ORIGINAL PAPER

Human papillomavirus infection in Polish HIV-exposed, uninfected children and their mothers

Alina Ciach[1], Tomasz Czerniak[1], Elżbieta Teterycz-Bwire[2], Leszek Szenborn[1]

[1]Department of Pediatric Infectious Diseases, Wroclaw Medical University, Wroclaw, Poland
[2]Ginemedica Medical Center, Wroclaw, Poland

Abstract

Introduction: Women living with HIV (WLWH) experience a higher burden of human papillomavirus (HPV) infection compared with the general population. Although immune dysregulation has been demonstrated in HIV-exposed, uninfected (HEU) children, their risk of HPV infection remains unclear. This study assessed the prevalence, genotype distribution, and clinical manifestations of HPV infection among pre-pubertal HEU children and their HIV-positive mothers in Poland.

Material and methods: Thirty HEU children (median age, 17.7 months) and 28 WLWH were examined, along with 24 HIV-unexposed controls. Genital swabs were tested for 35 HPV genotypes by PCR, and HPV 6/11/16/18 antibodies were assessed by ELISA. Mothers also underwent gynecological examination and cervical cytology.

Results: HPV DNA was detected in 12/ 28 mothers (43%), including 11 (39%) with high-risk HPV types and 21% with multiple infections (2-7 genotypes), most frequently HPV 16, 31, 52, and 83. Cytological abnormalities, such as inflammatory changes, LSIL, ASC-US, and ASC-H, occurred in 17%, 25%, 17%, and 8% of HPV-positive women, respectively. HPV DNA was found in 3/30 (10%) of HEU children and none among controls ($p = 0.24$), while HR-HPV accounted for 100% of HEU children infections. Nonavalent vaccine-covered genotypes were revealed in 43% of HPV infections in mothers and 75% in children. All serologic tests were negative.

Conclusions: Women living with HIV showed a high prevalence of HPV, with frequent multiple infections and predominance of high-risk genotypes. HPV DNA was detected in 10% of HIV-exposed, uninfected children. These findings underscore the need for larger longitudinal studies to better define HPV epidemiology in HEU children, and to inform preventive strategies.

HIV AIDS Rev 2026; 25, 1: 28-34
DOI: https://doi.org/10.5114/hivar/218818

Key words: human papillomavirus viruses, HIV, children, women, human papillomavirus vaccines.

Introduction

In women living with HIV (WLWH), an increased prevalence of human papillomavirus (HPV) infection (a higher rate of co-infections with multiple viral sub-types) and a prolonged duration of infection clearance have been demonstrated compared with the healthy population [1, 2]. HPV can be

Address for correspondence: Alina Ciach, Department of Pediatric Infectious Diseases, Wroclaw Medical University, Ludwika Pasteura 1, 50-367 Wroclaw, Poland, e-mail: alakaczmarzyk@gmail.com

Article history:
Received: 26.01.2026
Revised: 03.03.2026
Accepted: 03.03.2026
Available online: 08.03.2026

International Journal of HIV-Related Problems
HIV & AIDS Review

This is an Open Access journal, all articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 International (CC BY-NC-SA 4.0) License (http://creativecommons.org/licenses/by-nc-sa/4.0/)

transmitted from mother to child, with prenatal and perinatal transmission through direct contact with the mucous membranes of the genital tract considered the most common route [3, 4]. Horizontal transmission through caregiving activities during the early postnatal period is also possible [5]. In HIV-exposed, uninfected (HEU) children, immune system dysregulation has been demonstrated, involving both innate and adaptive (T and B cell-mediated) immune mechanisms, along with elevated markers of chronic immune activation and inflammation [6-8]. Consequently, this population has an increased risk of infectious diseases, though identifying specific pathogens with increased infection rates requires further research [6, 8]. The primary endpoint of this study was the prevalence of HPV DNA in HIV-exposed, uninfected children compared with controls. Secondary endpoints included HPV genotype distribution, mother-child genotype concordance, and clinical correlates.

