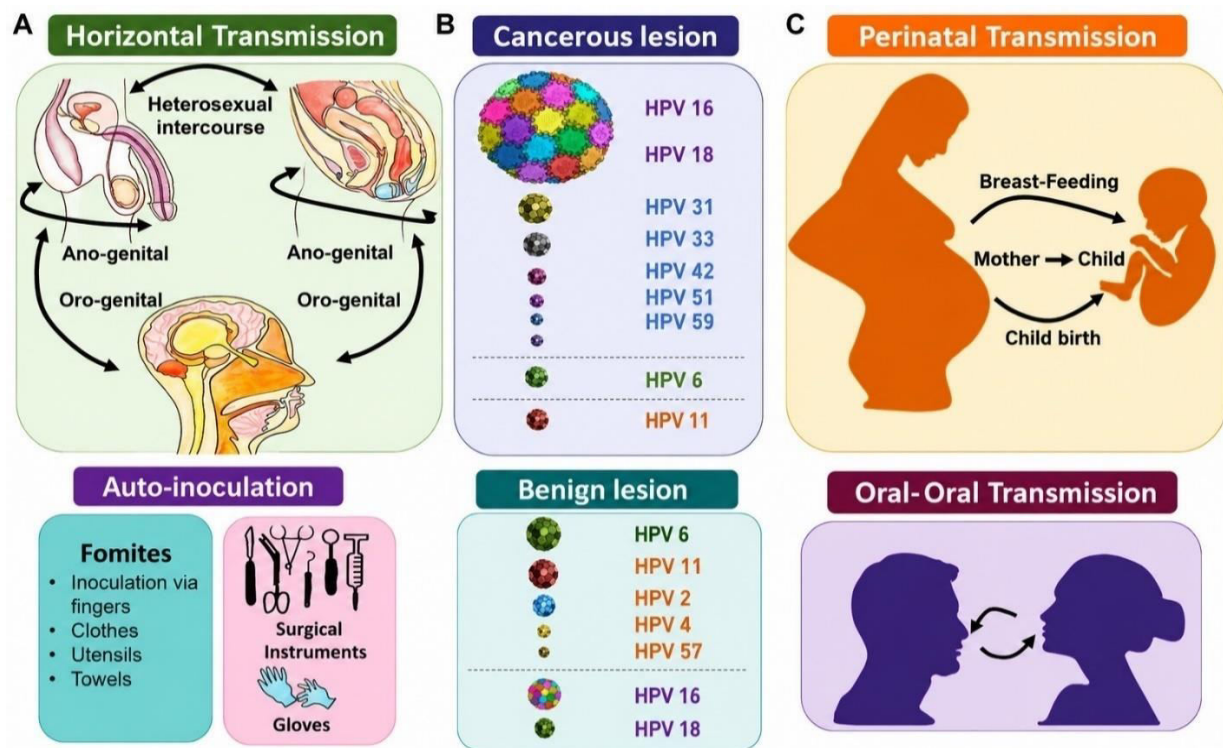


Figure.2 Main routes of transmission of human papillomavirus (HPV) and genotypes associated with benign and cancerous lesions (18)



Nevertheless, in several low-resource areas, visual methods such as VIA/VILI remain operational, despite their limitations in terms of specificity and reproducibility (2, 22).

Future prospects for HPV diagnosis lie in a logic of biological anticipation and precision medicine, aiming to identify not only the presence of the virus, but also its oncogenic activity and its potential for evolution. Innovations include the detection of viral oncogenes E6 and E7, allowing differentiation between transient infections and those with a high risk of malignant transformation (3, 17), as well as the use of cellular biomarkers such as p16INK4a and Ki-67, which refine the characterization of lesions with malignant potential. These are complemented by rapid point-of-care tests, designed for use in rural areas without extensive infrastructure, and artificial intelligence, increasingly used for the automated analysis of cytological and colposcopic images, reducing diagnostic subjectivity and improving reproducibility (21, 23). Finally, emerging innovations such as liquid biopsy and the detection of circulating viral DNA are paving the way for non-invasive, earlier approaches that can potentially be integrated into population-based screening strategies. Thus, this transition from a lesion-centered model to a molecular and predictive approach represents a major advance for public health, the impact of which will nevertheless depend on the ability of healthcare systems to guarantee the accessibility, sustainability, and effective integration of these technologies into organized screening programs (2, 17, 21, 23). The table below presents the evolution of diagnostic methods for high-risk oncogenic HPV infections, highlighting the shift from traditional approaches to current molecular techniques, as well as the innovations expected in the future.

