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Monitoring the presence of human papillomavirus in public areas indicates non-sexual transmission of the virus and environmental risk

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Abstract

Background In light of the potential for non-sexual transmission of human papillomavirus (HPV), this study aims to investigate the presence of α-HPV in public sanitary environments to explore the possibility of environmental transmission.

Methods Over a 30-day period, environmental samples were collected from three distinct areas across four locations, chosen based on their potential infection risk. The samples were analyzed for 23 HPV genotypes using real-time PCR.

Results A total of 360 environmental samples were collected during 30 days, with 87 samples testing positive for HPV (24.17%, 87/360). The positivity rates at the four locations—public restrooms, community health centers, obstetrics and gynecology hospitals, and general hospitals—were 13.33%, 14.44%, 30.00%, and 38.89%, respectively. Among the different sampling areas, door handles, washbasins, and squat toilets showed positive rates of 5.00%, 14.17%, and 53.33%, respectively. Of the HPV-positive samples, 53.8% exhibited moderate to high viral concentrations as defined by this study. A total of 19 HPV genotypes were detected, with high-risk types accounting for 88.46% and low-risk types for 11.54%. The five most frequently detected genotypes were 51, 16, 52, 58, and 56.

Conclusion HPV was detected in all four locations, with a more pronounced positivity rate found in general hospitals and obstetrics and gynecology hospitals. Squat toilets exhibited the highest positivity rate among sampling areas. High-risk HPV types were significantly more prevalent than low-risk types, and positive rates were higher in the high and medium concentration groups compared to the low concentration group. Notably, high-concentration HPV residues can persist for up to 7 h. These findings suggest that public restroom may serve as potential sources of HPV transmission, with shared public areas such as door handles, washbasins, and squat toilets representing possible routes for non-sexual transmission of the virus.

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Keywords Human papillomavirus, Environmental pollution, Non-sexual transmission, Genotype

Introduction

Cervical cancer is the fourth most common malignancy worldwide and the fourth leading cause of cancer-related deaths among women [1]. In 2022, approximately 660,000 new cases were reported, with over 94% of deaths occurring in low- and middle-income countries [2]. Patients with advanced cervical cancer generally have poor prognosis [3, 4]. The major risk factors for cervical cancer include persistent infection with high-risk HPV, smoking, multiple sexual partners, younger age at first intercourse, and prolonged use of oral contraceptives [5, 6]. Notably, approximately 95% of cervical cancers worldwide are caused by human papillomavirus (HPV) [7]. Most cases of cervical cancer can be prevented through HPV vaccination, as well as the screening and treatment of precancerous lesions caused by early HPV infections [8]. However, by the end of 2023, the global coverage rate for girls receiving the first dose of the HPV vaccine was only 27% [9], highlighting the ongoing importance of HPV infection prevention at present.

HPV is the primary pathogenic factor in the development of cervical cancer and is mainly transmitted through sexual contact. In recent years, non-sexual transmission routes, such as vertical transmission from mother to child, hand transmission between individuals, autoinoculation, and transmission through contaminated surfaces, have been increasingly reported [10]. For instance, a study involving 124 HPV-positive children found that anal-genital and oropharyngeal HPV infections in prepubescent children may be linked to non-sexual transmission during the perinatal or postpartum periods [11]. Additionally, research revealed a 12% prevalence of subclinical high-risk HPV infections in the foreskin of boys under 15 years old with no history of sexual activity [12]. Furthermore, a case report documented a 9-year-old premenarchal girl diagnosed with genital warts despite denying any history of sexual contact, including sexual abuse [13]. These findings present new challenges for the study and prevention of papillomavirus transmission. Given the detection of HPV positivity in individuals who have never engaged in sexual activity, it is essential to explore the potential causes of these infections. This study aims to assess the positivity rate and genotype distribution of HPV in female restroom environments across various public areas, thereby investigating the possible non-sexual transmission routes of HPV in public spaces.

