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HPV Prevalence, Subtype Distribution, and Vaccine-Associated Changes in Fuzhou Women Undergoing Health Examinations: An 11-Year Retrospective Study (2014–2025)

pengju cao
Fuzhou University Affiliated Provincial Hospital
Binbin Zhuang
Fujian Health College
Yongwen Tang
Anxi County Maternal and Child Health Hospital
Yang Yang
Gansu Provincial Hospital
Xiuqing Shen
Fuzhou University Affiliated Provincial Hospital
Shaoting Chen
shaotingchen@126.com
Fuzhou University Affiliated Provincial Hospital
Huan Zhou
Fuzhou University Affiliated Provincial Hospital

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HPV Prevalence, Subtype Distribution, and Vaccine-Associated Changes in Fuzhou Women Undergoing Health Examinations: An 11-Year Retrospective Study (2014–2025)

[#]Pengju Cao[1,2] [#]Binbin Zhuang[3] Yongwen Tang[4] Yang yang[5] Xiuqing Shen[1,2] *Shaoting Chen[1,2] *Huan Zhou[1,2]

1. Department of Clinical Laboratory, Fuzhou University Affiliated Provincial Hospital, Fuzhou, Fujian 350001, China.
2. Department of Clinical Laboratory, Shengli Clinical Medical College of Fujian Medical University, Fuzhou, Fujian 350001, China.
3. Fujian Health College, Fuzhou, Fujian 350101, China.
4. Anxi County Maternal and Child Health Hospital, Quanzhou, Fujian 362499，China.
5. Department of Clinical Laboratory, Gansu Provincial Hospital, Lanzhou730000 ,China

# These authors contributed equally to this work

Corresponding author: Huan Zhou E-mail: 13799386727@163.com and Shaoting Chen E-mail: shaotingchen@126.com

Background: To assess long-term trends in Human Papillomavirus (HPV) infection rates, subtype distribution, and age-specific patterns before and after HPV vaccination in Fuzhou, China, to inform cervical cancer prevention.

Methods: This retrospective study analyzed 29,041 women (aged 16–79) who underwent HPV genotyping at Fuzhou University Affiliated Provincial Hospital from February 2014 to December 2025. Participants were grouped into pre-vaccination (2014 – 2017; n=18,542) and post-vaccination (2020 – 2025; n=10,499). Using fluorescent microsphere hybridization, 27 HPV subtypes (17 high-risk, 10 low-risk) were detected; overall positivity, high-/low-risk rates, and type-specific prevalence were compared. Age–infection probability was modeled using restricted cubic spline regression.

Results: Overall HPV positivity was 15.77% (15.65% pre vs. 15.97% post; P=0.213). A persistent U-shaped age distribution was observed in both phases, peaking at ≤30 and ≥51 years (nadir 31-40). Post-vaccination, high-risk HPV decreased (14.49%→ 12.90%; RR=0.89; P<0.001), notably in the 31–40 group (RR=0.79); low-risk HPV increased (5.64% to 7.40%; RR=1.31; P<0.001), especially ≤30 years (RR=2.29). Bivalent-targeted types (HPV16/18) fell 48.0% and nine-valent-targeted types 13.4%, with greatest benefit at 31–40. Among young women, HPV16/18 declined while HPV6, HPV81, and HPV44 rose (HPV6 RR=4.24; HPV81 RR=3.17 in ≤30).

Conclusions: HPV vaccination in Fuzhou reduced HPV16/18 prevalence, yet rising non-vaccine types (HPV6, HPV81) in young women suggest possible type replacement. Continued molecular surveillance, broader vaccines, and age-specific screening are warranted.

Keywords: Human papillomavirus; HPV vaccination; Subtype distribution; Epidemiology; Type replacement

Background

Cervical cancer is a major public health challenge worldwide[1]. Persistent infection with high-risk human papillomavirus (HPV) is the primary etiological agent [2, 3], with progression to invasive cervical cancer potentially taking up to a decade[4]. HPV epidemiology varies substantially by region and time in prevalence, age-specific distribution, and circulating genotypes, highlighting the need for locally tailored prevention strategies[5].

Although HPV vaccine coverage increased after a public-benefit program launched in Fuzhou in 2017, the resulting epidemiological changes remain poorly characterized because most evidence derives from cross-sectional surveys lacking longitudinal trends. Using 11-year data split into pre- and post-vaccination phases, this study evaluated temporal changes in HPV infection rates, subtype distribution, and age-specific patterns, providing evidence to inform prevention strategies and WHO 2030 cervical cancer elimination goal[6].

