JIDC | THE JOURNAL OF INFECTION IN DEVELOPING COUNTRIES

Original Article

Cervical HPV in central China: regional prevalence, subtypes and pathology

Jin Guan[1 #], Hongliu Liu[2 #], Jilan Ll[3 #], Zhuolin Chang[3 #], Mingtao Zhang[2 #], Xuan Xiao[1]

[1] Department of Gynecology, Jingmen Central Hospital and Jingmen Central Hospital Affiliated to Jingchu University of Technology, Jingmen 448000, Hubei, China
[2] Department of Oncology, Jingmen Central Hospital and Jingmen Central Hospital Affiliated to Jingchu University of Technology, Jingmen 448000, Hubei, China
[3] Department of Gynecology, Gongli Hospital Affiliated with Shanghai University of Medicine and Health Sciences, Shanghai 200135, China

# Authors contributed equally to this work.

Abstract

Introduction: This study investigated the epidemiological characteristics, subtype distribution, and clinical correlations of cervical human papillomavirus (HPV) infection in Jingmen, China.

Methodology: A retrospective study of 5,155 women screened at Jingmen Central Hospital (2022–2024). The participants were categorized into 6 age groups: ≤ 20 years (n = 54), 21-30 years (n = 791), 31-40 years (n = 1,757), 41-50 years (n = 1,371), 51-60 years (n = 968), and ≥ 61 years (n = 214). HPV genotyping and histopathology were used to assess infection patterns and lesion correlations.

Results The infection rates of high-risk HPV (HR-HPV), low-risk HPV (LR-HPV), and mixed infections were 18.10% (933/5,155), 3.38% (174/5,155), and 3.38% (174/5,155), respectively. HR-HPV infections exhibited a bimodal age distribution, with peak prevalence in women aged ≤ 20 years (33.33%) and ≥ 61 years (39.25%). This age-related difference was statistically significant ($\chi^2 = 81.430$, $p < 0.001$). The dominant subtypes were HPV52 (23.5%), HPV16 (13.9%), and HPV58 (13.3%). Notably, HPV16 was significantly more prevalent in high-grade squamous intraepithelial lesions (HSIL; 44.3%) and cervical cancer (CC; 55.5%), compared with low-grade lesions ($p < 0.01$).

Conclusions: This study identified both adolescent/young women (≤ 20 years) and older women (≥ 61 years) as high-risk populations for HR-HPV infection. Notably, HPV16 (55.5%) exhibited significantly higher detection rates in cervical cancer cases, emphasizing the importance of prioritizing this subtype in region-specific vaccine-based prevention strategies. These findings underscore the need for tailored clinical management approaches based on viral subtype distribution and lesion severity.

Key words: HPV; cervical; carcinoma; oncogenic.

J Infect Dev Ctries 2026; 20(1):129-133. doi:10.3855/jidc.21716

(Received 10 April 2025 – Accepted 19 June 2025)

Introduction

The causal relationship between human papillomavirus (HPV) infection and cervical carcinogenesis was systematically established by Professor Zur Hausen's team—a breakthrough recognized with the 2008 Nobel Prize in Physiology or Medicine [1]. This foundational work built upon earlier morphological evidence of HPV in cervical lesions.

Over nearly five decades of extensive research, robust evidence has confirmed that persistent high-risk HPV (HR-HPV) infection is the central etiological factor in cervical cancer. More than 200 HPV genotypes have been identified to date, with approximately 40 subtypes capable of infecting the genital epithelium. The International Agency for Research on Cancer classifies subtype 16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, and 68 as high-risk based on their oncogenic potential [2]. Notably, persistent HR-HPV infection confers a 200- to 300-fold increased risk of progression to cervical cancer compared to transient infections, along with significantly poorer clinical outcomes [2]. Despite numerous epidemiological studies on HPV, region-specific dynamic surveillance data remain limited. This study analyzed cervical HPV screening results from 5,155 women in Jingmen, Hubei Province, with three primary objectives: (1) to update the HPV infection spectrum and subtype distribution in central China; (2) to delineate age-specific risk patterns among high-risk populations; and (3) to elucidate associations between HPV subtypes and the severity of cervical lesions, thereby informing precise prevention strategies.