Materials and methods

Sample collection

Based on the potential for non-sexual transmission routes of HPV infection, this study selected female restrooms in four types of locations as research subjects: general hospitals, obstetrics and gynecology hospitals, community health centers, and public restrooms. Sampling was conducted on 30 weekdays from May 15 to July 1, 2024. At 15:00 each weekday, swab samples were collected from three specific areas—door handles, washbasins, and squat toilets—in the fixed restrooms of the aforementioned locations (Fig. 1), resulting in a total of 360 samples (12 samples per day × 30 days). Additionally, to monitor the effective duration of viral persistence, we selected one weekday and the area with the highest HPV positivity rate for hourly environmental sampling, collecting an additional 36 swab samples for independent analysis.

Instruments and reagents

We used the following instruments and reagents for sample collection and analysis: Sterile sampling swab (Lot: 20240428, Hangzhou Aian Biotechnology Co., Ltd.); Nucleic acid extraction and purification kit (Lot: AS001663, magnetic bead method, Fosun Diagnostics (Shanghai) Co., Ltd.); Human papillomavirus nucleic acid typing test kit (Lot: 24004-2, PCR-fluorescent probe method, Shengxiang Biotechnology Co., Ltd.); Smart LabAssist-32 nucleic acid extraction analyzer (SN: E13200042, Taiwan Dot Nanotechnology Co., Ltd.); ABI7500 fluorescence quantitative PCR instrument (SN: 275054149, Applied Biosystems, USA).

Sample collection, HPV DNA extraction, PCR amplification, and genotyping

Samples were collected using flocking swabs soaked in 0.1 M phosphate-buffered saline (PBS) (pH 7.2) by smearing on target surfaces. Immediately after collection, samples were numbered and stored at 2–8 °C for transport to the molecular laboratory for HPV testing.

For processing, 500 μL of saline was added to each swab tube. The solution was mixed thoroughly and transferred to a 1.5 mL eppendorf tube. After centrifuging at 13,000 rpm for 10 min, 300 μL of the supernatant was discarded, leaving the precipitate and 200 μL of remaining liquid, which were transferred to a 96-well deep plate for nucleic acid extraction. The PCR reaction system consisted of 40 μL of the PCR reaction mixture and 10 μL of the DNA template. Quantitative fluorescence PCR was used to detect HPV, targeting 18 high-risk HPV genotypes (16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, 68, 26,

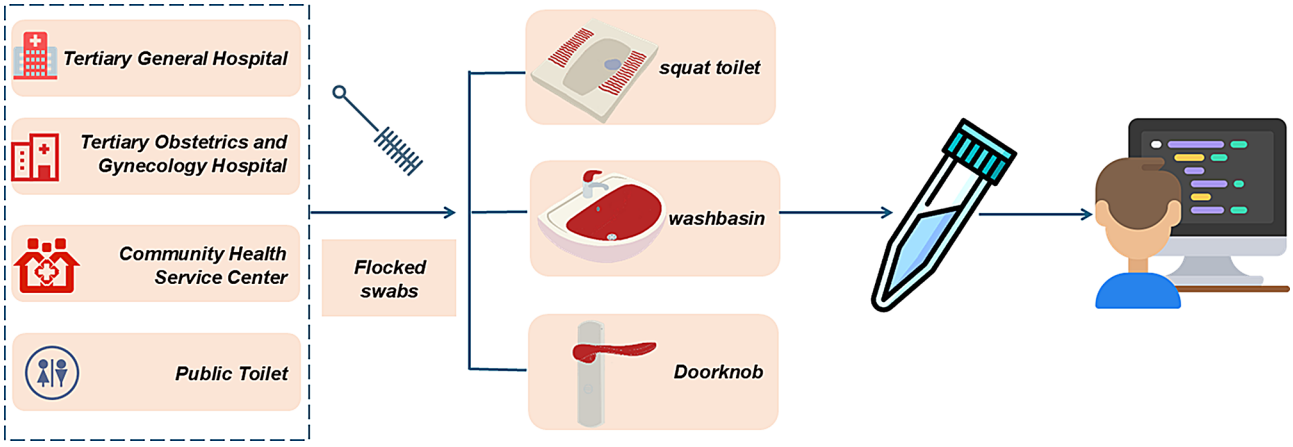

Fig. 1 Flowchart of sample collection from three different areas at four locations. (The red areas of the squat toilet, washbasin, and doorknob in Figure represent the specific sampling areas.)