1 Materials and methods

1.1 Study design and participants

This retrospective study included women who underwent HPV testing at the Health Examination Center of Fuzhou University Affiliated Provincial Hospital (February 2014-December 2025). Repeat samples from the same individual were excluded.

This retrospective observational study was reported in accordance with the STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) statement. The completed STROBE checklist is provided as Supplementary Material.

A total of 29,041 women aged 16-79 years (mean 44.02 +/- 12.93) were enrolled and divided by vaccination phase: Phase 1, pre-vaccination (February 2014-December 2017), n = 18,542; Phase 2, post-vaccination promotion (January 2020-December 2025), n = 10,499. HPV testing was suspended in 2018-2019 due to institutional

policy and these years were excluded. Participants were stratified into four age groups (≤30, 31-40, 41-50, ≥51). HPV positivity indicated any subtype; single infection denoted one subtype and multiple infection ≥2.

1.2 Ethics Statement

This study was approved by the Medical Ethics Committee of Fuzhou University Affiliated Provincial Hospital (No. K2026-08-002). Anonymized diagnostic data were used with informed consent waived by the committee; patients and the public were not involved.

1.3 Instrument, Reagents and HPV Genotyping

Cervical exfoliated cells were collected from the squamous-columnar junction with a cervical brush and preserved in cell preservation solution. DNA was extracted using an HPV nucleic acid kit (Tianlong, Xi'an) and amplified by PCR (Bioer FQD-96A, Hangzhou). Genotyping used an HPV typing kit (Tellgen, Shanghai) with fluorescent microsphere hybridization on a Luminex 200 system.

A total of 27 HPV subtypes were detected, including 17 high-risk (16, 18, 26, 31, 33, 35, 39, 45, 51, 52, 53, 56, 58, 59, 66, 68, 82) and 10 low-risk (6, 11, 40, 42, 43, 44, 55, 61, 81, 83) per IARC 2020 classification.

1.4 Statistical Methods

Associations between HPV infection and age, and type-specific prevalence changes before and after vaccination, were analyzed using R 4.2.0 and SPSS 26.0. Categorical data are shown as n (%). The chi-square test compared positivity between periods with Bonferroni correction; two-sided $P < 0.05$ was significant.

Rate ratios (RR) with 95% CIs were calculated as post/pre-vaccination positivity; absolute changes as post minus pre. Restricted cubic spline (RCS) regression with three knots modeled the age-infection probability relationship, adjusted for vaccination phase. For vaccine-targeted type analysis, infection rates of bivalent- and nonavalent-covered types were compared between periods by chi-square test.

2 Results

2.1 Descriptive analysis of the study population

Of 29,041 women, 18,542 (63.85%) in Phase 1 and 10,499 (36.15%) in Phase 2. Overall

HPV positivity rate was 15.77%, with no significant difference between phases (15.65% vs. 15.97%; P = 0.213). Age-specific prevalence exhibited a consistent U-shape in both periods, peaking at ≤30 and ≥51 years, with a nadir at 31-40 years (Table 1), largely driven by multiple infections, which followed a similar biphasic trend across both phases. In contrast, single infections increased monotonically with age in Phase 1, but shifted to a U-shaped pattern in Phase 2. Overall, single infections (12.35%) predominated over multiple infections (3.42%). Age-stratified analysis revealed a significant increase in multiple infections among women aged ≤30 years, from 3.95% to 5.64%.

Table 1. HPV infection status by age group and study period

Age	Phase 1 (Pre-vaccination, n=18,542)			Phase 2 (Post-vaccination, n=10,499)		
years	Single Infection	Multiple Infection	Total	Single Infection	Multiple Infection	Total
≤30	164 (11.37%)	57 (3.95%)	1443	79 (12.38%)	36 (5.64%)	638
31-40	492 (11.69%)	113 (2.68%)	4210	267 (9.71%)	58 (2.11%)	2751
41-50	809 (12.06%)	167 (2.49%)	6706	393 (12.07%)	84 (2.58%)	3256
≥51	802 (12.97%)	298 (4.82%)	6183	581 (15.08%)	179 (4.64%)	3854
Total	2267 (12.23%)	635 (3.42%)		1320 (12.57%)	357 (3.40%)	

2.2 Changes in high-risk and low-risk HPV infection rates

To evaluate the impact of vaccination on HPV risk category distribution, we calculated rate ratios of post versus pre-vaccination positivity, as shown in Figure 1. Among the eight age-and risk-specific comparisons, four were statistically significant: high-risk HPV declined significantly in the 31-40 years group, whereas low-risk HPV increased in the ≤30, 41-50, and ≥51 years groups. At the aggregate level, both the high-risk decrease and low-risk increase were statistically significant.