Management of high-risk oncogenic HPV infections: evolution, integrated strategy, and perspectives

Historical approach

The management of HPV infections was primarily based on the treatment of precancerous lesions and established invasive cancers. The main interventions included cryotherapy, conization (partial excision of the cervix), and major surgical procedures such as hysterectomy, often followed by radiotherapy and chemotherapy in

advanced stages. This delayed management was due to low screening coverage, limited access to specialized facilities, and frequent diagnosis at an advanced stage of the disease (2, 11, 14). In this context, strategies were essentially reactive rather than preventive, resulting in high morbidity and mortality (2, 11).

Current approach

Management is part of a comprehensive and structured strategy, recommended by the World Health Organization, based on a continuum of complementary interventions. Primary prevention is ensured by prophylactic HPV vaccination, specifically targeting major oncogenic genotypes (4, 5, 11, 12). Secondary prevention relies on organized screening, dominated by high-sensitivity HPV DNA tests, enabling the early detection of persistent infections before the onset of precancerous lesions (2, 8).

Treatment of precancerous lesions is now favored at an early stage through conservative techniques such as cryotherapy, thermocoagulation, and loop electrosurgical excision procedure (LEEP), often integrated into so-called "screen-and-treat" strategies, particularly suited to resource-limited settings such as that of the Congo. The management of invasive cancers remains based on a multimodal approach combining surgery, radiotherapy, and chemotherapy, depending on the stage of progression (2, 11, 24).

Furthermore, women living with HIV require special attention due to an increased risk of viral persistence and rapid progression to precancerous and cancerous lesions, justifying more frequent screening and enhanced follow-up (8, 9, 11). The introduction of self-collected vaginal swabs is also a significant advance, improving access to screening by reducing sociocultural, geographical, and organizational barriers.

Persistent limitations of the current model

Current care continues to face several major challenges. The lack of specific antiviral treatment to eliminate HPV limits therapeutic options to managing lesions and preventing their progression (3, 18). Moreover, inequalities in access to screening, molecular diagnostic, and specialized treatment services persist, particularly in rural areas, hindering the overall effectiveness of implemented strategies (2, 8, 10).

Therapeutic perspectives and future innovations

Future prospects point toward more targeted, personalized, and predictive medicine. Therapeutic vaccines targeting the viral oncoproteins E6 and E7 aim to stimulate an immune response capable of eliminating infected cells (17, 18, 23, 25). Immunotherapies, particularly immune checkpoint inhibitors, also represent a promising avenue for enhancing the antitumor response (18, 21, 23). In parallel, targeted and gene therapies currently under development, as well as the use of molecular biomarkers such as p16INK4a, Ki-67, and E6/E7 gene expression, will allow for better patient stratification and more precise treatment tailoring (3, 17, 25). The integration of digital health technologies and artificial intelligence should also improve patient monitoring, the quality of clinical data, and the organization of large-scale screening programs.

Implications of genotypic data for health strategies

Available epidemiological data reveal a high diversity of high-risk HPV genotypes, with a predominance of HPV 16 but also significant circulation of other types such as HPV 35, 52, 58, and sometimes 68 (1, 5, 7, 8, 10). This genotypic diversity highlights the limitations of vaccination strategies restricted to HPV types 16 and 18 and justifies the interest in the nonavalent vaccine, which offers broader coverage of the main oncogenic genotypes (5, 11, 12, 15).

Similarly, these data support the use of broad-spectrum molecular tests capable of detecting several high-risk genotypes, rather than tests limited to a few specific types (8, 10). This approach improves the sensitivity of screening and allows for better adaptation of prevention strategies to local epidemiological realities (2, 8).

In conclusion, this review highlights the magnitude of the challenge posed by high-risk oncogenic human papillomaviruses (HPV) in the Republic of Congo. Characterized by sustained viral circulation and significant genotypic diversity, this situation underscores the urgent need to strengthen prevention, screening, and surveillance strategies. A thorough understanding of the local epidemiology of HPV is therefore essential for effectively guiding public health policies and making progress toward a sustainable reduction in the burden of cervical cancer among Congolese women.

Conflicts of interest

The authors declare no conflicts of interest.

Funding

This research received no specific funding from public, commercial, or non-profit organizations.