53, 66, 73, 82) and 5 low-risk genotypes (6, 11, 42, 43, 81). Additionally, β-globin was used as an internal control to verify sample quality. The minimum detection limit was set to 400 copies/mL.

The amplification reaction conditions are as follows: UDG enzyme reaction: 50 °C for 2 min, 1 cycle; Taq enzyme activation: 94 °C for 5 min, 1 cycle; Denaturation: 94 °C for 15 s, 45 cycles; Annealing, extension, and fluorescence collection: 57 °C for 30 s, 45 cycles. For each batch, one positive and one negative control were included. The detection process and result interpretation are following the reagent and instrument manuals. Samples with a CT (Cycle Threshold) value ≥ 37.00 were retested. If the result remained positive upon retesting, it was recorded as positive.

Infection risk analysis

HPV infection risk refers to the possibility and severity of potential adverse effects on human health due to the presence or possible presence of HPV in restroom environments, which may be transmitted through certain pathways. The infection risk value (R) was used to express this.

$$Infection\ risk\ value\ (R) = \sum (Genotype\ score\ (Pi) * Concentration\ score\ (Ci))$$

Note

- i = 1, 2, 3, 4… (representing the different genotypes detected in a single sample).
- p = 1, 2, 3 (Detection of low-risk HPV types is scored as 1; high-risk HPV types, excluding HPV-16 and HPV-18, are scored as 2; HPV-16 and HPV-18 are scored as 3).
- c = 1, 2, 3 (Ct value ≥ 35 is scored as low concentration, 1 point; 30 < Ct value < 35 is scored as medium concentration, 2 points; Ct value ≤ 30 is scored as high concentration, 3 points).

Statistical analysis

Data analysis was performed using SPSS 27.0 statistical software. The comparison of HPV positivity rates among the four locations—general hospitals, obstetrics and gynecology hospitals, community health centers, and public restrooms—was conducted using the chi-square test (X^2). The HPV infection risk values (R) for these locations did not follow a normal distribution, as determined by the Shapiro-Wilk test; therefore the Kruskal-Wallis test was employed for group comparisons. A P-value of < 0.05 was considered statistically significant.

Results

Distribution of HPV genotypes

A total of 19 HPV genotypes were identified across four locations: public restrooms, community health centers, obstetrics and gynecology hospitals, and general hospitals. The five most frequently identified genotypes were HPV-51 (18.99%), HPV-16 (14.56%), HPV-52 (11.39%), HPV-58 (8.23%), and HPV-56 (6.96%). Among the low-risk types, the most common were HPV-42 (4.23%), HPV-43 (2.53%), and HPV-81 (2.53%). Overall, high-risk types constituted 88.61% of all detected HPV genotypes, while low-risk types accounted for 11.39% (Fig. 2A and B).

Distribution of HPV positivity

From the 360 environmental samples collected, 87 tested positive for HPV, yielding an overall positivity rate of 24.17%. Throughout the 30-day sampling period, HPV-positive days were recorded as follows: 11 days in public restrooms, 13 days in community health centers,