In the remaining age-specific comparisons, no statistically significant changes were detected for high-risk HPV in the ≤30, 41–50, or ≥51 years age groups. For low-risk

HPV, the 31–40 years age group exhibited no significant change.

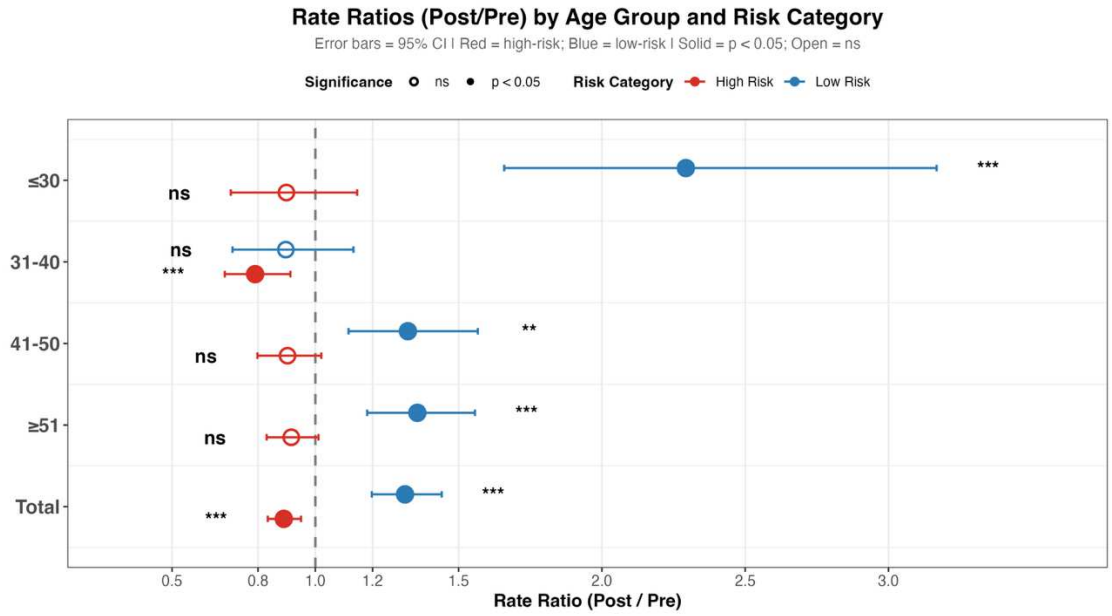


Figure 1. Rate ratios (post/pre) for HPV positivity by risk category and age group.
Red circles represent high-risk HPV types; blue circles represent low-risk HPV types. Solid circles indicate statistically significant differences ($P < 0.05$), while open circles indicate non-significance. Error bars indicate 95% confidence intervals. The dashed vertical line represents a rate ratio of 1.0. Asterisks denote significance levels: $P < 0.05$, *$P < 0.01$, **$P < 0.001$. **ns** = not significant.

2.3 HPV type-specific positivity rate analysis

Overall HPV prevalence alone is inadequate because subtype infection characteristics differ markedly[7]. Vaccination induced significant shifts in genotype detection rates [8]. Figure 2 shows positivity before and after vaccination by type and age. Pre-vaccination phase, HPV52 was highest across all age, particularly among women aged ≥51 years (2.88%) (Figure 2A). HPV52 remained predominant overall; in the ≤30 years subgroup, HPV81 ranked among the top three (2.19%) (Figure 2B).

Absolute changes (Figure 2C) showed HPV16 and HPV18 decreased by 1.05 and 1.03 percentage points, respectively. The largest increases were observed for HPV6 (+1.80 percentage points in ≤30 years), followed by HPV81 (+1.50 percentage points in ≤30 years) and HPV44 (+0.93).

The forest plot (Figure 2D) quantified these changes. Among the vaccine-targeted types, HPV18 declined most sharply in the 31-40 group (RR = 0.20, 95% CI: 0.09-0.44), with significant reductions in the 41-50 and ≥51 years groups (RR range: 0.33-

0.45). HPV33 declined significantly in the 41–50 and ≥51 years groups (RR range: 0.36–0.42) and HPV16 declined significantly in the 31–40, 41–50, and ≥51 years groups (RR range: 0.48-0.61). In contrast, HPV6 rose solely in the ≤30-year group (RR = 4.24, 95% CI: 1.82-9.88), followed by HPV81, which increased significantly in the ≤30, 41-50, and ≥51 years groups (RR range: 1.86-3.17). HPV44 increased in the ≤30 years (RR = 2.91, 95% CI: 1.32-6.42) and ≥51 years (RR = 1.76, 95% CI: 1.23-2.52) groups, while HPV61 increased in the 41–50 years and ≥51 years (RR =1.54-1.60) groups. In contrast, HPV52 and HPV53 showed minimal changes across all age groups, with RR values remaining close to 1.0.

