

# Human Papillomavirus Disease

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Panel's Recommendations
<ul style="list-style-type: none"><li>• Human papillomavirus (HPV) vaccination is recommended in people with HIV aged 9–12 (AIII) and for those aged 13–26 years who have not been previously vaccinated or have not completed the vaccine series (AIII).</li><li>• Ideally, HPV vaccine should be administered before an individual becomes sexually active (AIII).</li><li>• HPV vaccination is recommended starting at the age of 9 years for children with a history of sexual abuse.</li><li>• Bivalent, quadrivalent, and nonavalent HPV vaccines are approved in the United States, but only the nonavalent vaccine is currently available in the United States. Three doses are recommended for all people with HIV, regardless of sex or age at which the vaccine was administered.</li><li>• If the bivalent or quadrivalent vaccine was previously administered, revaccinating with a three-dose series using the nonavalent vaccine should be considered (CIII).</li><li>• Regardless of vaccination status, screening for cervical cancer should start at age 21 years (see the <a href="#">Human Papillomavirus Disease section of the Adult and Adolescent Opportunistic Infection Guidelines</a>) (AIII).</li><li>• Latex condoms should be used during every act of vaginal, anal, and oral sexual intercourse to reduce the risk of exposure to sexually transmitted pathogens, including HPV (AII).</li><li>• Anogenital warts in children should be treated per the <a href="#">2021 Centers for Disease Control and Prevention Sexually Transmitted Infections Treatment Guidelines</a>.</li></ul>
<p><i>Rating of Recommendations: A = Strong; B = Moderate; C = Optional</i></p> <p><i>Rating of Evidence: I = One or more randomized trials in children<sup>†</sup> with clinical outcomes and/or validated endpoints; I* = One or more randomized trials in adults with clinical outcomes and/or validated laboratory endpoints with accompanying data in children<sup>†</sup> from one or more well-designed, nonrandomized trials or observational cohort studies with long-term clinical outcomes; II = One or more well-designed, nonrandomized trials or observational cohort studies in children<sup>†</sup> with long-term outcomes; II* = One or more well-designed, nonrandomized trials or observational studies in adults with long-term clinical outcomes with accompanying data in children<sup>†</sup> from one or more similar nonrandomized trials or cohort studies with clinical outcome data; III = Expert opinion</i></p> <p><i><sup>†</sup>Studies that include children or children/adolescents, but not studies limited to postpubertal adolescents</i></p>

## Epidemiology

This chapter focuses primarily on prepubescent and pubescent children with HIV. Prepubescent is before puberty, generally age 0 to 8 years. Pubescent means attained puberty but not fully mature, which varies individually, but is generally started by age 13 in most females and by age 14 in most males in the United States. For complete recommendations on older adolescents (postpubescent defined by reaching sexual maturity rating 4), refer to the [Human Papillomavirus Disease section of the Guidelines for the Prevention and Treatment of Opportunistic Infections in Adults and Adolescents With HIV](#).

Human papillomavirus (HPV) infects cutaneous and mucosal squamous epithelium. More than 200 distinct types of HPV exist.<sup>1</sup> The majority of HPVs of clinical importance fall predominantly into the alpha HPV genus.<sup>1</sup> HPV can be detected on normal healthy mucosal and cutaneous surfaces but also is associated with anogenital warts in children and anogenital and oropharyngeal precancers

and cancers in adults and, in rare cases, in adolescents. HIV reflects a significant increased risk of HPV-associated disease in adults including cervical, anal, vulvar, and vaginal precancers and cancers and oropharyngeal cancers.<sup>2</sup> All of these precancers and cancers are rare in the general population.<sup>3</sup> In contrast, people who were previously treated for a childhood cancer have a fourfold to eightfold increase in oropharyngeal cancers and a ninefold to 14-fold increase for anal cancers, with the youngest age at diagnosis being 10 years of age.<sup>4</sup> How this translates to children with HIV is uncertain. The HIV status of the rare cases of cervical and anal cancers in children is unknown. Both cutaneous and anogenital warts also reflect a significant burden in children and adults with HIV.<sup>5</sup>

Certain types are found predominantly in cutaneous warts (such as HPV2), whereas other distinct mucosal types are associated with anogenital and oropharyngeal cancers. The mucosal HPV types found in cancers are referred to as high-risk HPV (hr-HPV) types. Of the approximately 40-plus genital (i.e., mucosal) HPV types, 12 types have been established as hr-HPV (16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59), and 6 as probable high-risk (26, 53, 66, 68, 73, 82).<sup>1</sup> HPV16 alone accounts for 50% of all squamous cell (SC) cervical cancers and 80% to 90% of all SC anal cancers.<sup>6</sup> Of the HPV-associated vulvar, vaginal, penile, and oropharyngeal cancers, 50% to 80% also are attributed to HPV16.<sup>7-9</sup> Skin warts associated with HPV are common in children, whereas mucosal warts (including anogenital and oral warts) are less common.<sup>10-14</sup>