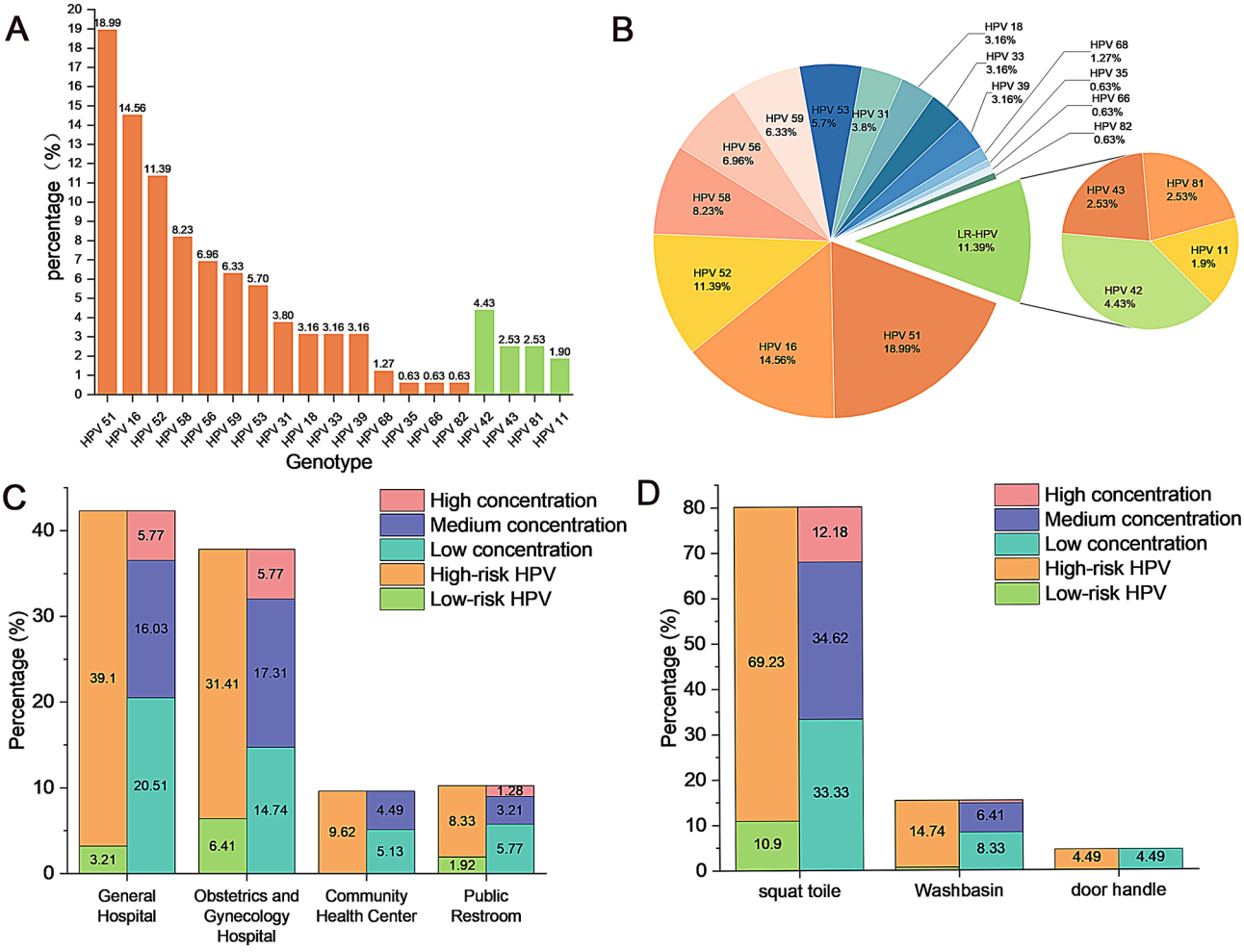

Fig. 2 The proportional distribution of HPV across different locations and areas. (**A**, **B**) Distribution of HPV genotypes in restroom environments; (**C**) Distribution of HPV genotypes and concentrations across four locations; (**D**) Distribution of HPV genotypes and concentrations in three sampling areas

Table 1 Distribution of HPV positivity [n / N (%)]

	General Hospital	Obstetrics and Gynecology Hospital	Community Health Center	Public Restroom	Total	X^2	*P* value
Squat toilet	22/30(73.33)	22/30(73.33)	10/30(33.33)	10/30(33.33)	64/120(53.33)	19.286	< 0.001
washbasin	12/30(40.00)	4/30(13.33)	1/30(3.33)	0/30(0.00)	17/120(14.17)	24.329	< 0.001
Doorknob	1/30(3.33)	1/30(3.33)	2/30(6.67)	2/30(6.67)	6/120(5.00)	0.702	0.873
Total	35/90(38.89)	27/90(30.00)	13/90(14.44)	12/90(13.33)	87/360(24.17)	22.305	< 0.001

Note: n represents the number of HPV-positive samples, N represents the total number of samples collected, and % indicates the proportion of HPV positivity rate

22 days in obstetrics and gynecology hospitals, and 23 days in general hospitals. The highest positivity rate was observed in general hospitals (38.89%), followed by obstetrics and gynecology hospitals (30.00%). HPV was detected in all three sampling areas (door handles, washbasins, and squat toilets), with positivity rates of 5.00%, 14.17%, and 53.33%, respectively (Table 1).