Methodology

Clinical data

A total of 5,155 women aged 18–79 years (mean age: 41 ± 11 years) who underwent cervical HPV screening at the Department of Gynecology outpatient

clinic of Jingmen Central Hospital between July 2022 and July 2024 were enrolled for this retrospective study. The participants were recruited during routine cervical cancer screening visits. They were stratified into 6 age groups: ≤ 20 years (n = 54), 21–30 years (n = 791), 31–40 years (n = 1,757), 41–50 years (n = 1,371), 51–60 years (n = 968), and ≥ 61 years (n = 214).

The inclusion criteria were: history of sexual activity; no prior HPV vaccination; screening performed outside menstruation, pregnancy, or acute vaginal inflammation; and signed informed consent. The exclusion criteria were: post-hysterectomy status, history of cervical cancer, and cervical local therapy within 6 months prior to screening.

The study protocol was approved by the Institutional Ethics Committee (Approval No. 2022-EC-015).

Specimen collection and detection

The samples were collected ≥ 72 hours after the resolution of menstruation, vaginal bleeding, active inflammation, or topical vaginal treatment. The participants were placed in a lithotomy position. After visualizing the cervix with a disposable sterile speculum, a dedicated cervical cytobrush (Cervi-Brush®; CooperSurgical, Trumbull, CT, USA) was inserted into the endocervical canal, rotated clockwise 5 times, held in place for 10 seconds, and then gently withdrawn.

The brush heads were immediately placed into 2 mL of ThinPrep® preservation solution in dedicated vials, refrigerated at 4 °C, and transported to the laboratory within 2 hours.

HPV genotyping was performed using real-time fluorescence polymerase chain reaction (Hybribio HPV Genotyping Kit; Hybribio Ltd., Chaozhou, China), which detects 23 HPV subtypes including 17 high-risk subtypes (16, 18, 31, 33, 35, 39, 45, 51, 52, 53, 56, 58, 59, 66, 68, 73, 82) and 6 low-risk subtypes (6, 11, 42, 43, 44, 81).

Statistical analysis

Statistical analyses were conducted using SPSS 19.0 (IBM Corporation, Armonk, NY, USA). Categorical variables were presented as frequencies (n) and percentages (%). Intergroup comparisons were performed using Chi square (χ^2) tests or Fisher's exact tests, as appropriate. Age-stratified HPV infection rates were calculated, with a significance threshold set at $p < 0.05$.

Results

HPV infection profiles

Among the 5,155 women screened, HPV genotyping revealed HR-HPV infections in 18.10% (933/5,155), comprising single-subtype infections (14.47%, 746 cases) and multiple-subtype infections (3.63%, 187 cases) (Table 1). Low-risk HPV (LR-HPV) infections were detected in 3.38% (174/5,155), while mixed high- and low-risk infections occurred in 3.38% (174/5,155).

Age-specific distribution of HR-HPV infections

HR-HPV infection rates exhibited a statistically significant bimodal age distribution ($\chi^2 = 81.430$, $p < 0.001$), with distinct peaks in adolescents (≤ 20 years: 33.33%, 18/54) and postmenopausal women (≥ 61 years: 39.25%, 84/214).

It is critical to clarify two distinct statistical definitions of HR-HPV infection prevalence:

(1) Strict HR-HPV infection: comprising solely single-type (n = 746) and multiple-type (n = 187) high-risk infections, totaling 933 cases (18.10%);

(2) Broad HR-HPV infection: additionally including high-risk types detected in mixed infections (n = 174), totaling 1,107 cases (21.47%) (detailed in Table 2).

This study employed the broad definition for age-distribution analysis to comprehensively characterize the epidemiological profile of high-risk HPV exposure, and intermediate age groups demonstrated lower prevalence. The prevalence by age group were: 17.83% (141/791) for 21–30 years, 17.93% (315/1,757) for 31–40 years, 21.01% (288/1,371) for 41–50 years, and 26.96% (261/968) for 51–60 years (Table 2).

Dominant HR-HPV subtype distribution

HPV52 (23.5%), HPV16 (13.9%), and HPV58 (13.3%) were the most prevalent subtypes, collectively accounting for 50.7% of all HR-HPV infections among

Table 1. Distribution of cervical HPV infection patterns among 5,155 women in Jingmen, Hubei Province.

HPV infection type	Cases (n)	Prevalence %, (95% CI)
Single HR-HPV Infection	746	14.47 (13.53–15.45)
Multiple HR-HPV Infections	187	3.63 (3.13–4.18)
LR-HPV Infections	174	3.38 (2.89–3.91)
Mixed HR/LR HPV Infections	174	3.38 (2.90–3.90)
Total	1281	24.85 (23.65–26.08)

HPV: human papillomavirus; HR-HPV: high-risk human papillomavirus; LR-HPV: low-risk human papillomavirus; CI: confidence interval.