HPV-associated cutaneous warts are transmitted by close person-to-person contact that is facilitated by minor trauma to the skin. Skin warts are most commonly associated with cutaneous HPV types 1, 2, 3, 4, 10, 27, and 57 and are associated with distinct wart histology.<sup>15,16</sup> The estimated prevalence of skin warts in immunocompetent children varies widely by population. A British cohort study of 9,263 schoolchildren found 3.9% prevalence at age 11 years and 4.9% prevalence at age 16 years, while a Dutch study of 1,465 schoolchildren found 15% prevalence at age 4 years that rose to 44% at age 11 years.<sup>11,12,17</sup> In comparison, children with compromised cellular immunity often have intense and widespread appearance of both cutaneous and mucosal warts. Unfortunately, no data are available on prevalence or incidence rates of skin warts in children with HIV. One retrospective study in South Africa examined 65 children presenting for treatment of anogenital warts. Out of the 23 children who required surgical intervention, those who were known to have HIV (12 patients) required a significantly higher number of surgical procedures to reach clinical resolution, compared to those known to not have HIV (6 patients).<sup>18</sup>

HPV-associated anogenital warts are known to be transmitted by sexual contact, thereby raising the concern of sexual abuse when diagnosed in prepubertal children.<sup>14,19</sup> Several studies have shown that anogenital warts can be found in children with no evidence of sexual abuse, suggesting that transmission may occur through other means, such as perinatally<sup>20</sup> or through other nonsexual means (e.g., autoinoculation or transmission from the hands or mouth of a caretaker).<sup>21-23</sup> The prevalence of HPV-associated anogenital warts varies by population and risk factors. For example, varying prevalences of HPV-associated anogenital warts have been reported in children: 0% in non-abused prepubertal children,<sup>13</sup> 0.17% of children referred to a tertiary care hospital,<sup>14</sup> and 1.8% in children with suspected sexual abuse.<sup>24</sup> HPV6 and HPV11 are the most common types detected in anogenital warts in children,<sup>25</sup> though cutaneous HPV types have also been detected.<sup>26</sup> A multicenter study in Germany found mucosal HPV types were more common in anogenital wart biopsies from those under 5 years of age, whereas cutaneous HPV types were more common in anogenital wart biopsies from those 5 to 12 years old.<sup>27</sup> In one study of children with anogenital warts, 24% of children had an adult family member with anogenital warts, 62% had a mother with cervical intraepithelial neoplasia (CIN), and 48% had a family member with extragenital warts,<sup>28</sup> suggesting that nonsexual transmission is a possible route of infection. Studies of self-inoculation are lacking in children, but

one adult study of 25 monogamous couples reported evidence of self-inoculation in 11 males and 4 females.<sup>29</sup> In a systematic review, among children with anogenital warts, older age was associated with a higher odds for sexual abuse (odds ratios [ORs] were 6.5–7.5 for children aged 3–4 years, 5–8 years, and 9–12 years, compared with 0–2 years).<sup>30</sup> Also, genital location (i.e., penile or vulvar) was associated with a higher odds (OR 5.93) for sexual abuse compared to the perianal location.

Oral papillomas also have been described in children, as well as sexually active adolescents, and are commonly associated with HPV6 and HPV11. A rarer condition that is also associated with HPV6 and HPV11 is juvenile onset recurrent respiratory papillomatosis (JORRP), which can be life threatening due to the risk of airway obstruction from wart lesions in the respiratory tract.<sup>31,32</sup> JORRP is a chronic condition that is more aggressive compared to adult onset recurrent respiratory papillomatosis (or AORRP), and management is limited to repeated surgical resection and few adjuvant treatment options. As this condition is not reportable, disease epidemiology is challenging to study. Data in the United States published in 1995 estimated an incidence of 4.3 per 100,000 children, compared to 1.8 per 100,000 adults.<sup>33</sup> A more recent systematic review published in 2024 estimated an incidence in the pre-vaccine era that ranges from 0.2 to 2.1 per 100,000 children, and a prevalence rate that ranges from 0.8 to 4.3 per 100,000.<sup>34</sup> Recent studies show a decline in JORRP with increasing HPV vaccine coverage, exemplified by an incidence rate ratio (IRR) of 0.2 between births in 2012 through 2013 compared with births in 2004 through 2005.<sup>35</sup> No data are available on its epidemiology in children with HIV.

Detection of HPV DNA in normal tissue of infants has been documented, suggesting that perinatal transmission also can occur.<sup>36</sup> Rates of HPV DNA detected in newborns vary significantly (0% to 70%), and when found in the infant, HPV type concordance between the mother and infant also is quite variable (<1% to 100%).<sup>37-39</sup> A systematic quantitative review of maternal–neonatal transmission concluded that pooled perinatal HPV transmission was around 6.5%.<sup>37</sup> Several authors have suggested that the rate of HPV detection in infants depends on the rate found in pregnant mothers.<sup>38,40</sup> Risks of DNA detection in newborns include maternal HPV status at delivery and the presence of anogenital lesions (i.e., condyloma or squamous intraepithelial lesions) in the mother.<sup>38,39</sup> Studies have concluded that pregnancy itself, even in the setting of HIV infection, is not associated with increased vulnerability to HPV acquisition.<sup>41</sup> In addition, women with cervical cancer are not likely to transmit HPV to their children.<sup>42</sup> However, women with HIV, in general, are much more likely to have HPV infections.