Ct values were utilized as relative indicators of viral load, categorized into three levels: low concentration ($Ct \geq 35$), medium concentration ($30 < Ct < 35$), and high concentration ($Ct \leq 30$). The proportions of samples in these categories were 12.82% for high concentration, 41.03% for medium concentration, and 46.15% for low concentration. Variability in concentrations was noted across the four locations, with low and medium concentrations being predominant overall; however, high concentrations were particularly evident in general hospitals and obstetrics and gynecology hospitals (Fig. 2C). Among the various sampling areas, washbasins and door handles primarily exhibited low concentrations, while high concentrations were largely found in squat toilets (Fig. 2D).

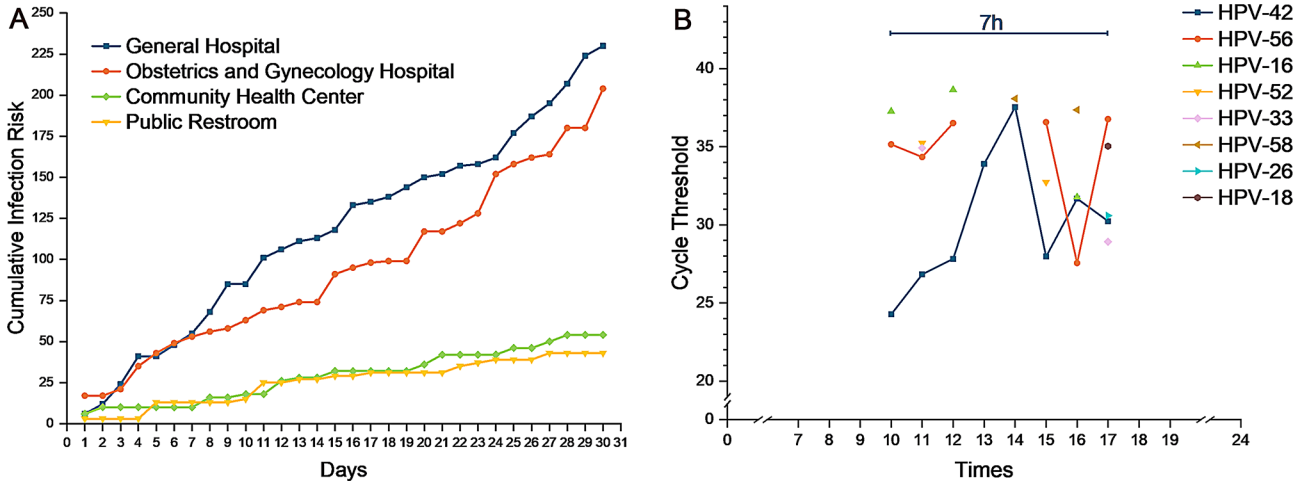

Fig. 3 Cumulative infection risk of HPV across different locations and observation of HPV environmental persistence. (**A**) Cumulative infection risk across different locations; (**B**) Distribution of HPV genotypes and changes in concentration Ct values in squat toilets of the obstetrics and gynecology hospital over a 24-hour period

Table 2 Monitoring of HPV contamination in restroom environments of obstetrics and gynecology hospitals [Negative / Positive (Genotype)]

Time	Cumulative Count	Squat toilet	washbasin	Doorknob
7:00	0	Negative	Negative	Negative
8:00	4	Negative	Positive (59)	Negative
9:00	16	Negative	Negative	Negative
10:00	29	Positive (16,56,42)	Negative	Negative
11:00	44	Positive (33,52,56,42)	Negative	Negative
12:00	56	Positive (16,56,42)	Negative	Negative
13:00	65	Positive (42)	Negative	Negative
14:00	79	Positive (58,42)	Positive (42)	Negative
15:00	89	Positive (52,56,42)	Negative	Negative
16:00	99	Positive (16,56,58,42,18)	Negative	Negative
17:00	104	Positive (33,56,26,42)	Negative	Negative
After Cleaning		Negative	Negative	Negative

HPV infection risk

Among the 87 HPV-positive environmental samples, there were variations in the number and concentration of detected HPV genotypes. To better assess the risk of HPV transmission in different locations, we calculated an infection risk value. The specific calculation formula for this analysis can be found in Sect. 1.4 (Infection Risk Analysis). We calculated cumulative R-value over the 30-day period for each of the four locations (Fig. 3A). Intergroup comparisons were performed using the Kruskal-Wallis test, revealing no significant statistical difference in infection risk between general hospitals and obstetrics and gynecology hospitals ($P=0.209$), nor between community health centers and public restrooms ($P=0.557$). However, the infection risk in general hospitals and obstetrics and gynecology hospitals was significantly higher than in community health centers and public restrooms ($P<0.001$).