Table 2. Age-stratified prevalence of HR-HPV infections.

Age group (years)	Screened (n)	Infected (n)	Prevalence (%)
≤ 20	54	18	33.33
21–30	791	141	17.83
31–40	1757	315	17.93
41–50	1371	288	21.01
51–60	968	261	26.96
≥ 61	214	84	39.25
Total	5155	1107	21.47

HR-HPV infection prevalence in this table includes both: (1) pure high-risk infections (single-type and multiple-type), and (2) high-risk types identified in mixed infections. The previously reported 18.10% prevalence refers solely in the case of pure HR-HPV infections. HR-HPV: high risk human papillomavirus.

the 746 single HR-HPV infections (Table 3).

Association between HR-HPV subtypes and cervical lesion severity

The distribution among 559 histopathologically confirmed cervical lesion cases was as follows: 435 cases (77.8%) of low-grade squamous intraepithelial lesions (LSIL), 106 cases (19.0%) of high-grade squamous intraepithelial lesions (HSIL), and 18 cases (3.2%) of cervical cancer (CC).

HR-HPV detection rates varied significantly across lesion grades (LSIL: 70.80% vs. HSIL: 84.91% vs. CC: 72.22%; $\chi^2 = 8.722$, $p = 0.013$). Notably, HPV16 and HPV58 exhibited pronounced oncogenic predominance in advanced lesions (Table 4).

HPV16 detection dynamics

A marked increase in HPV16 prevalence was observed with disease progression: 20.0% (87/435) for LSIL, 44.3% (47/106) for HSIL, and 55.5% (10/18) for CC ($\chi^2 = 35.036$, $p < 0.001$), underscoring its biological significance as a principal oncogenic driver.

HPV58 detection pattern

HPV58 also showed a progressive increase in detection rates: 12.0% (52/435) for LSIL, 14.2% (15/106) for HSIL, and 16.7% (3/18) for CC. however, no statistically significant differences were observed among the three lesion groups ($\chi^2 = 0.667$, $p = 0.716$).

Table 3. Relative frequency of HR-HPV subtypes in single infections (n = 746).

HPV genotype	Cases (n)	Proportion (%)
16	104	13.9
18	34	4.6
31	15	2.0
33	22	2.9
35	10	1.3
39	21	2.8
45	4	0.5
51	54	7.2
52	175	23.5
53	79	10.6
56	40	5.4
58	99	13.3
59	21	2.8
66	24	3.2
68	39	5.2
73	2	0.3
82	3	0.4
Total	746	100.00

HR-HPV: high risk human papillomavirus.

Discussion

Geographical heterogeneity in HPV epidemiology

The prevalence and subtype distribution of cervical HPV infections exhibit marked geographical heterogeneity. Recent epidemiological studies demonstrate substantial regional variations across China. The overall HPV prevalence was 13.3% in the Southeastern Zhejiang Province, with HR-HPV accounting for 10.2% and HPV16/52/58 collectively comprising > 90% of HR-HPV infections [3]. The overall HPV prevalence in Tibet Autonomous Region was 9.19%, including HR-HPV (7.05%), LR-HPV (2.14%), and mixed infections (1.32%); with HPV16, 33, 58, 52, and 31 as the predominant subtypes [4]. The regional HPV prevalence in Taiwan, China was reported to be 19.3% [5]. HR-HPV prevalence in Hainan Province was 10.91%, with bimodal peaks in the 20–29 years (14.87%) and 55–64 years (12.45%) age groups [6]. HPV prevalence in Xinjiang Urumqi was 12.46%, with HPV52 (17.11%) and HPV16 (16.03%) as dominant subtypes [7].

Table 4. Distribution of predominant HR-HPV subtypes across cervical lesion grades.

HPV subtype	LSIL (n = 435)	HSIL (n = 106)	CC (n = 18)
52	78 (17.9%)	13 (12.2%)	0
16	87 (20.0%)	47 (44.3%)	10 (55.5%)
58	52 (12.0%)	15 (14.2%)	3 (16.7%)
53	43 (9.9%)	6 (5.7%)	0
51	48 (11.0%)	9 (8.5%)	0
Other	127 (29.2%)	16 (15.1%)	5 (27.8%)

HR-HPV: high-risk human papillomavirus; LSIL: low-grade squamous intraepithelial lesion; HSIL: high-grade squamous intraepithelial lesion; CC: cervical cancer.