Material and methods

The study included 30 children, patients of the Department of Pediatric Infectious Diseases, Wroclaw, Poland. These children were followed for the evaluation of possible vertical HIV transmission, and all tested negative for maternal HIV infection. Among mothers, 28 HIV-positive women participated (two mothers had two children each), and none of them had been vaccinated against HPV infection. A control group of 24 children, matched for age and sex, was enrolled. Control participants had no risk factors for maternal HIV transmission and no chronic illnesses. The study was conducted between July 2011 and August 2012.

The visit protocol of both groups included: medical history (including clinical symptoms of recurrent respiratory papillomatosis caused by a HPV infection, e.g. hoarseness, cough, stridor, weak cry, or dysphonia), physical examination (including thorough physical examination of the oral cavity, genital and anal area), and collection of biological samples for virological testing using PCR for HPV DNA (swabs from genital areas – labia, vestibule, glans penis, foreskin) and blood samples for serological testing. Mothers underwent gynecological evaluation (performed by a gynecologist): pelvic examination, cervical cytology, vaginal and cervical swabs for virological testing, and blood sampling for serological testing. Samples from mothers and children were obtained on the same day.

Clinical specimens were collected using flocked swabs (FlOQSwabs®, Copan UTM collection system; Italy) with a liquid transport medium. PCR-based virological testing was performed by external commercial REX Laboratory (Wroclaw, Poland) using CLART Human papillomavirus 2 assay (Genomica; Madrid, Spain) to detect 35 HPV subtypes: 6, 11, 16, 18, 26, 31, 33, 35, 39, 40, 42, 43, 44, 45, 51, 52, 53, 54, 56, 58, 59, 61, 62, 66, 68, 70, 71, 72, 73, 81, 82, 83, 84, 85, and 89 [9-11].

Serological testing was done in a research laboratory of the Department of Pediatric Infectious Diseases, Wroclaw, Poland. Antibody levels were assessed using commercial enzyme-linked immunosorbent assay (ELISA) test, based on virus-like particles (VLPs) of HPV types 6, 11, 16, and 18 (EIA-4907, DRG Instruments GmbH; Germany). The assay employs recombinant L1 VLP antigens immobilized on microtiter plates, and was performed according to the manufacturer's instructions. Optical density values were measured spectrophotometrically, and results were interpreted using the manufacturer-defined cut-off for seropositivity. All samples were processed under identical laboratory conditions to minimize intra-assay variability [12, 13].

Proportions were summarized as percentages, with 95% confidence intervals (CIs) calculated using the Wilson score interval. Comparisons of categorical variables between groups were performed using two-sided Fisher's exact test. Statistical significance was defined as a p-value of < 0.05. Genotype-specific mother-child concordance was evaluated across a pre-defined panel of 35 HPV genotypes using observed agreement and Cohen's kappa (κ) coefficient, to account for agreement beyond chance. In addition, any type concordance was defined as the presence of at least one shared HPV genotype between mother and her child, while child-to-mother concordance was defined as the detection of all HPV genotypes identified in the child and its mother. All statistical analyses were performed using R statistical environment (R Foundation for statistical computing; Vienna, Austria).

The study was conducted in accordance with the Declaration of Helsinki of 1975, as revised in 2000, and the protocol was approved by the Bioethics Committee of the Wroclaw Medical University (approval number: KB-439/2007) on September 20, 2007. Informed consent for participation was obtained from all subjects involved in the study.

Results

Study group description

The study group comprised 30 HEU children (13 females and 17 males), aged 1 month to 5 years (median age, 17.7 months), while the 28 HIV-infected mothers were aged between 27 and 42 years (median age, 33 years). The interval between maternal HIV diagnosis and childbirth ranged from 0 to 13 years (median, 4.5 years). The control group included 24 children (13 females and 11 males), aged 6 months to 5 years (median age, 20.1 months).

Most pregnancies were properly managed to prevent vertical HIV transmission. Antiretroviral therapy (ART) was administered in 27 of 30 pregnancies (in 17/27 throughout pregnancy, and in 10/27 during the 2nd and 3rd trimesters). HIV viremia during pregnancy was documented in 19/30 cases, with undetectable viral load in 18/19 (< 40 copies/ml) and > 1,000 copies/ml in one case. Zidovudine was administered intravenously during delivery in all cases (30/30), and all children were delivered via cesarean section (24 elective, 6 emergency).