Monitoring of HPV environmental contamination in obstetrics and gynecology hospitals during workdays

To investigate HPV detection at various time intervals and the duration of environmental contamination, we conducted a 24-hour monitoring study in a randomly selected restroom of an obstetrics and gynecology hospital, sampling hourly. We also recorded the number of restroom users during each time period. Notably, after the 17:00 sampling, the outpatient clinic closed and a disinfectant solution containing 500 mg/L of chlorine was used for cleaning. A total of 36 samples were collected from this monitoring effort, which were not included in the overall sample size for the positivity rate and genotype distribution statistics. Among these 36 samples, 10 tested positive for HPV, with the highest positivity rate found in squat toilets (Table 2). High-concentration HPV (HPV-42) contamination was detected continuously in the environment for 7 h, while low-concentration HPV contamination had a shorter duration of detection (Fig. 3B).

Discussion

HPV is the primary pathogenic factor for cervical cancer, primarily transmitted through sexual contact. However, cases of genital warts in children who are virgins or have no history of sexual abuse have drawn increased attention to non-sexual transmission routes [10].

In China, HPV-16, HPV-58, and HPV-52 are the most prevalent HPV genotypes across various regions [14–16].

From 360 samples collected across four different environmental settings, a total of 19 HPV genotypes were

identified using the HPV 23-genotyping kit. The most frequently detected genotypes were HPV-51, HPV-16, HPV-52, HPV-58, and HPV-56. These findings are largely consistent with the overall HPV prevalence trends in China and align closely with the distribution of HPV genotypes reported by Qin Song et al. in Hangzhou [17]. According to their study, the most common HPV genotypes were HPV-52, HPV-58, HPV-51, HPV-56, and HPV-16. The high detection rate of HPV-51 in Hangzhou may be related to the increase in HPV vaccination coverage in recent years. Currently, five HPV vaccines have been approved for use in mainland China: three imported vaccines—Cervarix (HPV-16 and − 18), Gardasil (HPV-6, -11, -16, and − 18), and Gardasil 9 (HPV-6, -11, -16, -18, -31, -33, -45, -52, and − 58)—which were introduced in 2016, 2017, and 2018, respectively. Additionally, two domestic vaccines—Cecolin (HPV-16 and − 18) and Walrinvax (HPV-16 and − 18)—were approved in 2019 and 2022, respectively. None of these vaccines include HPV-51 [18]. Additionally, we hypothesize that HPV-51 may have a higher survival rate in the environment, though this requires further research and data for validation. Based on these findings, this study provides theoretical support for the development of an HPV-51 vaccine, warranting further investigation.

Among the four locations we selected—general hospitals, obstetrics and gynecology hospitals, community health centers, and public restrooms—general hospitals and obstetrics and gynecology hospitals had the highest number of days with HPV-positive detections, followed by community health centers, while public restrooms had the lowest. The infection risk in general hospitals and obstetrics and gynecology hospitals was significantly higher than that in community health centers and public restrooms. This suggests that high patient traffic and a greater prevalence of infections may contribute to the increased risk of HPV contamination in hospital environments. S. Strauss et al. [19] have also reported that hospital environments are high-risk areas for HPV contamination. In our analysis of three different sampling areas (door handles, washbasins, and squat toilets), we found that squat toilets had the highest HPV positivity rate and the most notable high concentration of the virus. This may be closely related to aerosol contamination from urine splashes during use and flushing. Previous experiments have shown that flushing squat toilets generates aerosol plumes, which can potentially deposit viral particles on surfaces [20]. Additionally, a study by Huihui Zhang et al. [21] found that the average release intensity of viruses during each flush of a squat toilet is 33.5 times greater than that of bacteria. Our study also detected HPV-positive results on the washbasins and door handles (Table 1; Fig. 2D). Research by C. Sonnex et al. [22]. has indicated that individuals infected with genital HPV carry the same HPV types on their hands, suggesting that HPV may also be transmitted through genital-hand contact. Therefore, in addition to urine contamination, HPV may also be transmitted via the contaminated hands of infected individuals who touch toilet door handles or sink surfaces.