This study conducted in Jingmen revealed a higher HR-HPV prevalence (18.10%) than the national average (15.1% in a 2019 meta-analysis) [8], with HPV52 (23.5%), HPV16 (13.9%), HPV58 (13.3%), HPV53 (10.6%), and HPV51 (7.2%) as dominant subtypes—a pattern aligning with broader Chinese population characteristics (consistent with The Lancet Regional Health 2023 HPV genotyping report) [9].

Age-specific risk dynamics

The observed bimodal age distribution of HR-HPV infections—with peaks in adolescents (≤ 20 years, 33.33%) and postmenopausal women (≥ 61 years, 39.25%)—reflects distinct pathophysiological and behavioral risk profiles. In the younger cohort, the elevated risk was associated with early sexual debut (< 18 years), high-frequency intercourse (≥ 5 times/month), multiparity, and limited protective behaviors (e.g., inconsistent condom use) [10]. Among the older cohort, increased susceptibility may result from estrogen depletion-induced mucosal atrophy, impaired local immunity, and delayed viral clearance [11]. Notably, data from the United States Centers for Disease Control and Prevention (CDC) show a higher HPV prevalence in women who have sex with women (28.6%) compared to heterosexual women (20.3%), with comparable HPV16/18 burdens [12].

Oncogenic subtype progression and screening implications

Persistent HR-HPV infection—observed in approximately 20% of cases beyond 12 months—is the critical oncogenic driver, facilitating viral genome integration and immune evasion [13–15]. The findings in this study underscore the leading role of HPV16 in cervical carcinogenesis, with detection rates rising from 20.0% in LSIL to 44.3% in HSIL, and 55.5% in CC ($p < 0.001$). These trends support a risk-stratified screening algorithm:

(1) HPV16-positive women should undergo immediate colposcopy due to the high risk of cervical cancer (55.5% in the cohort).
(2) HPV53/51-positive women without cytological abnormalities may delay biopsy.

Globally, HPV16/18 account for 70% of cervical cancers [16], consistent with the findings in this study (HPV16: 55.5% in CC cases).

Multidimensional prevention framework

The current HPV management practice prioritizes lesion treatment over viral eradication, reinforcing the essential role of vaccination and screening. Nonvalent vaccines (targeting HPV6/11/16/18/31/33/45/52/58) demonstrate > 90% efficacy against HSIL+ [17], covering 100% of the top three HR-HPV subtypes in Jingmen (HPV52/16/58).

The following strategic priorities are recommended for HPV prevention: integration of bivalent vaccines into regional immunization programs, expansion of monitoring for oncogenic risk associated with HPV52/16, and acceleration of domestic vaccine development (two 1st-generation vaccines approved; 20-valent candidates in preclinical phase) [18].

A multidimensional prevention model should combine behavioral interventions to reduce vaginal douching and promote barrier protection. Biomedical strategies should include treating coinfections (e.g., vaginitis) and optimize screening intervals. Social support should tailor services for sexual minorities and high-parity populations.

Conclusions

Although this study provides essential baseline data, the moderate sample size may limit the statistical power to assess rare subtypes. As a single-center study in Jingmen City (population ≈ 2.6 million; Hubei Provincial Statistical Yearbook 2023 [19]), the findings may not fully represent the diverse demographics of Central China. Future efforts should include multicenter validation and causal inference modeling to refine risk stratification. Cost-effectiveness analyses are also needed to guide resource allocation for vaccination scale-up, particularly targeting adolescent and postmenopausal populations.

Corresponding author

Xuan Xiao, Master of Medicine.
168 Xiangshan Avenue, Jingmen 448000, Hubei Province, China.
Tel: +86-724-2305901
Fax: +86-724-2305901
Email: 295119417@qq.com

Conflict of interest

No conflict of interest is declared.