All mothers reported 100% adherence to infant ART prophylaxis.

The reported risk factors for horizontal HPV transmission included bed-sharing with a parent (4/30, 13.3% of children) and overcrowded living conditions, defined as > 2 persons per room (8/30, 26.7% of children).

HPV in mothers

HPV DNA was detected in cervical swabs in 42.9% of mothers (12/28; 95% CI: 26.5-60.9, calculated using the Wilson score method) (Table 1). Twenty-two different HPV types were identified, including HPV 6, 11, 16, 18, 31, 33, 35, 40, 42, 44, 51, 52, 54, 56, 58, 59, 61, 62, 66, 68, 82, and 83. Multiple infections (2-7 types) were found in 21.4% of women (6/28; 95% CI: 10.2-39.5), with an average of 2.9 genotypes per patient.

High-risk (HR) HPV genotypes were present in 39.3% of mothers (11/28; 95% CI: 23.6-57.6), while only one woman (1/28, 3.6%; 95% CI: 0.6-17.9) was infected exclusively with low-risk (LR) HPV types (HPV 11, 83). The most frequently detected genotypes were HPV 16, 31, 52, and 83. HR-HPV accounted for 60% and LR genotypes for 40% of all infections. Among HIV/HPV-co-infected mothers, 5 of 12 (41.7%; 95% CI: 19.3-68.0) reported current or previous clinical symptoms of HPV infection, and 8 of 12 (66.7%; 95% CI: 39.1-86.2) women had abnormal cytological findings (Table 2).

Among identified infections in mothers, nonavalent vaccine-covered genotypes were found in 43%.

HPV in children

HPV DNA was detected in 10.0% of HEU children (3/30; 95% CI: 3.5-25.6) (Table 1), compared with 0.0% in the control group (0/24; 95% CI: 0.0-13.7); however, the between-group difference did not reach statistical significance (Fisher's exact test, $p = 0.24$).

Genotype-specific concordance was assessed across a pre-defined panel of 35 HPV genotypes in three mother-child pairs. Pooled across all pairs (105 genotype-level observations), the observed agreement was 86.7%, while

Table 1. Summary of demographics, and clinical and cytological findings of all HPV-positive mothers of the study. Children results align with that of their mothers

Mother, age (years)	Mother, clinical signs	Mother, cervical HPV DNA*	Mother, cervical cytology	Child, age	Child, clinical signs	Child, anogenital HPV DNA*
30	Asymptomatic	**31, 33,** 42, **59,** 61	LSIL	53 months old male	Asymptomatic	**16**
35	Asymptomatic	**16,** 42, **52**, 54, **56**	LSIL	40 months old female	Asymptomatic	**52**, **56**
27	Cervical leukoplakia, vaginal warts	**16, 31**, **51**, **58**	LSIL	35 months old female	Asymptomatic	**33**
29	Asymptomatic	**31**	Inflammatory changes	18 months old female	Asymptomatic	N/A
28	Asymptomatic	**66**	ASC-H	4 months old male	Asymptomatic	N/A
41	Asymptomatic	**52**	ASC-US	15 months old male	Asymptomatic	N/A
21	Vaginal and labial warts	**6, 52**, 62, **66**, **82**, 83	Inflammatory changes	1 month old female	Asymptomatic	N/A
27	Asymptomatic	11, 83	Normal	4 months old male	Asymptomatic	N/A
33	Labial warts	40, 44, **58**, 61, 62, **68**, 83	Squamous epithelial cells without atypia, no encocervical cells	11 months old male	Asymptomatic	N/A
34	History of labial and cervical warts removal	**18**	Squamous epithelial cells without atypia, no encocervical cells	42 months old male	Asymptomatic	N/A
34	Asymptomatic	**16**	ASC-US	40 months old female	Asymptomatic	N/A
30	Asymptomatic	35	Normal	1 month old female	Asymptomatic	N/A

**HR-HPV (high-risk HPV) genotypes are marked in bold.*

LSIL – low-grade dysplasia, ASC-US – atypical squamous cells of undetermined significance, ASC-H – atypical squamous cell, cannot exclude high-grade lesion, N/A – not applicable

