Review Article

Challenges and Advances in Cervical Cancer Prevention for HIV-Positive Individuals

Abstract: Human papillomavirus (HPV) is a well-known cause of cervical cancer, with HIV infection exacerbating the prevalence of high-risk HPV (HR-HPV) and cervical intraepithelial neoplasia (CIN). ART has been shown to reduce the likelihood of developing squamous intraepithelial lesions (SILs) and HR-HPV prevalence, although its impact on invasive cervical cancer remains inconclusive. In a study in Accra, 250 sexually active HIV-positive women exhibited a high HPV prevalence, emphasising the need for regular screening. HPV testing, visual inspection with acetic acid (VIA), and cytology-based tests are primary screening methods, each with specific advantages and limitations. Cervical cancer rates in sub-Saharan Africa are alarmingly high, necessitating targeted vaccination and prevention strategies.

Keywords: HIV; HPV; prevalence; vaccination strategy; HPV screening; cervical intraepithelial neoplasia

1. Introduction

Human papillomavirus (HPV) is a well-established cause of cervical cancer, with HIV infection increasing the prevalence of high-risk HPV (HR-HPV) and cervical intraepithelial neoplasia (CIN). HIV and HPV share transmission routes, and HIV exacerbates cervical carcinogenesis through mechanisms such as inhibition of tumour suppressor genes and alteration of cell cycle regulation. Chronic inflammation and cytokine imbalances, notably increased IL-10 levels, are linked to cervical cancer progression. Despite ART improving overall health, cervical cancer remains a significant issue, highlighting the need for better screening and prevention [1,9–18].

Despite challenges, HPV vaccination offers hope for cancer control, highlighting the importance of scientific approaches to overcome barriers and achieve vaccination target.

2. HPV and Cervical Dysplasia in HIV-Positive Women

A cross-sectional study in Brazil aimed to evaluate cytokine levels in cervicovaginal lavage and their association with HPV infection, HIV viral suppression, and other factors among HIV-positive women. This study revealed a high prevalence of specific HPV types, including HPV52, HPV51, HPV16, HPV18, HPV35, and HPV66. Notably, HR HPV prevalence was higher in HIV-positive men (25.7%) compared to HIV-negative men (15.8%). HPV16 was the most common subtype, with age and lower CD4 counts increasing the risk of cervical dysplasia. Most patients were asymptomatic,

with peak HPV incidence observed in the 20-40 age group. These findings underscore the importance of routine Pap smear screening for early detection and management of dysplasia in infected women [2,19–22].

3. ART and HPV-Related Cervical Lesions

Women on HAART showed a reduced likelihood of developing squamous intraepithelial lesions (SILs). Evidence suggests that HAART, particularly when combined with higher CD4 counts, reduces HR-HPV prevalence. Severe immunosuppression without HAART increases HR-HPV risk. While ART reduces the prevalence of CIN2 and CIN3, its impact on invasive cervical cancer remains inconclusive. Enhanced surveillance and further research on ART's impact on cervical cancer are needed [3,23]. Women living with HIV (WLWH) face a high risk of cervical cancer due to high-risk Human papillomavirus (hrHPV). This study examined HPV prevalence, genotypes, and cervical cancer risk factors in 250 sexually active WLWH in Accra. Cervical swabs tested for HPV showed a 60% overall prevalence, with 44.4% for hrHPV. Employment and HAART were protective against hrHPV. Genotype analysis revealed 25% had hrHPV group 1 (including types 16 and 18) and 46.8% had multiple HPV types. The study highlights the need for regular HPV screening in high-risk groups to prevent cervical cancer [24]. Cervical cancer screening methods are consistent across women. These methods include visual inspections (VIA and VILI), cytology-based tests (Pap smear and LBC), and HPV DNA testing. VIA allows training of lower cadre staff and offers the benefit of concurrent screening and treatment, although it has a high false positive rate. The choice of method depends on available resources [25–29].