Author Contributions

Dimitry Moudiongui Mboundou Malanda: Investigation, formal analysis, writing—original draft. Paola Candyse Tsimba Lemba: Validation, methodology, writing—reviewing. Bredin Roch Bissala Nkounkou:—Formal analysis, writing—review and editing. Archange Michel Emmanuel Mboundou Malonga: Investigation, writing—reviewing. Anicet Luc Magloire Boumba: Resources, investigation writing—reviewing.

Data Availability

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethical Approval Not applicable.

Consent to Participate Not applicable.

Consent to Publish Not applicable.

Conflict of Interest The authors declare no competing interests.

References

1. Magagi LH, Lagare A, Bowo-Ngandji A, *et al.*, Prevalence and distribution of human papillomavirus (HPV) infection genotypes in West African populations: a systematic review and meta-analysis. *Infect Agents Cancer*. 2025; 20: 62. <https://doi.org/10.1186/s13027-025-00659-x>
2. Lekoane KMB, Kuupiel D, Mashamba-Thompson TP, *et al.*, Data on the prevalence, incidence, mortality, and trends of human papillomavirus-associated cancers in sub-Saharan Africa: an

- exploratory systematic review. *BMC Cancer*. 2019; 19: 563. <https://doi.org/10.1186/s12885-019-5781-3>
3. Wootton LM, Morgan EL. Ubiquitin and ubiquitin-like proteins in HPV-induced carcinogenesis. *Oncogene*. 2025; 44: 713-723. <https://doi.org/10.1038/s41388-025-03310-6>
4. Clifford GM, Smith JS, Plummer M, Muñoz N, Franceschi S. Human papillomavirus types in invasive cervical cancer worldwide: a meta-analysis. *Br J Cancer*. 2003; 88(1): 63-73. <https://doi.org/10.1038/sj.bjc.6600688>
5. Seyoum A, Assefa N, Gure T, Seyoum B, Mulu A, Mihret A. 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: 890880. <https://doi.org/10.3389/fpubh.2022.890880>
6. Malanda DMM, Mouamba FG, Boumba ALM, Miankoulila NP, Lemba PCT, Nkounkou BRB, *et al.*, Prevalence and genotypic distribution of human papillomavirus (HPV) in precancerous lesions of the cervix in Brazzaville, Republic of the Congo. *J Biosci Med*. 2026; 14: 440-456. <https://doi.org/10.4236/jbm.2026.143033>
7. Boumba LMA, Hilali L, Mouallif M, *et al.*, Specific human papillomavirus genotypes in 125 high-grade squamous cell lesions and cases of invasive cervical cancer in Congolese women. *BMC Public Health*. 2014; 14: 1320. <https://doi.org/10.1186/1471-2458-14-1320>
8. Tsimba Lemba PC, Boumba LMA, Péré H, *et al.*, Human papillomavirus genotype distribution by cytological status and associated risk factors in the general population of Congolese women living in urban and rural areas: implications for cervical cancer prevention. *Infect Dis Now*. 2023; 53(8): 104762. <https://doi.org/10.1016/j.idnow.2023.104762>
9. Akolbout-Manguilla MN, Kombo Bayonne ES, Makela N, *et al.*, Prevalence of high-risk human papillomaviruses and associated factors in women living with HIV and followed at the outpatient treatment center in Brazzaville, Congo. *Universal J Public Health*. 2023; 11(6): 961-969. <https://doi.org/10.13189/ujph.2023.110621>
10. Nganga PC, Boumba LMA, Lemba Tsimba PC, *et al.*, Prevalence and genotyping of human papillomavirus among women in the departments of Niari and Bouenza, Republic of the Congo. *J Biosci Med*. 2022; 10(1): 64-77. <https://doi.org/10.4236/jbm.2022.101007>
11. Denny L, Adewole I, Anorlu R, *et al.*, Human papillomavirus prevalence and type distribution in invasive cervical cancer in Sub-Saharan Africa. *Int J Cancer*. 2014; 134(6): 1389-1398. <https://doi.org/10.1002/ijc.28425>
12. Ogembo RK, Gona PN, Seymour AJ, *et al.*, Prevalence of human papillomavirus genotypes among African women with normal cervical cytology and neoplasia: a systematic review and meta-analysis. *PLoS One*. 2015; 10(4): e0122488. <https://doi.org/10.1371/journal.pone.0122488>
13. Jendoubi-Ferchichi M, Satouri L, Ghoul F, *et al.*, Phylogeny and classification of human papillomavirus 16 and 18 variants based on E6 and L1 genes in Tunisian women with cervical lesions. *Asian Pac J Cancer Prev*. 2018; 19(12): 3361-3366. <https://doi.org/10.31557/APJCP.2018.19.12.3361>
14. Okerosi S, Mokoh LW, Rubagumya F, *et al.*, Human papillomavirus-associated head and neck malignancies in Sub-Saharan Africa: a systematic review. *JCO Global Oncol*. 2023; 9: e2200259. <https://doi.org/10.1200/GO.22.00259>
15. Nyide B, Thomas M, Banks L, *et al.*, Genetic diversity of selected high-risk HPV types prevalent in Africa and not covered by current vaccines: a pooled sequence data analysis. *Int J Mol Sci*. 2025; 26(22): 11056. <https://doi.org/10.3390/ijms262211056>
16. Cornet I, Gheit T, Franceschi S, *et al.*, Genetic variants of human papillomavirus type 16: phylogeny and classification based on E6 and CSF. *J Virol*. 2012; 86. <https://doi.org/10.1128/JVI.00483-12>
17. Boumba LM, Assoumou SZ, Hilali L, *et al.*, Genetic variability in E6 and E7 oncogenes of human papillomavirus type 16 from Congolese cervical cancer isolates. *Infect Agent Cancer*. 2015; 10: 15. <https://doi.org/10.1186/s13027-015-0010-4>
18. Aggarwal N, Yadav J, Chhakara S, *et al.*, Phytochemicals as potential chemopreventive and chemotherapeutic agents for HPV-driven head and neck cancer: current evidence and future prospects. *Front Pharmacol*. 2021; 12: 699044. <https://doi.org/10.3389/fphar.2021.699044>
19. Fokom-Domgue J, Combescure C, Fokom-Defo V, *et al.*, Performance of alternative strategies for primary cervical cancer screening in Sub-Saharan Africa: systematic review and meta-analysis. *BMJ*. 2015; 351: h3084. <https://doi.org/10.1136/bmj.h3084>