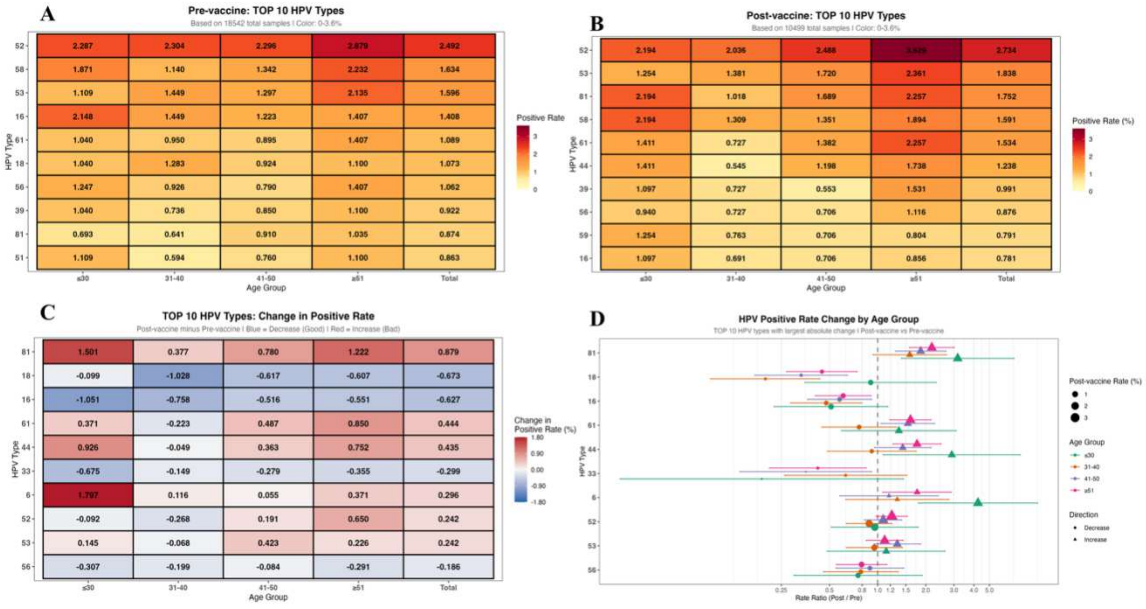


Figure 2. HPV positivity rates before and after vaccination. (A) Pre-vaccination positivity rates. (B) Post-vaccination positivity rates. (C) Absolute changes (post minus pre); red = increase, blue = decrease. (D) Forest plot of rate ratios (post/pre) by age group; error bars = 95% CI, dashed line = RR = 1. Colors: green (≤30 years), orange (31–40 years), purple (41–50 years), pink (≥51 years).

2.4 Bivalent vaccine target types analysis

For the bivalent vaccine (HPV16/18), aggregate positivity was stable (15.65% vs. 15.97%), but HPV16/18 declined from 2.52% to 1.31% (-1.21; -48.0%), while non-targeted types rose from 13.13% to 14.66% (+1.53) (Figure 3A).

Age-stratified analysis (Figure 3B-3D) showed pronounced age-dependence. HPV16/18 reductions were greatest in 31-40 (-1.73, -65.5%), then 41-50 (-1.40, -55.1%)

and ≥51 (-1.11, -45.1%), smallest in ≤30 (-1.03, -35.4%).

RCS (Figure 3C) confirmed that the U-shaped age distribution of HPV16/18 became more pronounced post-vaccination, with the nadir at 31-40 years showing the largest decline and the younger peak (≤30 years) the least.