4. Systematic Review and Meta-Analysis on ART and HPV

A comprehensive review and meta-analysis of 31 studies assessed ART's association with HR HPV prevalence and cervical abnormalities. The analysis included 6,537 women for HR HPV prevalence and 9,288 for high-grade cervical lesions (HSIL-CIN2+). ART was associated with a lower prevalence of HR HPV (adjusted odds ratio [aOR] 0.83) and a reduced prevalence of HSIL-CIN2+ (0.65). Longitudinal studies indicated that ART decreased HSIL-CIN2+ incidence (0.59), reduced progression of SIL (adjusted hazard ratio [aHR] 0.64), and increased regression of SIL or CIN (1.54). Among 15,846 women, ART was linked to a reduced incidence of invasive cervical cancer (crude HR 0.40). These findings suggest that early ART initiation and adherence may reduce cervical lesion incidence and progression, highlighting the need for further cohort studies to confirm these effects [4]. Observational studies vary in design and outcomes. With the growing number of women on ART, better understanding the effects of ART, immune recovery, and virological control on HPV and cervical lesion progression is crucial for effective screening programmes [30–32].

The effect of antiretroviral therapy (ART) on anal high-risk HPV and lesion progression in people living with HIV is not well understood. This systematic review and meta-analysis examined the association of ART, HIV-RNA plasma viral load (PVL), and CD4 cell counts with anal HPV infection, anal intraepithelial neoplasia (AIN), and anal cancer. From 6777 studies, 122 matched the inclusion criteria, covering 417,006 individuals. ART users had a 35% lower prevalence of high-risk HPV, with prolonged ART reducing high-risk HPV prevalence by 10% per year [33].

Recent epidemiologic studies have shown a significant increase in anal cancer among specific male sub-populations, particularly men who have sex with men and HIV-positive individuals. In addition, this highlights that squamous cell carcinoma of the anus (SCCA) is an escalating issue for women in the United States, especially those who are HIV-positive [34–36].

This study systematically reviewed publications on the epidemiology of anal HPV infection, anal intraepithelial neoplasia (AIN), and anal cancer in women, focusing on research from January 1997 to September 2013, during the combined antiretroviral therapy era. The review included 37 publications on anal HPV and cytology and 23 on anal cancer. Among HIV-positive women, anal high-risk HPV (HR-HPV) prevalence ranged from 16-85% [37].

5. HPV Prevalence among Men and Anal Cancer

HPV is a significant cause of sexually transmitted diseases, with high-risk types such as HPV16 and HPV18 linked to various cancers, including cervical, penile, anal, and head-and-neck cancers. The prevalence of HPV and related cancers is notably high in sub-Saharan Africa, where both HPV rates are elevated, and effective screening is limited. A systematic review and meta-analysis of 11 studies involving 9,342 men from sub-Saharan Africa revealed a high HPV prevalence, with overall rates ranging from 19.1% to 100%. HPV16 and HPV52 were the most common HR types, while HPV6 was the predominant low-risk type. No clear age-related trend in HPV prevalence was observed. These findings indicate a substantial HPV burden among men in the region and underscore the potential benefits of HPV vaccination programs [7].

This study assessed anal HPV and HSIL prevalence in men aged 50+ in San Francisco. Among 129 men who have sex with men living with HIV (MSMLWH) and 109 not living with HIV (MSM-not-LWH), 47% and 37% had anal HSIL, respectively. Given that treating HSIL can prevent anal cancer, screening for anal cancer in older MSM is recommended [38]. Anogenital HPV infection is the most common STI globally, affecting skin and mucosal cells, and is linked to various lesions and cancers, including anal carcinoma. Immunosuppression, increases the risk of HPV acquisition and anal dysplasia. Anal cancer is a prevalent non-AIDS-defining disease in people living with HIV, especially women and MSM [39–47]. Men who have sex with men (MSM) face a high risk of HPV-associated anal cancer. Despite the high prevalence of HPV and precursors, progression to cancer appears lower than for cervical lesions, highlighting the need for large, prospective studies to develop screening guidelines [48].