In one study, 19.7% of infants born to mothers with HPV and 16.9% of infants born to mothers who were HPV negative at delivery were found to be HPV positive in their mouth or anogenital area at some time during a mean follow-up period of 14 months, suggesting that perinatal transmission is not the sole source of oral or genital HPV infection in infants.<sup>38</sup> Although maternal history of condyloma at time of delivery has been a well-described risk factor for appearance of JORRP, the risk remains quite low, with estimates of 7 per 1,000 births with a maternal history of genital warts.<sup>43</sup> In a parent–child study in Finland, the cumulative detection rates for high-risk HPV from the child’s genital and oral samples were 36% and 42%, respectively.<sup>44</sup> However, persistence of HPV was less common, with persistent oral HPV in 10% of infants and persistent genital HPV in 1.5% of infants. A relatively recent systematic review of the prevalence of HPV in pediatric tonsils showed a range from 0% to 21%; the largest study observed no infections.<sup>45</sup> Together, these data show that in the general population oral and genital perinatal transmission can occur, whereas persistence is unusual when infection is acquired (whether through vertical or horizontal transmission).

Even less is known about HPV detection in normal tissue in children with HIV. A study of adolescents with perinatally acquired HIV showed that 30% of girls had an abnormal (atypical SCs of undetermined significance [ASC-US] or greater) Pap test.<sup>46</sup> The mean age at the time of the first Pap test was 16.7 years (range 13–23 years). The observational study also noted that 23 cases of condyloma were reported in those younger than age 13. In a small study of Brazilian infants, maternal HIV was noted to be a risk factor for neonatal transmission.<sup>40</sup> Another study of adolescents with perinatal HIV aged 11 to 16 years in Côte D’Ivoire found an HPV prevalence of 3.6% for all HPV types and a prevalence of 2.8% for oncogenic HPV obtained from vaginal swabs collected by a midwife.<sup>47</sup> Of the 250 participants, 12 (4.8%) reported having had vaginal intercourse. Interestingly, risk for HPV was not associated with vaginal intercourse, whereas the practice of intravaginal cleansing with water only or water plus soap was significantly associated with HPV infection even when accounting for vaginal intercourse. In a small study of 50 girls aged ≤18 years who reported never having sexual contact, 30% of the girls who acquired HIV perinatally had HPV detected from external genital samples compared to 7% of girls who were perinatally exposed but uninfected.<sup>48</sup> HPV DNA also was found to be twice as common in the oral cavity than in the HIV-uninfected control group.<sup>49</sup> Of interest, 21% of women with HIV were found to have HPV, including hr-HPV and low-risk HPV types, detected in breast milk samples.<sup>50</sup> These data together suggest that children with perinatally acquired HIV may be more vulnerable to maternal transmission of HPV because of higher rates of HPV in this group of mothers and higher rates of HPV persistence in the neonatal and infant period due to immunosuppression. Unfortunately, there are no large mother–infant studies of HPV transmission and persistence in women with HIV.

Genital HPV is most commonly a result of sexual transmission. Epidemiology of genital HPV is covered in [Adult and Adolescent Opportunistic Infection Guidelines](#).<sup>2</sup> Rates of HPV are higher in adolescents and adult women with HIV than those without HIV.<sup>51-53</sup> As with HPV, CIN and condyloma also are more common in women with HIV.<sup>54-58</sup>

Although the incidence of anogenital HPV infection in sexually active youth is high, longitudinal studies have demonstrated that 80% to 90% of infections in youth without HIV are transient and spontaneously regress.<sup>59-61</sup> Although anal HPV acquisition is associated with anal intercourse, several studies in both children and adults suggest that other sexual and nonsexual routes of anal acquisition are possible.<sup>60,62,63</sup>

The higher prevalence of HPV infections in populations with HIV may result partly from increased HPV persistence in these patients. In one study of adolescents with HIV, approximately 50% cleared their HPV infections.<sup>57</sup> Detection of anal HPV also is higher in youth with HIV.<sup>62</sup>

Persistent infection with hr-HPV types is associated with increased risk of CIN and cervical and vulvovaginal carcinoma in women and of anal intraepithelial neoplasia (AIN) and anal carcinoma in women and men. Rates of HPV-associated cancers—including cervical, vulvar, vaginal, penile, anal (men and women), and oropharyngeal—are higher in individuals with HIV<sup>64-66</sup> and believed to result predominantly from the increased risk of persistent infection in this group. For adolescents, refer to the [Human Papillomavirus Disease section of the Adult and Adolescent Opportunistic Infection Guidelines](#).

Although combination antiretroviral therapy (ART) has dramatically altered HIV’s natural history, its impact on HPV and HPV-associated neoplasia is less clear. Several studies have shown that HPV prevalence and rates of CIN and AIN have not been reduced with ART,<sup>67-69</sup> whereas cervical cancer rates have decreased in most racial and ethnic groups. In contrast, anal cancer rates have increased in

individuals with HIV.<sup>70</sup> The increase in cervical precancer and decrease in cervical cancer is thought to be due to increased cervical cancer screening, leading to earlier detection and treatment of precancers and therefore lower rate of cancers.

Other risks associated with of cervical cancer include lack of cervical cancer screening, prolonged use of hormonal contraception, parity, smoking, and immunocompromising conditions other than HIV.<sup>71</sup>

## Clinical Manifestations

### ***Genital, Anal, Skin, Oral, and Respiratory Tract Warts***

Genital HPV types cause hyperplastic, papillomatous, and verrucous squamous epithelial lesions (warts) on skin and mucus membranes, including anal, genital, oral, nasal, conjunctiva, gastrointestinal, bladder, and respiratory tract mucosa. Lesions in the genital area are often referred to as condyloma acuminata. Warts can be single or present with multiple lesions and often appear as papules or flat, smooth, or pedunculated lesions. Common sites for skin warts are the hand, elbows, knees, and feet. Another manifestation of warts in the respiratory tract is JORRP, which can present with hoarseness and difficulty breathing.