References

1. Zur Hausen H (1977) Human papillomaviruses and their possible role in squamous cell carcinomas. In Arber W, Henle W, Hofschneider PH, Humphrey JH, Klein J, Koldovský P, Koprowski H, Maaløe O, Melchers F, Rott R, Schweiger HG, Syruček L, Vogt PK, editors. Current Topics in Microbiology and Immunology. Vol 78. Berlin Heidelberg: Springer. 1–30. doi: 10.1007/978-3-642-66800-5_1.
2. Oyouni AAA (2023) Human papillomavirus in cancer: infection, disease transmission, and progress in vaccines. J

Infect Public Health 16: 626–631. doi: 10.1016/j.jiph.2023.02.014.
3. Ye J, Cheng X, Chen X, Ye F, Lü W, Xie X (2010) Prevalence and risk profile of cervical human papillomavirus infection in Zhejiang Province, southeast China: a population-based study. Virol J 7: 66. doi: 10.1186/1743-422X-7-66.
4. Jin Q (2009) Prevalence of human papillomavirus infection in women in Tibet Autonomous Region of China. Chinese Journal of Obstetrics and Gynecology 44: 898–902. doi: 10.3760/cma.j.issn.0529-567x.2009.12.005.
5. Lin H, Ma YY, Moh JS, Ou YC, Shen SY, Chang Chien CC (2006) High prevalence of genital human papillomavirus type 52 and 58 infection in women attending gynecologic practitioners in South Taiwan. Gynecol Oncol 101: 40–45. doi: 10.1016/j.ygyno.2005.09.028.
6. Dou QR (2024) Report on high-risk human papillomavirus infection and screening for cervical cancer and precancerous lesions among women in Hainan Province between 2019 and 2022. Chinese Journal of Nosocomiology 34: 1210–1216. doi: 10.11816/cn.ni.2024-231459.
7. Ayipari A (2023) Analysis of 2589 cases of cervical human papillomavirus infection in Urumqi of China. Chin J Clin Obstet Gynecol 24: 73–74. doi: 10.13390/j.issn.1672-1861.2023.01.023.
8. Wang XD (2023) Analysis of influencing factors for high-risk human papillomavirus infection. Medical Journal of Peking Union Medical College Hospital 14: 153–158. doi: 10.12290/xhyxzz.2022-0193.
9. Fu YY (2024) Risk factors for high-risk human papillomavirus infection and the influence on vaginal microbiota. Chin J Clin Obstetrics GynEcol 25: 4–7. doi: 10.13390/j.issn.1672-1861.2024.01.002.
10. Muñoz N, Bosch FX, De Sanjosé S, Herrero R, Castellsagué X, Shah KV, Snijders PJF, Meijer CJLM (2003) Epidemiologic classification of human papillomavirus types associated with cervical cancer. N Engl J Med 348: 518–527. doi: 10.1056/NEJMoa021641.
11. Sharifian K, Shoja Z, Jalilvand S (2023) The interplay between human papillomavirus and vaginal microbiota in cervical cancer development. Virol J 20: 73. doi: 10.1186/s12985-023-02037-8.
12. Pal A, Kundu R (2020) Human papillomavirus E6 and E7: the cervical cancer hallmarks and targets for therapy. Front Microbiol 10: 3116. doi: 10.3389/fmicb.2019.03116.
13. Nelson CW, Mirabello L (2023) Human papillomavirus genomics: understanding carcinogenicity. Tumour Virus Res 15: 200258. doi: 10.1016/j.tvr.2023.200258.
14. Jia XH (2023) Progress in research of long-term protective efficacy of human papillomavirus vaccine. Chinese Journal of Epidemiology 44: 851–854. doi: 10.3760/cma.j.cn112338-20221025-00905.
15. Schiffman M, Doorbar J, Wentzensen N, De Sanjosé S, Fakhry C, Monk BJ, Stanley MA, Franceschi S (2016) Carcinogenic human papillomavirus infection. Nat Rev Dis Primers 2: 16086. doi: 10.1038/nrdp.2016.86.
16. Yu L, Majerciak V, Zheng ZM (2022) HPV16 and HPV18 genome structure, expression, and post-transcriptional regulation. IJMS 23: 4943. doi: 10.3390/ijms23094943.
17. Williamson AL (2023) Recent developments in human papillomavirus (HPV) vaccinology. Viruses 15: 1440. doi: 10.3390/v15071440.
18. Chen Q (2023) Development and application of prophylactic human papilloma virus vaccines in China. Zhongua Liu Xing Bing Xue Za Zhi. 27: 249–253. doi: 10.16462/j.cnki.zhjbkz.2023.03.001.
19. Hubei Provincial Bureau of Statistics (2023) Hubei Statistical Yearbook 2023. Beijing: China Statistics Press 538 p.