Table 2. Clinical and cytological findings of HIV/HPV co-infected mothers

Outcome/finding	*n/N*	%	95% CI (Wilson)
Clinical manifestations			
Genital warts	3/12	25.0	8.9-53.2
Leukoplakia of the cervix	1/12	8.3	1.5-35.4
History of genital wart excision	1/12	8.3	1.5-35.4
No clinical signs of HPV infection	7/12	58.3	32.0-80.7
Cytological findings			
Inflammatory changes	2/12	16.7	4.7-44.8
LSIL	3/12	25.0	8.9-53.2
ASC-US	2/12	16.7	4.7-44.8
ASC-H	1/12	8.3	1.5-35.4
Non-diagnostic smear	2/12	16.7	4.7-44.8
Normal cytology	2/12	16.7	4.7-44.8

LSIL – low-grade dysplasia, ASC-US – atypical squamous cells of undetermined significance, ASC-H – atypical squamous cell, cannot exclude high-grade lesion

Table 3. Comparison of HPV-positive and HPV-negative HEU children

	HPV-positive HEU children, *n* = 3	HPV-negative HEU children, *n* = 27
Child		
Age, median	40 months	16 months
Females, *n* (%)	1/3 (33.3%)	12/27 (44.4%)
Living conditions		
Bedsharing, *n* (%)	0/3	4/27 (14.8%)
Overcrowding, *n* (%) (> 2 persons/room)	1/3 (33.3%)	7/27 (25.9%)
Persons/room, average	2.1	2.1
Mother		
Any type of HPV present	3/3 (100%)	9/27 (33.3%)
HR-HPV present	3/3 (100%)	8/27 (29.6%)
Multiple HPV infections present	3/3 (100%)	3/27 (11.1%)

HR-HPV – high-risk HPV, HEU – HIV-exposed, uninfected

Cohen's κ was low (κ = 0.17), consistent with the predominance of concordant negative results. At least one shared HPV genotype (any type concordance) and complete child-to-mother concordance were each observed in one of three pairs (33.3%).

None of the children showed clinical signs of respiratory or genital HPV infection, suggestive all children were asymptomatic carriers. In all three HPV-positive cases, the children slept separately; in one case, the family lived in overcrowded conditions (seven people, two rooms) (Table 3).

Among identified infections in children, nonavalent vaccine-covered genotypes accounted for 75%.

Serological testing

Serological ELISA testing for HPV was negative in all participants, regardless of PCR findings.

Discussion

A high prevalence (43%) and unfavorable profile of HPV infections were found among HIV-positive women; 21% had multiple (2-7) HPV genotypes and 39% carried at least one HR-HPV type. These results are in line with findings from other studies on women living with HIV. A systematic review of data from 2011–2022 involving 10,336 WLWH, predominantly living in Europe and North America, found HPV infection in 37% and HR-HPV in 33.9% of women [1]. Among HR-HPV-positive women, cytological abnormalities, such as ASC-US, LSIL, HSIL, were reported in 3.5%, 9.5%, and 4.2%, respectively. In our study, ASC-US and LSIL rates were higher and comparable with Indian Chinese and American populations of WLWH [14-16]. The most common genotypes (in order: 16, 31, 52, 83) are consistent with those reported by Clifford *et al.* [17] in a 2006 meta-analysis (16, 58, 18, 52, 31, 33). A study by Bardin *et al.* [18] with 834 healthy women in Warsaw, Poland, reported an overall HPV prevalence of 16.6% (24.2% in the 24-35 years age group). High-risk HPV types were detected in 11.3% of participants, while low-risk types were found in 9.8%. Multiple infections occurred in 4.6% of women. Abnormal cytological findings were identified in 4.2% of participants, including ASC-US (1.2%), LSIL (2.6%), and HSIL (0.4%). In our study, the high prevalence of multiple infections (50%) and high-risk genotypes (92%), along with the frequent occurrence of cytological abnormalities (67%) among HIV/HPV co-infected mothers, reinforce the importance of existing cervical cancer and HPV infections screening guidelines for women living with HIV [19, 20].