### ***Precancerous and Cancerous Lesions***

Genital lesions associated with HPV include high-grade CIN, vulvar intraepithelial neoplasia (VIN), vaginal intraepithelial neoplasia (VAIN), and AIN. Most intraepithelial neoplasia are asymptomatic. Cancers associated with hr-HPV types include cervical, vulvar, vaginal, penile, anal, and oropharyngeal, specifically at the base of the tongue and tonsils. Cancers often are asymptomatic but also can be associated with bleeding, pain, or a palpable mass.

## Diagnosis

### ***Genital, Anal, Oral, and Skin Warts***

Most cutaneous and anogenital warts can be diagnosed by visual inspection.<sup>10</sup> In some instances, anogenital warts can raise suspicion for sexual abuse. If sexual abuse is suspected, referring to clinics specializing in providing abuse evaluations should be considered.<sup>10</sup> Speculum examination is not recommended for prepubertal children in the office setting. If intravaginal lesions from HPV or sexual abuse are suspected, appropriate referral to an expert in pediatric gynecology and in sexual abuse is recommended as vaginotomy may be required and should be performed under anesthesia. [Anoscopy or a digital rectal examination](#) is not routinely indicated. If the lesions do not respond to standard therapy or the warts are pigmented, indurated, fixed, or ulcerated, biopsy may be needed.

Patients in whom cancer or JORRP is suspected should be referred to an expert for diagnosis and management.

### ***Intraepithelial and Squamous Cell Cancers***

Cytology is not recommended in non–sexually active prepubescent children with HIV nor in those who have been sexually abused. However, if intraepithelial or SC cancers are suspected, the same cytology and colposcopic techniques used to detect CIN in people without HIV should be used in

people with HIV. Cytology is a screening test for cervical cancer (see the Prevention section below). However, histology remains the gold standard for confirming CIN and invasive cancers. In sexually active individuals, the entire genitalia and anal canal should be inspected carefully for visual signs of warts, intraepithelial neoplasia, or invasive cancers. Vaginal, vulvar, anal, and oral cancers often can be palpated by digital examination of the vaginal, vulvar, intra-anal, and oral pharyngeal regions. Definitive diagnoses are made by histology. CIN, AIN, VAIN, and VIN can often be visualized using colposcopy and high-resolution anoscopy, but definitive diagnosis is made by biopsy.

## Role of Human Papillomavirus DNA/RNA Testing

HPV testing is not helpful in diagnosing or managing visible genital, skin, or oral warts. HPV testing is not recommended in any circumstance for adolescent girls or boys (aged <20 years), regardless of whether they have HIV, because of the high rates of HPV infection.<sup>2</sup> High rates of HPV infection do not implicate disease necessarily as most HPV infections will spontaneously regress, even among those with HIV. HPV testing is currently not recommended in the case of sexual abuse.

## Prevention Recommendations

### *Preventing Exposure*

If sexually active, individuals with HIV should use latex or polyurethane condoms during every act of vaginal, anal, and oral sexual intercourse to reduce the risk of exposure to (or transmission of) sexually transmitted pathogens (**AII**). Efficacy of condoms is covered in the [Human Papillomavirus Disease section of the Adult and Adolescent Opportunistic Infection Guidelines](#).

### *Human Papillomavirus Vaccine*

The bivalent, quadrivalent, and nonavalent vaccines have been shown to prevent HPV16 and HPV18 infections and associated precancers in females. The quadrivalent vaccine has been shown to prevent HPV16 and HPV18 infections and precancers in males, and immunobridging studies for the nonavalent vaccine suggest equal protection. The quadrivalent and nonavalent vaccines also protect against HPV6 and HPV11 infections and associated genital warts in females and males; the nonavalent vaccine also protects against five other oncogenic HPV types (31, 33, 45, 52, 58).<sup>72-75</sup> After 2016, only the nonavalent vaccine has been available in the United States. Because the HPV vaccine prevents infection and is not therapeutic, it ideally should be administered before potential exposure to HPV through sexual contact (**AIII**). Studies of HPV vaccine in adults with HIV have shown little to no therapeutic efficacy.<sup>73,76,77</sup> There are no randomized clinical trials in those with HIV in the age group 13 to 26 years of age.

The nonavalent HPV vaccine is recommended for routine vaccination of females and males with HIV aged 9 to 12 years (**AIII**) and for those aged 13 to 26 who have not been previously vaccinated or have not completed the vaccine series (**AIII**) (see the [Use of 9-Valent Human Papillomavirus \[HPV\] Vaccine: Updated HPV Vaccination Recommendations of the Advisory Committee on Immunization Practices](#)). A three-dose series is recommended regardless of the age when the vaccination was started. The second dose should be administered 1 to 2 months after the first dose, and the third dose should be administered 6 months after the first dose.