Anal cancer, caused by HPV, is a significant risk for HIV-infected men. This cross-sectional study from the CARH·MEN cohort at Hospital Germans Trias i Pujol (Spain) examined HPV prevalence and genotype distribution in HIV-infected men, including MSM and men who have sex with women (MSW). Cytological abnormalities were found in 40% of MSM and 20% of MSW [49]. This study of people living with HIV and a history of malignancy found high prevalence rates of HR-HPV types 16 and 18, with 89% infected. Anal HSIL was observed in 38% of patients. The results underscore the need for regular anal cancer screening in this population [50].

This study investigated anal high-grade squamous intraepithelial lesions (HSIL) among men who have sex with men (MSM) and transgender women, who started antiretroviral therapy during acute HIV acquisition in Bangkok. Of 93 participants with a median age of 26, 11.8% had baseline histologic anal HSIL. The incidence of new HSIL was 19.7 per 100 person-years [51].

In recent decades, cervical cancer incidence has decreased due to cervical cytology screening, which identifies precursors such as high-grade cervical intraepithelial neoplasia (CIN) 2-3. Ablation of these lesions has notably reduced cervical cancer rates. The highest incidence of cervical cancer is now seen in regions without routine screening. Anal cancer, biologically similar to cervical cancer, is also linked to human papillomavirus (HPV). High-grade anal intraepithelial neoplasia (HGAIN), the anal equivalent of CIN, can progress to anal cancer. Screening for HGAIN involves anal cytology and high-resolution anoscopy (HRA)-directed biopsy, mirroring cervical cancer detection techniques. Given the similarities between cervical and anal cancer, it is hypothesised that removing HGAIN could potentially reduce anal cancer risk. Additionally, a high proportion of HIV-positive individuals have HGAIN, suggesting that large numbers would need treatment. Risks associated with unnecessary treatments and the need for further research on biomarkers to predict cancer progression are also concerns. Moreover, there are practical issues such as the limited number of trained clinicians and the costs and potential adverse effects of HGAIN treatments [52–59].

6. HPV and HIV in Tanzania

A study in Tanzania assessed HPV prevalence and type distribution among 1,813 men, revealing an overall HPV prevalence of 20.5%. The most common HR HPV types were HPV52, HPV51, HPV16, HPV18, HPV35, and HPV66. HIV-positive men had a significantly higher prevalence of HR HPV (25.7%) compared to HIV-negative men (15.8%, $P = 0.0027$). Although HPV16, HPV18, and multiple HR HPV types were somewhat more common in HIV-positive individuals, these differences were not statistically significant for other HPV types. This study highlights the high prevalence of specific

HPV types and suggests that HIV status has a limited impact on HPV type distribution. Consequently, HPV vaccines are likely effective in preventing HPV infection irrespective of HIV status [8].

A study in Dar es Salaam, Tanzania, examined the prevalence and association of human papillomavirus (HPV) infections with HIV among cervical cancer and non-cancer patients. Researchers analysed tumour biopsies from 53 women with cervical or vaginal cancer and cervical swabs from 359 non-cancer patients. HPV types 16 and 18 were detected in 38% and 32% of cervical cancer biopsies, respectively. Among non-cancer patients, 59% had HPV-DNA, with types 16 and 18 present in 13.2% and 17.5% of cases. HPV type 18 prevalence was notably high in Tanzania compared to other regions. Key risk factors for HPV infection in non-cancer patients included young age, HIV infection, trichomonas vaginalis infection, single status, early sexual debut, and young age at menarche. HIV-positive patients were more likely to have HPV types 16 and 18 but did not show an increased risk of cervical abnormalities or cancer. Overall, the rate of cervical cytological abnormalities was 2.8%, and HIV prevalence among cervical cancer patients was low at 3%. The study concluded that there was no clear association between HIV infection and cervical cytological abnormalities or cancer in this population [60].