Although the difference in HPV DNA prevalence between HEU children and controls did not reach statistical significance, the point estimate (10.0%) and its wide 95% CI indicate that HPV exposure in this population cannot be excluded, and warrants further investigation in larger cohorts. Data on HPV prevalence in HEU children are scarce. Moscicki *et al.* [21] found HPV in 5/51 (9.8%) anogenital and 1/52 (1.9%) oral samples among 52 HEU children living in USA (mean age, 6.2 ± 4.8 years), identifying both high-risk (HR) (16, 52) and low-risk (LR) (6, 42, 84) genotypes. Another study examining 125 HEU adolescents from USA (median age, 14.5 years), detected HPV DNA in 1.6% of oral samples, with no difference in prevalence in comparison with children living with HIV [22]. Coker *et al.* [23] compared HPV prevalence in oral samples of HIV-infected and uninfected mothers from Nigeria (HR: 17% vs. 8%; LR: 31% vs. 25%), along with their children (mean age, 10 years). Overall HPV prevalence was highest in HIV-infected children, followed by HIV-unexposed and HEU groups. However, in terms of HR-HPV, exposed but uninfected (HEU) participants showed higher prevalence (33%) compared with HIV-infected (21%) and unexposed (8% of HPV DNA-positive findings). Among HEU children, HPV-16 was the most frequent genotype. This study demonstrated a strong agreement in HPV detection across mother-child pairs. In general, these findings suggest that HPV exposure in early childhood among HEU children may not be uncommon, although estimates remain unclear due to small sample sizes.

The detection of HPV DNA in HEU children and the partial genotype concordance observed in mother-child pairs are compatible with both vertical and early horizontal transmissions. However, the study design does not allow for a definitive distinction between these routes. All children were delivered by cesarean section, and several were older than infants at the time of sampling, making postnatal horizontal exposure plausible. A 2005 meta-analysis by Medeiros *et al.* [24] estimated vertical HPV transmission risk rate of 6.5%. Similar rates were reported in a recent HERITAGE cohort study (7.2%) [25]. Maternal-child genotype concordance was 85% at birth and 33% after 6-24 months, with mean infection duration of 3.9 months and horizontal infection incidence of 9.6% between 6 and 24 months [26]. The persistence of HPV infections in healthy children was documented in several other studies, ranging from 6 weeks to 26 months, depending on study follow-up period, and correlated with maternal HR-HPV persistence [5]. The incomplete concordance between genotypes detected in mothers and their children observed in our study and other reports, may be attributed to the elimination of the virus from mucosal surfaces prior to sample collection or to a latent state with periodic HPV reactivation [5]. Higher HPV DNA detection rates have been reported in placental and trophoblastic tissue of HIV-infected women compared with healthy women [27]. However, data on vertical HPV transmission, specifically among WLWH and their children, remain insufficient. The persistence duration and early-life horizontal transmission rates of HPV among HEU children are also unknown. At present, given the limited number of available studies and the lack of long-term follow-up, the clinical significance of HPV infections in prepubertal period remains unclear [28]. Although cellular immune alterations have been reported in HEU children, an increased incidence of disease manifestations – such as premalignant or malignant lesions – has not been demonstrated in HEU children compared with healthy children to date [6, 8, 29].

HPV vaccination has been shown to be more effective when administered to younger children prior to HPV exposure associated with sexual debut [30, 31]. This raises the question of whether broader HPV exposure among HEU children in early childhood could translate into reduced vaccine effectiveness when these individuals are vaccinated in adolescence or adulthood. However, to our knowledge, there are currently no studies directly evaluating HPV vaccine effectiveness specifically in HEU children compared with healthy peers.

Although serological testing may provide useful insights into cumulative HPV exposure at the population level, its value remains limited due to low seroconversion rates and methodological variability across research [32]. In the current study, serological testing for HPV was negative in all participants, irrespective of HPV DNA status. This finding was expected, since previous studies demonstrated low seroconversion rates following natural HPV infection, particularly among women living with HIV [33]. Moreover, ELISA assay used in the study targets antibodies against only four HPV genotypes (6, 11, 16, and 18), limiting its ability to capture immune responses to other circulating types. Also, other studies reported relatively low sensitivity of HPV VLPs ELISA tests [13], further supporting the limited utility of HPV serology for individual-level diagnosis or exposure assessment in this population.