If the vaccination schedule is interrupted, the series can be continued and does not need to be restarted. The nonavalent HPV vaccine may be used to complete a series started with the

quadrivalent or bivalent vaccines. Currently, for those with HIV, three doses are recommended at any age, which is in contrast to the general population, for whom two doses are recommended for those younger than age 15 years.<sup>10</sup> A three-dose series of the HPV vaccine is recommended starting at the age of 9 years<sup>78</sup> in those with a history of sexual abuse. For those who have completed a vaccination series with the recombinant bivalent or quadrivalent vaccine, many experts would give an additional full series (three doses) of vaccination with recombinant nonavalent vaccine, but no data exist to define who might benefit or how cost effective this approach might be **(CIII)**. The additional five hr-HPV types covered by the nonavalent vaccine were found in 4.2% to 18.3% of HPV-associated cancers in men and women in the United States, depending on the cancer's location.<sup>79</sup> If the person had severe immunosuppression at the time of initial HPV vaccination, re-immunizing with the HPV vaccine should be considered once immune reconstitution is achieved.

## **Human Papillomavirus Vaccine Efficacy**

HPV vaccine studies in immunocompetent children and adults show excellent immunogenicity, efficacy, and safety.<sup>10</sup> Based on clinical trials and immunobridging studies, the Advisory Committee on Immunization Practices recommends routine vaccination for 11 to 12 year olds with a statement that the HPV vaccination series can be administered starting at 9 years of age.<sup>80</sup> The American Academy of Pediatrics now recommends starting the vaccine series anytime between ages 9 and 12, in consideration of the best timing for family acceptance and completion of the vaccine series.<sup>10</sup> This was based on retrospective observational data that showed that initiation of the vaccine at ages 9 to 10 years was associated with greater on-time completion of the series (by age 15), compared to initiation at ages 11 to 12.<sup>81,82</sup> Although recent evidence suggests that one dose of the HPV vaccine has reasonable protection in healthy people, a single dose is not currently endorsed in the United States at any age<sup>83,84</sup> and is not recommended for those with HIV.<sup>85</sup> HPV vaccine studies in adults with HIV show lower but measurable antibodies<sup>86</sup> compared to adults without HIV. Unfortunately, little data on efficacy were collected in these studies. Few studies of HPV vaccine in children with HIV are available.<sup>87</sup>

To date, studies of children with HIV show similar findings as studies of adults with HIV with lower but measurable antibodies compared with individuals without HIV. In the only randomized clinical trial of the quadrivalent HPV vaccine for children with HIV in the United States, researchers found the vaccine to be safe and immunogenic in children aged 8 to 11 years.<sup>88</sup> Serum antibodies to HPV6 and HPV18 were 30% to 50% lower than in historic age-matched immunocompetent controls. Eighteen months after the third vaccine dose, 94% to 99% had antibodies to HPV6, HPV11, and HPV16; however, only 76% had antibodies to HPV18. This group also was given a fourth dose, which demonstrated an excellent anamnestic response for all the vaccine-associated HPV types.<sup>89</sup> After a follow-up period of 2.5 to 4 years, antibody titers were higher in the four-dose group, although seropositivity rates were similar to those who had received three doses.<sup>90</sup> Lower antibody titers were correlated with lower CD4 T lymphocyte (CD4) percentage and with higher CD8 percentage and HIV RNA. Most other non-randomized vaccination studies show similar or slightly lower seroconversion rates in children with HIV compared with children without HIV, with decreasing seropositivity over time, particularly for anti-HPV18 titers; in one study, only 67% were positive at 24 months postvaccination.<sup>87,91,92</sup> One trial of children with HIV aged 9 to 14 years old in Kenya with three quadrivalent HPV doses found high seroconversion rates 12 months postvaccination; 4 years postvaccination, seropositivity rates were 77% for HPV18, 80% for HPV11, 83% for HPV6, and 90% for HPV16.<sup>87,91</sup> Antibody titers remained higher for those with undetectable versus detectable HIV RNA at time of vaccination. One study has examined a two-dose schedule in children aged 9 to 15 years with good immune reconstitution. The comparison group consisted of youth aged 15 and

older with or without immune reconstitution and children aged 9 to 15 without immune reconstitution who received a three-dose schedule. In this study, girls received the bivalent vaccine whereas boys received the quadrivalent vaccine. The geometric mean titers (GMTs) for HPV16 and 18 were similar 1 to 3 months after the last dose between the two groups for both boys and girls; longer-term data were not available.<sup>93</sup>

In a randomized trial comparing the bivalent and quadrivalent vaccines in 546 young women aged 15 to 25 years (257 with HIV and 289 without HIV), GMT was higher for those without HIV versus those with HIV. Antibody titers peaked at 1 month and plateaued by Month 24. Antibody response was higher for those with perinatally acquired HIV than for those with sexually transmitted HIV.<sup>94</sup>

Few studies have examined efficacy. In a single-arm, open-label study, incident rates of persistent HPV in 279 girls and women with HIV vaccinated with the quadrivalent HPV vaccine were compared to previously published rates for vaccinated women without HIV.<sup>95</sup> In the per protocol efficacy population, after a mean follow-up of 2 years, the rate ratio for persistent HPV for women with HIV versus women without HIV using reported rates was 11.7 (95% confidence interval [CI], 2.6–52.1), demonstrating reduced efficacy in women with HIV. This study had several limitations, including its relevance to children given that most participants were adults.