A cross-sectional study at Bugando Medical Centre in Mwanza, Tanzania, focused on HPV genotypes associated with cervical squamous intraepithelial lesions (SIL) in HIV-infected women. Between August and October 2014, cervical cells from 255 HIV-positive women (mean age 39.2 years) were analysed using PCR and sequencing. HPV DNA was detected in 54.1% of the participants, with 26 different genotypes identified, including 17 high-risk (HR) types. The most prevalent HR genotypes were HPV-52, HPV-58, HPV-35, and HPV-16. Women with low CD4 counts, particularly below 100, and those with SIL had significantly higher risks of HPV infection. The study noted that women with low CD4 counts had a 1.20 risk ratio for HPV positivity, while those with SIL had a 1.37 risk ratio. It emphasises the predominance of less common HR HPV types in HIV-positive women with compromised immune systems and highlights the need to evaluate HPV vaccine effectiveness in regions with high HIV prevalence [61].

A study using a deterministic transmission-dynamic compartment model assessed the impact of interventions on cervical cancer in Tanzania from 1995 to 2070. Tanzania has a high cervical cancer incidence rate of 59.1 per 100,000 women annually. The model found that voluntary medical male circumcision (VMMC) prevented 2,843 cervical cancer cases and 1,039 deaths from 1995 to 2020. By 2070, VMMC is projected to reduce cervical cancer incidence and mortality rates by 28% and 26%, respectively. Additionally, the combined use of antiretroviral therapy (ART) and targeted pre-exposure prophylaxis (PrEP) is expected to lower cervical cancer incidence and mortality rates to 35.82 and 25.35 per 100,000 women by 2070. These findings highlight the significant long-term impact of prevention efforts in reducing cervical cancer incidence and mortality in Tanzania [62].

Women in Tanzania face a high risk of cervical cancer, particularly due to human papillomavirus (HPV) infection. Despite efforts to prevent and treat cervical cancer, high-risk HPV (HR-HPV) positivity rates remain alarmingly high, reaching 46.7% in some regions. To combat this, the World Health Organization (WHO) has set "90–70–90" targets for 2030, aiming for 90% of girls to be fully vaccinated against HPV by age 15, 70% of women to be screened twice by ages 35 and 45, and 90% of cervical cancer cases to receive proper treatment. Tanzania, with the highest cervical cancer incidence in East Africa, reported 10,241 new cases in 2020. Tanzania''s' Ministry of Health introduced a nationwide HPV vaccination programme in 2018 for 14-year-old girls after a successful pilot in the Kilimanjaro region. By the end of 2019, vaccine coverage reached 78% for the first dose and 49% for the second, but challenges remain, particularly for older, unvaccinated populations. In addition to vaccination, a nationwide screening and treatment programme using visual inspection with acetic acid (VIA) and cryotherapy has been in place since 2011 to treat precancerous lesions. Ongoing efforts are essential to ensure comprehensive HPV prevention and treatment across all age groups [63–76].

7. HPV and Cervical Cancer in Sub-Saharan Africa

Cervical cancer rates in sub-Saharan Africa (SSA) are critically high, with over 75,000 new cases and 50,000 deaths annually. The region is characterised by a diverse range of high-risk (HR) HPV genotypes, particularly HPV-16, -18, -35, and -52. A systematic review of 27 studies involving 16,506 participants revealed a pooled HR HPV prevalence of 34% (95% CI: 29-39%). HPV-16 was the most common genotype (13.8%), followed by HPV-52 (9.9%) and HPV-18 (9%). Variations in HR HPV prevalence across SSA emphasise the need for region-specific vaccination strategies [6].