In our monitoring HPV contamination in the women's restrooms at an obstetrics and gynecology hospital, we observed that low-concentration HPV contamination persisted for a short duration, with detection lasting no longer than 3 h. In contrast, high-concentration HPV contamination remained in the environment for a longer period, with continuous detection lasting up to 7 h (Fig. 3D). These results suggest that HPV persistence in the environment is closely related to its concentration. Low-concentration contamination is more easily eliminated within a short time, while high-concentration contamination may persist longer, thereby increasing the risk of potential transmission. It is known that HPV is resistant to several disinfectants but is sensitive to hypochlorite-based disinfectants [23, 24]. After sampling at 17:00, we immediately cleaned the environment using a chlorine-based disinfectant at a concentration of 500 mg/L, and subsequent test results were negative. This underscores the importance of proper disinfectant in effectively eliminating HPV contamination.

In conclusion, HPV was detected at all four locations, with significantly more days of HPV-positive results observed in general hospitals and obstetrics and gynecology hospitals compared to community health centers and public restrooms. HPV was present in all three sampling areas (door handles, washbasins, and squat toilets). Our analysis revealed that high-risk HPV genotypes were much more frequently identified than low-risk genotypes, with medium to high concentrations of HPV accounting for a substantial proportion of positive results. The 24-hour sampling analysis indicated that high-concentration HPV remained in the environment for an extended period. These findings suggest that restroom environments may serve as potential sources of HPV contamination, with door handles, washbasins, and squat toilets in public areas representing possible non-sexual transmission routes for the virus. Consequently, it is recommended that greater emphasis should be placed on the disinfection and cleaning of these areas to mitigate the risk of non-sexual transmission.

Abbreviations

HPV Human papillomavirus
CT Cycle Threshold

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Author contributions
ZHT, XZD, XCW designed and supervised the project. YP, ZLW, PY collect the samples. XZD and JMP performed the experiments, analyzed the data and wrote the manuscript. XXY and WWL edited the manuscript. All authors read and approved the manuscript and agree to be accountable for all aspects of the research in ensuring that the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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Data availability
The datasets generated and analyzed during the current study are available from the corresponding author upon reasonable request. All relevant data are included in the manuscript.

Declarations

Human ethics and consent to participate declarations
This study does not involve human participants, and therefore, Human Ethics and Consent to Participate declarations are not applicable.

Competing interests
The authors declare no competing interests.