20. Rahman R, Clark MD, Collins Z, *et al.*, Cervical cancer screening decentralized policy adaptation: an African rural-context-specific systematic literature review. *Glob Health Action*. 2019; 12(1): 1587894. <https://doi.org/10.1080/16549716.2019.1587894>
21. Shi L, Chen H, Zhang Z, *et al.*, Evolving HPV diagnostics: current practice and future frontiers. *Front Cell Infect Microbiol*. 2025; 15: 1681779. <https://doi.org/10.3389/fcimb.2025.1681779>
22. Twabi HH, Sibande W, Mhango O, *et al.*, Acceptability of self-sampling for high-risk HPV DNA testing for primary cervical cancer screening among women in Thyolo, Malawi: a qualitative study. *PLOS Glob Public Health*. 2025; 5(9): e0004763. <https://doi.org/10.1371/journal.pgph.0004763>
23. Wang X, Wang Q, Ding G, *et al.*, Artificial intelligence for colposcopic and cytological image analysis in early cervical cancer detection. *iScience*. 2026; 29(2): 114627. <https://doi.org/10.1016/j.isci.2026.114627>
24. Cubie HA, Campbell C. Cervical cancer screening: the challenges of complete pathways of care in low-income countries. *Women's Health (Lond)*. 2020; 16: 1745506520914804. <https://doi.org/10.1177/1745506520914804>
25. Trimble CL, Morrow MP, Kraynyak KA, *et al.*, Safety, efficacy, and immunogenicity of VGX-3100, a therapeutic synthetic DNA vaccine targeting HPV 16 and 18 E6 and E7 proteins. *Lancet*. 2015; 386(10008): 2078-2088. [https://doi.org/10.1016/S0140-6736\(15\)00239-1](https://doi.org/10.1016/S0140-6736(15)00239-1)

How to cite this article:

Dimitry Moudiongui Mboundou Malanda, Paola Candyse Tsimba Lemba, Bredin Roch Bissala Nkounkou, Archange Michel Emmanuel Mboundou Malonga and Anicet Luc Magloire Boumba. 2026. High-Risk Oncogenic Human Papillomavirus Infection among Women in the Republic of the Congo: A Systematic Review. *Int.J.Curr.Microbiol.App.Sci*. 15(7): 20-31. doi: <https://doi.org/10.20546/ijcmas.2026.1507.003>