HPV testing is being considered as a primary cervical cancer screening method in developing countries. A study comparing visual inspection with acetic acid (VIA) and cytology for detecting cervical intraepithelial neoplasia Grade 2 or higher (CIN2+) in Cameroon found VIA less effective than cytology, with sensitivity and specificity of 25.0% and 74.2%, respectively, compared to cytology's sensitivity of 90.0% and specificity of 85.2% [77]. Additionally, a review of 15 studies showed that visual inspection with Lugol's iodine (VILI) had higher sensitivity (95.1%) compared to VIA (82.4%), making VILI a potential alternative for primary screening in SSA [78].

This review examined policy documents and literature from Kenya, Tanzania, and Uganda, involving consultations with key government and NGO personnel in Tanzania and Uganda. The consultations, involving 25 to 30 individuals in each country, focused on current policies, practices, and the decision-making processes for updating screening policies. The findings emphasise the opportunities and challenges of introducing HPV testing in these regions, highlighting the need for evidence-based policies to improve cervical cancer prevention. This study assessed the feasibility of implementing HPV testing as the primary screening method for cervical cancer in sub-Saharan Africa, particularly in Kenya, Tanzania, and Uganda. It emphasised the importance of integrating HPV testing with existing healthcare infrastructure, particularly HIV/AIDS initiatives, to improve cervical cancer screening and treatment outcomes. The study explored the infrastructural requirements for integrating HPV vaccination and screening programmes with existing health services in sub-Saharan Africa. It identified key barriers to vaccine delivery, such as limited healthcare infrastructure and funding, and suggested that school-based vaccination programmes and rapid HPV testing could enhance coverage in GAVI-eligible countries. This manuscript outlines the infrastructural and logistical needs for introducing HPV vaccination and VIA or HPV testing in sub-Saharan Africa. It argues for a combined approach to vaccinating pre-adolescent girls and screening women over 30 to reduce the burden of cervical cancer, emphasising the role of international funding and partnerships in facilitating this effort [79–82].

HPV vaccination has significantly improved the prospects for cervical cancer prevention in SSA. GAVI's inclusion of the HPV vaccine has accelerated its introduction in eligible countries. School- and campaign-based vaccination programmes, along with investments in infrastructure, will further expand coverage. Population models estimate that vaccinating pre-adolescent girls at 70% coverage could prevent over 670,000 cervical cancer cases across SSA, making HPV vaccination highly cost-effective in most GAVI-eligible countries [83]. Rwanda's successful HPV vaccination programme serves as a model for other SSA nations, highlighting the importance of government support, school-based delivery, and social mobilisation [84].

Cervical cancer is the leading cause of cancer deaths among women in sub-Saharan Africa (SSA), contributing to 24.55% of global cervical cancer mortality. HPV vaccination presents a critical opportunity for cancer control, with 18 SSA countries having national vaccination programmes by 2020. However, large population countries face lower vaccination coverage, and the success of national rollouts may not match that of initial demonstration projects. To improve and sustain high coverage, prioritising West African countries and learning from the experiences of early adopters is crucial [85].

A study in sub-Saharan Africa (SSA) evaluated the knowledge, awareness, and acceptability of cervical cancer, HPV, and HPV vaccines. While there was high willingness to vaccinate, knowledge about HPV and the vaccines was low. Only six countries met the GAVI Alliance's criteria for HPV vaccine support, with 70% DTP3 coverage being a key requirement. Most SSA countries are unprepared for national HPV vaccine rollouts. To address this, education and pilot programmes are

essential to improve awareness, increase acceptability, and qualify for GAVI support, which provides funding to enhance vaccine access in low-income countries [86].