The high prevalence and unfavorable profile of HPV infections among women living with HIV (WLWH), characterized by multiple co-infections and predominance of high-risk (HR) HPV genotypes, contribute to the substantial HPV exposure of their youngsters. In these children, vertical and horizontal transmissions in early life represent the main sources of HPV infection during the pre-pubertal period.

The limited number of studies investigating HPV infections in pre-pubertal HEU children, which differ in methodology (e.g., sampling from anogenital or oral sites) and geographic origin of participants, along with the considerable variability of reported findings and high proportion of HR-HPV genotypes (100% in the present study), underscores the need for further standardized research in this field.

Potential strategies to address the increased HPV exposure among HEU and HIV-infected children, may include extending HPV vaccination programs to younger age groups, and evaluating the inclusion of additional HPV genotypes in vaccines based on epidemiological data. However, the most effective and currently feasible approach to protecting children remains the vaccination of WLWH. In a report by

Krankowska *et al*. [1], based on a substantial WLWH cohort, the HPV vaccination rate was found to be extremely low (5.6%). Furthermore, the overall HPV vaccination coverage among Polish adolescents (17.04% as of February 2026) remains below the target level [34]. As demonstrated in our study, the high proportion of vaccine-covered genotypes emphasizes the potential benefits of vaccination for both mothers and their children. The indirect benefit of reducing children's HPV exposure through maternal vaccination, provides an additional argument in favor of promoting HPV immunization among women living with HIV.

A major strength of this study is its focus on a largely unexplored topic, i.e., HPV infections in pre-pubertal children exposed to, but uninfected with HIV, while also considering the prevalence and genotype profile of HPV infections in their mothers. The primary limitation of the study is the small sample size, which reduced the statistical power of the findings and limited the ability to analyze potential risk factors for HPV infection. Another limitation pertains to the cross-sectional nature of the study is based on a one-time HPV DNA testing and applied PCR method, which detects the presence of HPV DNA regardless of the virus's replicative activity. and only genital area swab collection, which may present limited sensitivity comparing to multiple sites swabs collection. A longitudinal follow-up with repeated HPV DNA testing at predefined time intervals, conducted in a larger cohort stratified by age groups and incorporating swabs from multiple anatomical sites (including both the oral cavity and the anogenital region), would allow for a more accurate assessment of the sources, prevalence, and persistence of HPV infections among HEU children. Concurrent HPV testing of the children's fathers could also provide valuable insights for interpreting HPV risk among HEU children.

Conclusions

In the studied group of women living with HIV, a high prevalence and unfavorable profile of HPV infections were observed, characterized by multiple infections and the predominance of genotypes with high oncogenic potential, supporting the existing cervical cancer and HPV infections screening guidelines for women living with HIV.

Among HIV-exposed, uninfected (HEU) children, HPV infection was detected in 10% of cases, with high-risk types predominating; although our study design did not allow for a definitive determination of the route of transmission.

These findings highlight the need for larger, longitudinal studies to better characterize HPV epidemiology in HEU children and to inform preventive strategies, including vaccination policies.

Disclosures

1. Institutional review board statement: This study was approved by the Bioethics Committee of Wroclaw Medical University (approval number: KB-439/2007) on September 20, 2007.
2. Assistance with the article: None.
3. Financial support and sponsorship: This research was funded by Wroclaw Medical University, grants number 1758 and ST-880/2014-2016. The APC was funded by Wroclaw Medical University, grant number SUBZ. C180.25.096.
4. Conflicts of interest: None.