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References

1. Hyuna S, Jacques F, Rebecca LS, Mathieu L, Isabelle S, Ahmedin J, Freddie B. Global cancer statistics 2020: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. Cancer J Clin. 2021;71(3):209–49.
2. Cervical cancer https://www.who.int/news-room/fact-sheets/detail/cervical-cancer
3. Paul AC, Jhingran A, Ana O, Lynette D. Cervical cancer. Lancet. 2019;393(10167):169–82.
4. Stefano C, Mattia T, Martina A, Marco La V, Canio M, Vito Andrea C, Vittorio P, Ferdinando Antonio G, Salvatore Gueli A, Giuseppe C, et al. Post treatment sexual function and quality of life of patients affected by cervical cancer: A systematic review. Medicina-lithuania. 2023;59(4):704.
5. Cancer, ICoESoC. Comparison of risk factors for invasive squamous cell carcinoma and adenocarcinoma of the cervix: collaborative reanalysis of individual data on 8,097 women with squamous cell carcinoma and 1,374 women with adenocarcinoma from 12 epidemiological studies. Int J Cancer. 2006;120(4):885–91.
6. Nike O, Kathryn AR, Claire LN, Bhautesh J. Cervical cancer risk factors in eight West African countries: cross-sectional analysis of the demographic and health survey 2017–20. Lancet. 2022;400:S68.
7. Sok King O, Sarah Krull A, Shyamala T, Rei H, Ruchi P, Harindra J, Kayo T, Aliza KCB, Abhishek S, Ashrafun N, et al. Towards elimination of cervical cancer–human papillomavirus (HPV) vaccination and cervical cancer screening in Asian National cancer centers alliance (ANCCA) member countries. Lancet Reg Health - Western Pac. 2023;39:100860.
8. Karla A, Mauricio M, Miriam C, Rachel M, Montserrat S. Removing global barriers to cervical cancer prevention and moving towards elimination. Nat Rev Cancer. 2021;21(10):607–8.
9. Global immunization coverage. 2023 https://www.who.int/news-room/fact-sheets/detail/immunization-coverage
10. Petca A, Borislavschi A, Zvanca M, Petca R-C, Sandru F, Dumitrascu M. Non-sexual HPV transmission and role of vaccination for a better future (Review). Experimental Therapeutic Med. 2020;20(6):1–1.
11. Kelly S, Charles RW, Daniel JK, Sara HS. Anogenital and respiratory tract human papillomavirus infections among children: age, gender, and potential transmission through sexual abuse. Pediatrics. 2005;116(4):815–25.
12. Michela de M, Andrea H, Fritz W, Georg S, Tobias K, Matthias W. High-risk human papilloma virus infection of the foreskin in asymptomatic boys. Urology. 2013;81(4):869–72.
13. Selina V, Eva L, Lisa H. Pediatric condyloma acuminata. J Pediatr Adolesc Gynecol. 2013;26(6):e121–2.
14. Tianyu W, Lin L, Jingjing D, Yu Y, Qianlan W, Tao G, Jingjing Z, Zhuoyu Z, Jun Z. Prevalence and human papillomavirus (HPV) genotype distribution in Suzhou, China. Hum Vaccines Immunotherapeutics. 2023;19(2):2241309.
15. Yetian R, Hui L, Min L, Guangxu C, Xinxin X, Ling H, Fang L. A retrospective analysis of human papillomavirus (HPV) prevalence and genotype distribution among 25,238 women in Shanghai, China revealed the limitations of current HPV-based screening and HPV vaccine. Cancer Epidemiol. 2023;84:102372.
16. Jiang H, Jie D, Huan W, Yongtao Y, Xiaomao L. Prevalence and characteristics of cervical human papillomavirus genotypes and cervical lesions among 58630 women from Guangzhou, China. J Infect Public Health. 2023;16(10):1531–6.
17. Qin S, Xiaoxia W. Prevalence and genotype distribution of HPV in Hangzhou, China. Clin Lab. 2024;70(6):240139.
18. Chao Z, Yun Z, Jingran L, Mingzhu L, Yujing S, Lihui W. Opportunities and challenges for human papillomavirus vaccination in China. Hum Vaccines Immunotherapeutics. 2024;20(1):2329450.
19. Strauss S, Sastry PLK, Sonnex C, Simon E, Jim G. Contamination of environmental surfaces by genital human papillomaviruses. Sex Transm Infect. 2002;78(2):135–8.
20. Tengfei Z, Lifang Y, Zilong G, Feng W. Particle exposure risk to a lavatory user after Flushing a squat toilet. Sci Rep. 2022;12(1):21088.
21. Huihui Z, Xingliang S, Qiuke X, Alvin CKL. Experimental study on droplet and bioaerosol emissions from Flushing a squat toilet. Build Environ 2024, 250.
22. Sonnex C, Strauss S, Jim G. Detection of human papillomavirus DNA on the fingers of patients with genital warts. Sex Transm Infect. 1999;75(5):317–9.
23. Lori IR, Andrew LC, Philip RG, Samina A, Janice M, Reem H, Joseph Che Yen W, Jeff TW, Craig M. Hypochlorous acid as a disinfectant for High-risk HPV: insight into the mechanism of action. J Med Virol. 2022;94(7):3386–93.
24. Jordan MM, Eric JR, Michael JC, Craig M, Richard AR. Susceptibility of high-risk human papillomavirus type 16 to clinical disinfectants. J Antimicrob Chemother. 2014;69(6):1546–50.

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