8. HPV and ART Interaction

In 2013, 35 million people were living with HIV globally, with 71% in sub-Saharan Africa. Cervical cancer remains the most common female cancer in the region, with HR-HPV DNA present in 99.7% of cervical cancers. HPV types 16 and 18 are responsible for about 70% of cases. Despite the introduction of prophylactic HPV vaccines, HPV 16's persistence and resistance to clearance remain challenges. WHO recommends HPV vaccination for adolescent girls and women up to 26 years in resource-limited settings. Access to ART has improved in sub-Saharan Africa, yet its impact on HPV 16 prevalence and cervical cancer remains inconclusive. A meta-analysis indicated that women on HAART had a lower HR-HPV prevalence, suggesting a primary prevention opportunity for unvaccinated women. However, HPV 16's ability to evade immune surveillance compared to other genotypes poses challenges. Enhanced surveillance is needed to understand HAART's impact on HPV 16 and cervical cancer. The review highlights the need for further research to clarify HAART's role in managing HPV 16 and cervical cancer in HIV-positive women in Africa [5].

In Kampala, Uganda, a study from 2017 to 2020 found that women living with HIV (WLWH) on antiretroviral therapy (ART) had a higher prevalence of high-risk HPV (hrHPV) and cervical intraepithelial neoplasia (CIN) compared to HIV-negative women. Despite ART, WLWH were at increased risk for multiple hrHPV types and high-grade cervical abnormalities, highlighting the need for targeted cervical cancer prevention and research [87]. Vulvar cancer (VC), though rare, is increasingly prevalent, particularly among younger women due to HPV. Early-stage VC is typically managed with surgery, while advanced cases require multimodal treatments including chemotherapy and radiotherapy. Due to its rarity, randomised controlled trials are limited, and prognosis remains poor. Recent advances in immunoncology highlight the potential of immune checkpoint inhibitors (ICIs) in improving outcomes [88]. Cervical cancer development is linked to the integration of human papillomavirus (HPV) genomes into host chromosomes, leading to epigenetic changes and dysregulation of host gene expression. Using H'PV Integrated Site Capture' (HISC) and H'PV16-Specific Region Capture Hi-C' techniques, we discovered that HPV integration typically occurs through microhomology-mediated repair (MHMR), which can result in host sequence deletion or amplification. Chromatin interactions between the integrated HPV genome and host chromosomes disrupt host gene regulation within topologically associating domains (TADs). These findings suggest that HPV integration influences host gene expression through changes in genome interactions rather than proximity to cancer-causing genes [89].

HIV and HPV co-infection is prevalent, especially among men who have sex with men (MSM), and is associated with impaired HPV clearance due to HIV-induced immune dysfunction. Despite effective antiretroviral therapy (ART), HPV-related cancer rates remain high among PLWH. Public health strategies, including universal HPV vaccination and targeted screening programs, aim to reduce HPV transmission and related disease burden [90]. Figure 1 shows a representation of the HPV genome.