There has been interest in single-dose HPV vaccination; in 2022, the World Health Organization recommended single-dose vaccination as an option for girls and boys aged 9 to 20 years. This recommendation was not extended to individuals known to be immunocompromised or people with HIV.<sup>85</sup> In an observational study in the United States and Puerto Rico, HPV vaccine–type antibody levels were measured from stored sera in 310 youth with perinatally acquired HIV and 148 youth who were perinatally HIV exposed but without infection. GMTs were lower for all HPV vaccine types in those with perinatally acquired HIV. However, GMTs were similar whether participants received one, two, or three doses. For all four types, younger age, lower HIV viral load at first vaccine dose, and fewer years from last vaccine dose to sample collection were each independently associated with higher GMTs. The cumulative prevalence of abnormal cervical cytology (ASC-US or greater) was almost 60% for girls with perinatally acquired HIV and 4% for those who were perinatally exposed to but did not acquire HIV.<sup>96</sup> Among girls who were sexually active and vaccinated with the quadrivalent HPV vaccine, the IRR for those with perinatally acquired HIV was 5.2 (95% CI, 0.7–41.7) compared with youth who have exposure to but who do not have HIV. When restricted to those who initiated vaccination prior to sexual debut, the IRR was attenuated but those with perinatally acquired HIV continued to be at higher risk (IRR = 3.0; 95% CI, 0.4–25.7). The number of vaccine doses, GMTs, and number of sexual partners were not associated with abnormal cytology. There were marginal associations with low CD4, high viral load, and lack of ART at first vaccination dose. Most recently, a South African study evaluated single-dose bivalent HPV vaccination.<sup>97</sup> This study conducted repeat cross-sectional surveys among adolescent girls aged 17 to 18 years. The vaccine program was available to girls from one school district aged 15 to 16 years old in 2019. The postvaccine survey included those who had been eligible for that vaccine program. The prevaccination survey recruited girls from the same school district aged 17 to 18 years old in 2019 and therefore too old for the vaccination program. HPV16 and HPV18 prevalence was lower in the postvaccination survey versus the prevaccination survey (adjusted prevalence ratio = 0.65; 95% CI, 0.51–0.83), with no difference by HIV status. Ongoing studies will continue to evaluate the efficacy and duration of immune response in youth with HIV.

## ***Preventing Disease***

### **Circumcision**

There is evidence that circumcision in males without HIV reduces the rates of oncogenic HPV infection of the penis<sup>98-103</sup> and is associated with lower risk of penile cancer<sup>104,105</sup> and cervical cancer in sexual partners.<sup>106</sup> The prevalence of hr-HPV has a relatively wide variability, ranging from 0% to 83%.<sup>107,108</sup> Because of the lack of studies in those with HIV and the lack of benefit suggested by other studies,<sup>109</sup> evidence is insufficient to recommend infant or adult male circumcision solely for the purpose of reducing the risk of oncogenic HPV infection in those with HIV or their sex partners in the United States.

### ***Preventing Cervical Cancer***

Women with HIV should have cervical screening cytology (liquid-based or Pap test) when they have reached the age of 21 years, regardless of their sexual history, as per the [Adult and Adolescent Opportunistic Infection Guidelines \(AIII\)](#). Cytology is not recommended in non-sexually active prepubescent children with HIV nor in those who have been sexually abused.<sup>110</sup>

### ***Preventing Vaginal and Vulvar Cancer***

Routine screening for vaginal or vulvar cancer in children with HIV is not recommended.

### ***Preventing Anal Cancer***

Anal cancer screening in children with HIV is not recommended.

## **Treatment Recommendations**

### ***Treating Disease***

#### **Treating HPV-Associated Warts**

Multiple treatment options for HPV-associated skin and external genital lesions are typically offered to adults and adolescents, as no single treatment is universally ideal for all patients or all lesions.<sup>111</sup> It is important to note that no topical treatment is approved by the U.S. Food and Drug Administration for genital warts for those under age 12, because there are few studies of safety and efficacy in children.<sup>112,113</sup> If untreated, anogenital warts can resolve spontaneously, remain unchanged, or increase in size or number. Regression of cutaneous (nongenital) and genital warts occurs in approximately 30% of cases within 6 months and 90% in several years, even without treatment.<sup>10</sup> Thus, monitoring for spontaneous resolution is a reasonable option. Considerations that may prompt treatment for those under age 12 are similar to considerations for older patients, including symptoms (itching, burning, discomfort). They may also be associated with secondary infections or considerable psychosocial distress about the appearance. However, in children, treatment decisions must consider their sensitive skin, low pain tolerance, and potential for psychological distress regarding treatment itself. For instance, repeated applications of uncomfortable topical treatments may be poorly tolerated for some patients. There are no clinical guidelines available for pain management for children receiving treatment for HPV-associated lesions. Clinicians may attempt using common topical anesthetics used in other routine pediatric procedures such as lidocaine

2.5%/prilocaine cream 5% (sold as EMLA), but evidence for efficacy is lacking. In cases of strong indication for treatment and concern for pain and/or psychological distress, sedation could be considered.

Treatment guidelines for anogenital warts are found in the [2021 Centers for Disease Control and Prevention Sexually Transmitted Infections Treatment Guidelines](#) and apply to both populations with HIV and those without HIV.<sup>111</sup> Treatment can induce wart-free periods, but the underlying viral infection can persist, with potential for recurrence, especially for individuals who are immunosuppressed due to HIV. Immunosuppressed patients may have larger or more numerous warts and may not respond as well as immunocompetent individuals to treatment.<sup>111,114-116</sup> Topical treatments may be ineffective in patients with large or extensive lesions.