References

1. Krankowska D, Mazzitelli M, Ucak HA, Orviz E, Karakoc HN, Mortimer H, et al. Screening and prevention of HPV-related anogenital cancers in women living with HIV in Europe: results from a systematic review. HIV Med 2024; 25: 769-793.
2. Wielgos AA, Pietrzak B. Human papilloma virus-related premalignant and malignant lesions of the cervix and anogenital tract in immunocompromised women. Ginekol Pol 2020; 91: 32-37.
3. Freitas AC, Mariz FC, Silva MAR, Jesus ALS. Human papillomavirus vertical transmission: review of current data. Clin Infect Dis 2013; 56: 1451-1456.
4. Park H, Lee SW, Lee IH, Ryu HM, Cho AR, Kang YS, et al. Rate of vertical transmission of human papillomavirus from mothers to infants: relationship between infection rate and mode of delivery. Virol J 2012; 9: 80. DOI: 10.1186/1743-422X-9-80
5. LaCour DE, Trimble C. Human papillomavirus in infants: transmission, prevalence, and persistence. J Pediatr Adolesc Gynecol 2012; 25: 93-97.
6. Salvi M, Fioretti B, Alberti M, Scarvaglieri I, Arsuffi S, Tiecco G, et al. Understanding HIV-exposed uninfected children: a narrative review. Viruses 2025; 17: 442. DOI: 10.3390/v17030442.
7. Afran L, Garcia Knight M, Nduati E, Urban BC, Heyderman RS, Rowland-Jones SL. HIV-exposed uninfected children: a growing population with a vulnerable immune system? Clin Exp Immunol 2014; 176: 11-22.
8. Wedderburn CJ, Evans C, Abrams EJ, Bamford A, Bekker A, Bunders MJ, et al. Research gaps for children who are HIV-exposed but uninfected: outcomes of a research prioritisation workshop. Lancet HIV 2025; 12: e802-e810. DOI: 10.1016/S2352-3018(25)00166-3.
9. Bonde J, Rebolj M, Ejegod DM, Preisler S, Lynge E, Rygaard C. HPV prevalence and genotype distribution in a population-based split-sample study of well-screened women using CLART HPV2 Human Papillomavirus genotype microarray system. BMC Infect Dis 2014; 14: 413. DOI: 10.1186/1471-2334-14-413.
10. Pista A, Verdasca N, Oliveira A. Clinical performance of the CLART human papillomavirus 2 assay compared with the hybrid capture 2 test. J Med Virol 2011; 83: 272-276.
11. CLART HPV 2 – CLART HPV 2-Amplification – Genomica [Internet]. Available at: https://genomica.com/clart-hpv-2-clart-hpv-2-amplification/.
12. Microsoft Word – EIA-4907_HPV IgG_2011-10-27_e [Internet]. Available from: https://search.cosmobio.co.jp/cosmo_search_p/search_gate2/docs/DRG_/EIA4907.20130618.pdf (Accessed: 12.01.2026).
13. Lupi S, Bergamini M, Guidi E, Gregorio P. Cross-sectional seroprevalence of antibodies against 6, 11, 16 and 18 human papilloma virus (HPV) types among teenagers and young women in Italy. Ann Ist Super Sanita 2014; 50: 171-177.
14. Husainy FA, Jose T, Kaur EJ, Singh S, Nair VG. Impact of antiretroviral therapy on cervical pre-cancerous lesions: a prospective cohort study from a tertiary care institute in India. HIV AIDS Rev 2025; 24: 201-205.
15. Wang Q, Ma X, Zhang X, Ong JJ, Jing J, Zhang L, et al. Human papillomavirus infection and associated factors for cervical intra-