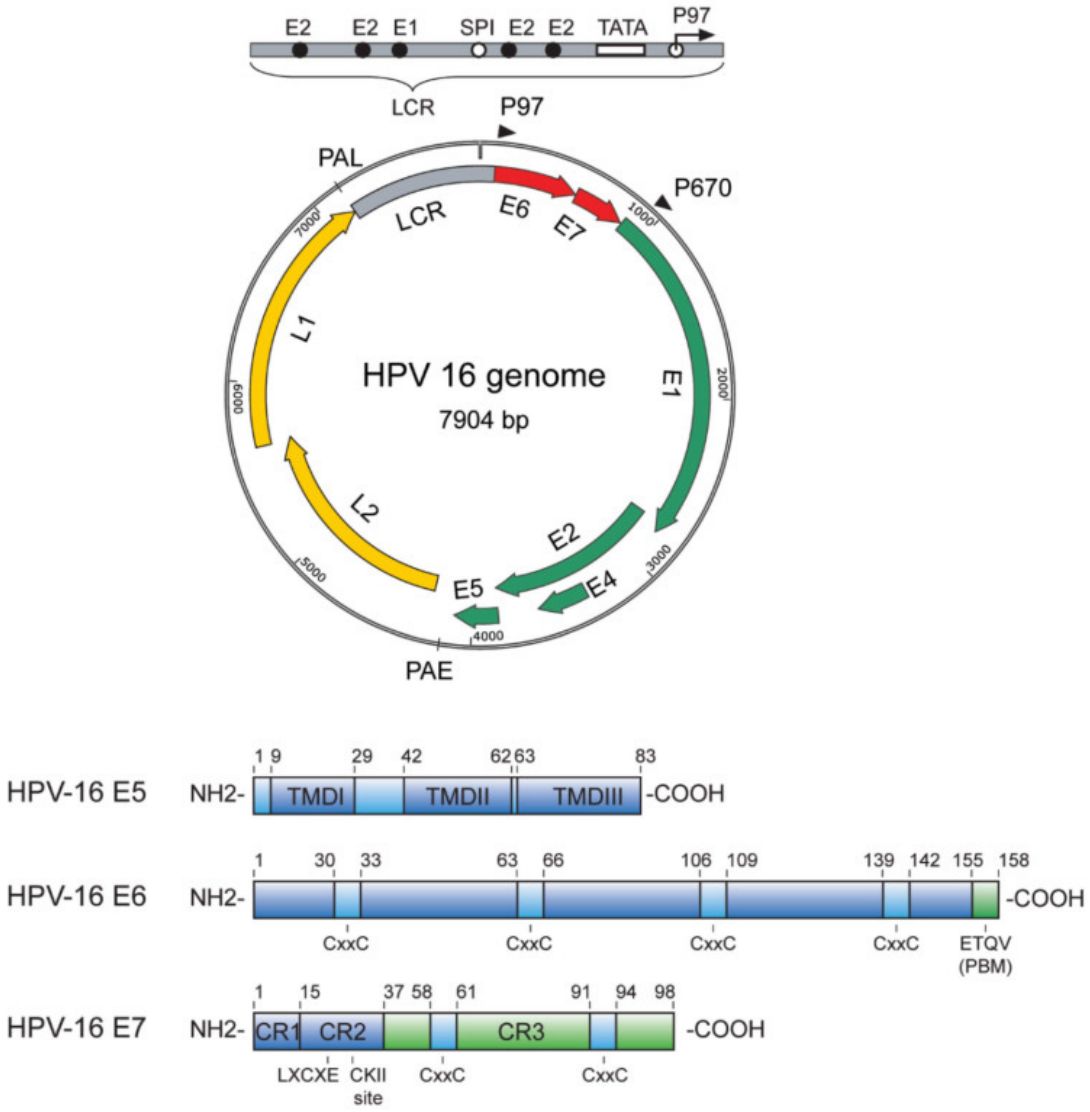

Figure 1. Representation of the HPV genome. Taken from [91].

HIV-infected individuals have a higher incidence of HPV-associated oropharyngeal cancers compared to those without HIV. HIV may also increase the risk of other cancers, including those of the head, neck, liver, lung, and kidney. This study reveals that prolonged exposure to cell-free HIV-1 virions or HIV-1 proteins gp120 and tat induces epithelial-mesenchymal transition (EMT) and enhances invasiveness in HPV-16-immortalised anal and cervical epithelial cells, as well as in HPV-16-infected and HPV-16-negative oral cancer cells. EMT, driven by gp120 and tat, leads to cell detachment and reattachment with intermediate EMT markers, including stem cell markers CD133 and CD44. This suggests that HIV-1 promotes de-differentiation of neoplastic cells into cancer stem cells, which may be resistant to treatment. Interventions targeting TGF-β1, MAPK signalling, vimentin, and restoring E-cadherin expression, alongside ART, could mitigate HIV-1's' role in advancing both HPV-associated and HPV-independent epithelial cancers [92].