As stated in the [Human Papillomavirus Disease section of the Adult and Adolescent Opportunistic Infection Guidelines](#), patient-applied or parent-applied treatments include podofilox (0.5%) solution or gel (**BII**), imiquimod (3.75% or 5%) cream (**BII**), and sinecatechins (15%) ointment (**BIII**).<sup>112,113,117-119</sup> Topical podofilox 0.5% is approved for use in adults aged >18 years, and some limited small studies in children have found efficacy rates similar to older patients, with minimal side effects.<sup>117,120</sup> Imiquimod 3.75% or 5% is approved for use in children >12 years, and limited case reports support the safety and efficacy in younger children.<sup>117,121-123</sup> Sinecatechins 15% is approved for use in adults aged >18 years and also has limited case reports for children.<sup>118,119</sup> Because some treatments are contraindicated in pregnancy, women who might be pregnant should be advised on how to avoid exposure while applying them to their child.

For warts for which provider-applied topical treatments might be more appropriate, no data are available on the use of bichloroacetic acid (80% to 90% aqueous solution) in children. Trichloroacetic acid (TCA) is an option; however, limited data exist on its use in children (**BIII**).<sup>112,113,124,125</sup> Because TCA is a powerful acid, great care should be taken to avoid applying an excess amount. If an excess amount is inadvertently applied, immediate steps should be taken (e.g., cover with baking soda, wash with liquid soap, or powder with talc) to neutralize the excess acid to avoid further injury or scarring of the surrounding skin.

Alternative treatments for adults include cidofovir topical gel (1%), intralesional interferon-alfa (IFN- $\alpha$ ), 5-fluorouracil (5-FU)/epinephrine gel implant, or podophyllin resin. Cidofovir gel (1%) is a topical preparation that has been evaluated in a limited number of adults for treatment of anogenital HPV infection. Evaluation of cidofovir for anogenital warts in children is lacking, but a limited number of case reports support the option of cidofovir for recalcitrant cutaneous (nongenital) warts in children (**CIII**).<sup>126-130</sup> The precaution is that topical cidofovir can be absorbed systemically and is known to be nephrotoxic and has been associated with renal failure.<sup>131</sup> Injectable therapy (such as with IFN- $\alpha$  or 5-FU/epinephrine gel implant) has not been evaluated in children and should be offered only by a qualified expert in severe recalcitrant cases because of inconvenient routes of administration, frequent office visits, and a high frequency of systemic adverse effects (**CIII**). Although off-label, intralesional cidofovir with variable formulations has also been used for recalcitrant warts.<sup>132</sup> Podophyllin resin (10% to 25%) should not be used in children, due to risks of neurotoxicity, bone marrow suppression, and liver dysfunction.

Lesions can be removed by cryotherapy or surgery (**BIII**). Cryotherapy (application of liquid nitrogen or cryoprobe) must be applied until each lesion is thoroughly frozen. Treatment can be repeated every 1 to 2 weeks, up to four times. Lesions can be removed surgically by tangential scissor, tangential shave excision, curettage, or electrosurgery.

Limited data are available on treatment of oral warts in people with HIV. Limited lesions can be treated with provider-applied therapies, such as TCA or surgical excision. Extensive lesions should be referred to an expert.<sup>135</sup>

Laryngeal papillomatosis generally requires referral to a pediatric otolaryngologist. Treatment is directed at maintaining the airway, rather than removing all disease.

### **Treating Histologically Confirmed CIN, VIN, VAIN, AIN, and Anogenital Cancers**

For adolescents with HIV who have CIN, VIN, VAIN, AIN, or anogenital cancers, refer to the [Human Papillomavirus Disease section of the Adult and Adolescent Opportunistic Infection Guidelines](#).

### **Role of Antiretroviral Therapy**

Severe immunosuppression is associated with greater HPV-associated morbidity and mortality and lower HPV vaccine immunogenicity and efficacy. Maintaining low viral load and a normal CD4 cell count is recommended. HPV vaccine should be administered at the time of optimal immune health. Depending on the circumstances, it may be best not to defer awaiting immune reconstitution; rather, re-immunize once immune reconstitution is reached.

### ***Monitoring of Adverse Events (Including IRIS)***

Monitoring for toxicity and recurrences is required during and after treatment of genital warts. The major toxicity of podofilox, imiquimod, and sinecatechins ointment is inflammation at the application site. The major toxicity of cryotherapy is local pain and skin irritation, which can be tolerable for adults but challenging for children and should be discussed with caregivers. Adequate local pain management is essential for all caustic treatments. Topical anesthetics are favored, but use of such anesthetics may not be sufficient, and traditional distraction techniques would be another concurrent approach in children.<sup>133,134</sup> The major toxicities of surgical treatment for genital warts are local pain, bleeding, and secondary infection. The major toxicities associated with acid cauterization are local pain and irritation or ulceration of adjacent normal skin. Topical cidofovir can be absorbed systemically and is known to be nephrotoxic and has been associated with renal failure.<sup>131</sup> Intralesional IFN- $\alpha$  can be associated with systemic toxicities of IFN- $\alpha$ , including fever, fatigue, myalgia, malaise, depression, and other influenza-like symptoms. Scarring can occur with any of the above treatment modalities. Topical cidofovir may result in systemic absorption and can be associated with renal toxicity.<sup>131</sup>

Secondary infections are not uncommon if ulcerations occur, and close monitoring post-treatment for treatment-related toxicity is warranted. Treatment of CIN with ablative and excisional modalities can be associated with several adverse events such as pain and discomfort, intraoperative hemorrhage, postoperative hemorrhage, infection, and cervical stenosis. Treatment of AIN is associated with adverse events, including ulcerations, abscesses, fissures, and fistulas.