epithelial neoplasia in women living with HIV in China: a cross-sectional study. Sex Transm Infect 2019; 95: 140-144.
16. Ortiz AP, Tamayo V, Scorsone A, Soto-Salgado M, Febo I, Piovanetti P, et al. Prevalence and correlates of cervical HPV infection in a clinic-based sample of HIV-positive Hispanic women. Papillomavirus Res 2017; 4: 39-44.
17. Clifford GM, Gonçalves MA, Franceschi S; HPV and HIV Study Group. Human papillomavirus types among women infected with HIV: a meta-analysis. AIDS 2006; 20: 2337-2344.
18. Bardin A, Vaccarella S, Clifford GM, Lissowska J, Rekosz M, Bobkiewicz P, et al. Human papillomavirus infection in women with and without cervical cancer in Warsaw, Poland. Eur J Cancer 2008; 44: 557-564.
19. EACS. Cancer: Screening Methods [Internet]. Available from: https://eacs.sanfordguide.com/en/eacs-part2/cancer/cancer-screening-methods (Accessed: 11.11.2025).
20. Human Papillomavirus Disease: Adult and Adolescent OIs | NIH [Internet]. 2024. Available from: https://clinicalinfo.hiv.gov/en/guidelines/hiv-clinical-guidelines-adult-and-adolescent-opportunistic-infections/human (Accessed: 11.11.2025).
21. Moscicki AB, Puga A, Farhat S, Ma Y. Human papillomavirus infections in nonsexually active perinatally HIV infected children. AIDS Patient Care STDs 2014; 28: 66-70.
22. Moscicki AB, Farhat S, Yao TJ, Ryder MI, Russell JS, Van Dyke RB, et al. Oral human papillomavirus in youth from the pediatric HIV/AIDS cohort study. Sexual Trans Dis 2016; 43: 498-500.
23. Coker MO, Schlecht NF, Osagie E, Akhigbe P, Obuekwe ON, Bromberg Y, et al. Baseline prevalence of oral human papillomavirus in mother-child pairs with and without HIV infection. JAMA Netw Open 2024; 7: e2451512. DOI: 10.1001/jamanetworkopen.2024.51512.
24. Medeiros LR, Ethur AB, Hilgert JB, Zanini RR, Berwanger O, Bozzetti MC, et al. Vertical transmission of the human papillomavirus: a systematic quantitative review. Cad Saude Publica 2005; 21: 1006-1015.
25. Khayargoli P, Niyibizi J, Mayrand MH, Audibert F, Monnier P, Brassard P, et al. Human papillomavirus transmission and persistence in pregnant women and neonates. JAMA Pediatr 2023; 177: 684-692.
26. Bénard EA, Carceller AM, Mayrand MH, Lacroix J, Niyibizi J, Laporte L, et al. Human papillomavirus persistence, recurrence, and incidence in early childhood. J Infect Dis 2025; 232: e213-e222. DOI: 10.1093/infdis/jiaf213.
27. Chisanga C, Eggert D, Mitchell CD, Wood C, Angeletti PC. Evidence for placental HPV infection in both HIV positive and negative women. J Cancer Ther 2015; 6: 1276-1289.
28. Tomecka P, Karwowska A, Kuźnicki J, Skinderowicz K, Wojno A, Markut K, et al. HPV infection in children and adolescents – a comprehensive review. J Clin Med 2025; 14: 2425. DOI: 10.3390/jcm14072425.
29. Abu-Raya B, Kollmann TR, Marchant A, MacGillivray DM. The immune system of HIV-exposed uninfected infants. Front Immunol 2016; 7: 383. DOI: 10.3389/fimmu.2016.00383.
30. Ellingson MK, Sheikha H, Nyhan K, Oliveira CR, Niccolai LM. Human papillomavirus vaccine effectiveness by age at vaccination: a systematic review. Hum Vaccin Immunother 2023; 19: 2239085. DOI: 10.1080/21645515.2023.2239085.
31. Rostami Varnousfaderani M, Khoshnazar Z, Zeratie H, Hosseini Koukamari P. Optimizing HPV vaccine effectiveness: impact of vaccination age and dose schedule on immunogenicity and cervical cancer prevention. Front Public Health 2025; 13: 1544220. DOI: 10.3389/fpubh.2025.1544220.
32. Schiffman M, Safaeian M, Wentzensen N. The use of human papillomavirus seroepidemiology to inform vaccine policy. Sex Transm Dis 2009; 36: 675-679.
33. Kelly H, Faust H, Chikandiwa A, Ngou J, Weiss HA, Segondy M, et al. Human papillomavirus serology among women living with HIV: type-specific seroprevalence, seroconversion, and risk of cervical reinfection. J Infect Dis 2018; 218: 927-936.
34. Power BI Report [Internet]. Available from: https://app.powerbi.com/view?r=eyJrIjoiNzBlZmZlYmItYzg0Ny00YWM1LWEyMjctNWZmZDU5NjFhNTc1IiwidCI6IjU1NjBmMjQ2LTZjMTktNDgzMS1iNTllLWExZGI4NDc0NjI3ZSIsImMiOjl9 (Accessed: 02.03.2026).