The study explores the interaction between Aurora kinase A (AurA) and the E6 oncoprotein of human papillomavirus (HPV), which is crucial for HPV-induced carcinogenesis. AurA, a mitotic regulator often dysregulated in cancers, binds preferentially to the E6 protein, and this association is vital for the proliferation and survival of HPV-positive cells. The research identified that the C-terminus of E6, upstream of its PDZ binding motif, is essential for forming the AurA-E6 complex in

the nucleus. The level of E6 expression correlates positively with AurA expression. Functionally, the AurA-E6 interaction regulates the expression of cyclin E and phosphorylated histone H3, impacting G1/S and mitotic phases of the cell cycle. Depleting AurA also reduced the invasiveness of HPV-positive cells, though inhibiting AurA alone may not fully diminish the oncogenic potential of E6. This study reveals how HPV E6 exploits AurA to disrupt cell cycle checkpoints and drive cancer progression. These insights suggest that targeting the AurA-E6 complex could be a promising therapeutic strategy for HPV-associated cancers [93].

The study addresses the increasing incidence of oropharyngeal squamous cell carcinoma (OPSCC) and the need to understand its etiology for effective treatment. Using an integrative genomics approach, researchers analysed RNA-Sequencing (RNA-Seq) data from 46 HPV-positive head and neck squamous cell carcinoma cases and 25 normal controls. Differential marker selection, based on a log2FoldChange (FC) score of 2 and adjusted p-value < 0.01, identified 714 genes, which were then refined to 73 using the Particle Swarm Optimization (PSO) algorithm. Machine learning models revealed seven key genes—ECT2, LAMC2, DSG2, FAT1, PLOD2, COL1A1, and PLAU—that significantly contribute to model performance. These genes were linked to OPSCC through gene set enrichment analysis, protein-protein interactions, and disease ontology mining. Survival analysis highlighted strong over-expression of three key genes in OPSCC samples from "The Cancer Genome Atlas." These findings provide crucial insights into OPSCC pathogenesis and potential targets for therapy [94].

9. Research in the Caribbean

HPV and HIV, both sexually transmitted infections, contribute to the prevalence of cervical dysplasia and cancer in women. In Jamaica, cervical cancer is the second leading cause of cancer-related deaths, with a rate of 27.5 per 100,000 women. This study examines the seroprevalence of anti-HIV antibodies in women with abnormal Pap smears to assess HIV's' influence on cervical dysplasia. Using ELISA for screening and Western blot for confirmation, the seroprevalence of HIV was found to be 0.85% [94]. Cervical cancer is a leading cause of cancer death among women in Jamaica, second only to breast cancer. This study investigated the association between blood type and cervical dysplasia/cancer in 319 women, including 234 with abnormal Pap smears and 85 controls. Blood types A, B, AB, and O were distributed similarly to the general population. A slight association was found between blood type O and cervical dysplasia/cancer, with stronger links to factors such as multiple sexual partners, biological fathers, children, and hormonal contraceptive use [95].

10. Conclusions

Cervical cancer, particularly among women living with HIV (WLWH), remains a significant global health challenge, highlighting the need for effective prevention and screening strategies. While antiretroviral therapy (ART) has improved survival rates for HIV-positive individuals, its effect on cervical cancer, especially invasive cases, remains unclear. High-risk HPV types play a major role in cervical cancer development, and although ART reduces the prevalence of HPV and cervical lesions, further research is required to determine its full impact on cancer incidence.

Screening methods such as HPV testing, visual inspection with acetic acid (VIA), and cytology are vital in cervical cancer prevention and management. Additionally, HPV vaccination programmes, particularly in sub-Saharan Africa, show promise in reducing cervical cancer rates. However, barriers to vaccine implementation need to be addressed. Integrating HPV vaccination into existing healthcare systems and improving access to screening services are crucial steps.

Research indicates that combining vaccination with regular screening is both cost-effective and essential for reducing cervical cancer mortality. Continued efforts in research, policy development, and resource allocation are necessary to address the challenges of cervical cancer prevention and control, particularly in regions with high HIV and HPV prevalence [96].

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