An immune reconstitution-like syndrome related to HPV-associated oral warts in adults with HIV has been observed in which occurrence of oral warts was associated with decreased HIV RNA levels with ART.<sup>136</sup> Immune reconstitution in response to viral load reduction may result in a return of marked inflammatory responses against latent oral HPV infection. Some studies,<sup>136,137</sup> but not others,<sup>138</sup> have reported an increase in oral warts following ART initiation.

### ***Preventing Recurrence***

Monitoring after therapy for cervical disease should follow the [Human Papillomavirus Disease section of the Adult and Adolescent Opportunistic Infection Guidelines](#). No recommendations exist for preventing recurrence of external genital warts.

### ***Managing Treatment Failure***

Treatment failure is defined as the persistence or recurrence of lesions after appropriate therapy and is more common in those with HIV compared to general population. For persistent or recurrent genital warts, re-treatment with any of the modalities previously described should be considered, preferably with an alternative modality to the one that previously failed (AIII). Genital warts often require more than one course of treatment. Recalcitrant warts should be managed by experienced clinicians and referred for excisional therapy. See the [Human Papillomavirus Disease section of the Adult and Adolescent Opportunistic Infection Guidelines](#) for management of recurrence of anogenital intraepithelial lesions.

### ***Discontinuing Secondary Prophylaxis***

This consideration is not applicable.

## Dosing Recommendations for Prevention and Treatment of Warts Associated With Human Papillomavirus in Children

Indication	First Choice	Alternative*	Comments/Special Issues
Primary Prophylaxis	HPV vaccine	N/A	See <a href="#">Figure 1. Recommended Immunization Schedule for Children With HIV Infection Aged 0 to 18 Years</a> for detailed vaccine recommendations.
Secondary Prophylaxis	N/A	N/A	N/A
Treatment	<p>Monitoring for spontaneous resolution is a reasonable option; 30% resolve spontaneously within 6 months and 90% within several years.</p> <p><b>Patient- or Parent-Applied Treatment Options</b></p> <ul style="list-style-type: none"> <li>Imiquimod (3.75% or 5%) cream applied topically at night and washed off in the morning for 3 nonconsecutive nights a week for up to 16 weeks (BII).</li> <li>Podofilox (0.5%) solution/gel applied topically two times daily for 3 consecutive days a week. Withhold treatment for 4 days and repeat the cycle weekly up to four times (BIII).</li> <li>Sinecatechins (15%) ointment applied three times daily for up to 16 weeks, until warts are cleared completely and not visible (BIII).</li> </ul> <p><b>Provider-Applied Treatment Options</b></p> <ul style="list-style-type: none"> <li>TCA (80% to 90%) applied topically weekly for up to 3 to 6 weeks (BIII).</li> </ul>	<p><b>Patient- or Parent-Applied Treatment Options</b></p> <ul style="list-style-type: none"> <li>Cidofovir topical gel (1%) is an experimental therapy studied in adults with HIV that is commercially available through compounding pharmacies and has very limited use in children; systemic absorption can occur with potential for renal toxicity (CIII).</li> </ul> <p><b>Provider-Applied Treatment Options</b></p> <ul style="list-style-type: none"> <li>Intralesional IFN-<math>\alpha</math> and 5-FU/epinephrine gel implant are generally not recommended because of high cost, difficult administration, potential for systemic side effects, and lack of testing in children (CIII).</li> </ul> <p>* These alternative therapies should include consultation with infectious disease and dermatological specialists.</p>	<p>When choosing treatment options, parent and child comfort in application should be considered.</p> <p>Children have a low pain threshold and, generally, sensitive skin.</p> <p>Adequate topical anesthetics to the genital area should be given before caustic modalities are applied. For young children, these approaches are poorly tolerated due to treatment-related and postoperative pain, and as a result may require general anesthesia. Therefore, these should be mainly reserved for children with extensive lesions.</p> <p>Many of these agents are contraindicated in pregnancy and have potential teratogenic effect. When treatment options are considered, the potential for pregnancy should be discussed and proper precautions during pregnancy explained.</p>

	<ul style="list-style-type: none"> <li>• Cryotherapy with liquid nitrogen or cryoprobe applied every 1 to 2 weeks up to four times <b>(BIII)</b>.</li> <li>• Surgical removal either by tangential excision, tangential shave excision, curettage, or electrocautery <b>(BIII)</b>.</li> </ul>		<p>ART has not been consistently associated with reduced risk of HPV-related abnormalities in individuals with HIV.</p> <p>Most treatments for genital warts cannot be used in the oral mucosa; some oral warts can be treated with TCA or surgical excision.</p> <p>Laryngeal papillomatosis generally requires referral to a pediatric otolaryngologist. Treatment is directed at maintaining the airway, rather than removing all disease.</p>
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Key: 5-FU = 5-fluorouracil; ART = antiretroviral therapy; HPV = human papillomavirus; IFN- $\alpha$  = interferon- $\alpha$ ; TCA = trichloroacetic acid

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