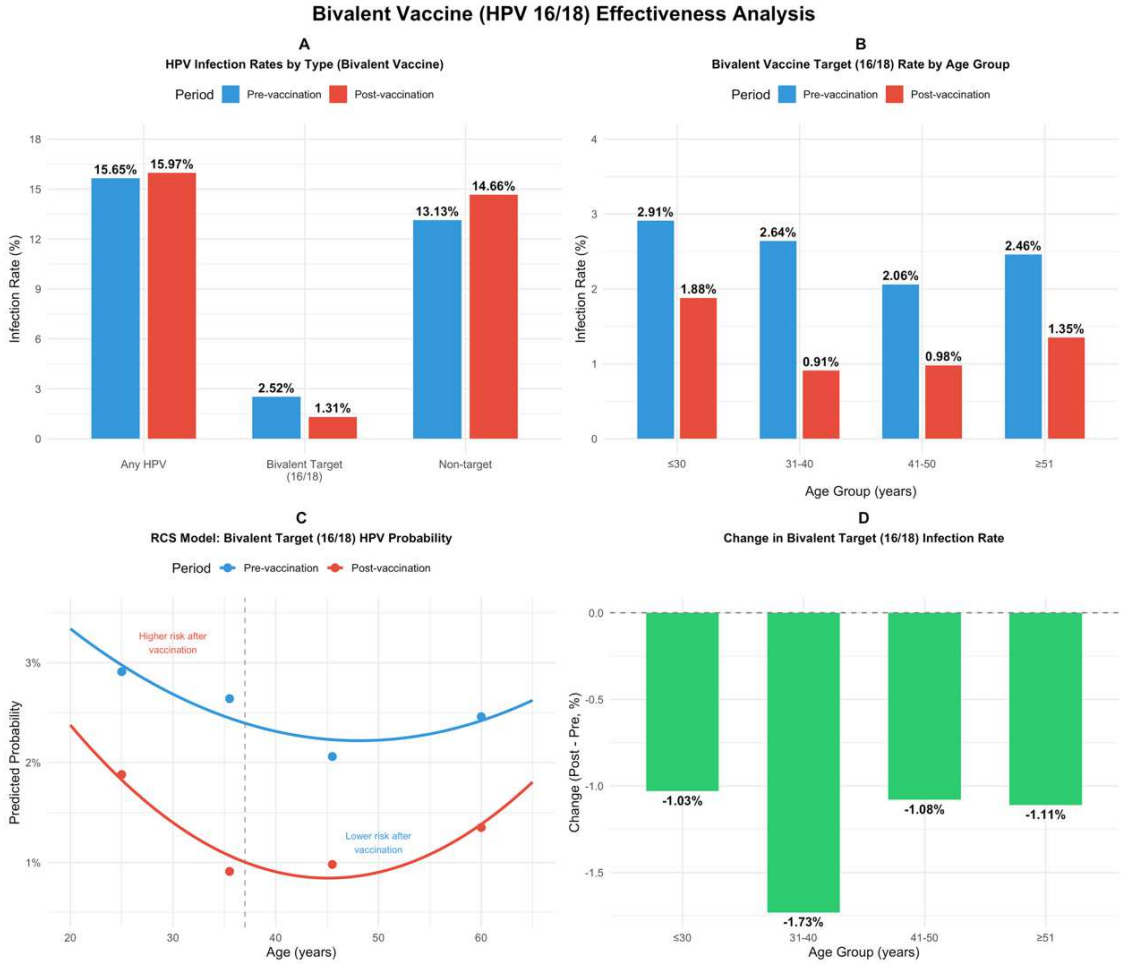


Figure 3. Bivalent HPV vaccine effectiveness analysis

(A) Overall infection rates for any HPV, bivalent vaccine target types (HPV16/18), and non-target types in pre-vaccination (blue) and post-vaccination (red) periods.

(B) Age-specific infection rates of bivalent target types across four age groups.

(C) RCS models showing predicted probability of bivalent target HPV infection by age. Shaded areas represent 95% confidence intervals.

(D) Changes in bivalent target infection rates (post minus pre) by age group.

2.5 Nine-valent vaccine target types analysis

For the 9-valent vaccine (types 16, 18, 31, 33, 45, 52, 58, 6, 11), targeted-type

prevalence declined from 7.64% to 6.62% (-1.02; -13.4%), while non-targeted types rose from 8.01% to 9.35% (+1.34); overall positivity was unchanged (Figure 4A). Age-stratified analysis (Figure 4B, 4D) revealed greater heterogeneity than the bivalent. The largest reduction was in 31-40 (7.05% to 5.23%; -1.82; -25.8%), then 41-50 (6.80% to 5.59%; -1.21; -17.8%). The >=51 group changed minimally (8.04% to 8.00%; -0.04 pp), while <=30 increased paradoxically (7.83% to 9.25%; +1.42; +18.1%). RCS (Figure 4C) showed a more pronounced U-shape post-vaccination, with probability at 20 years rising (~7% to ~9.25%), and decreasing in the 35-50 range.

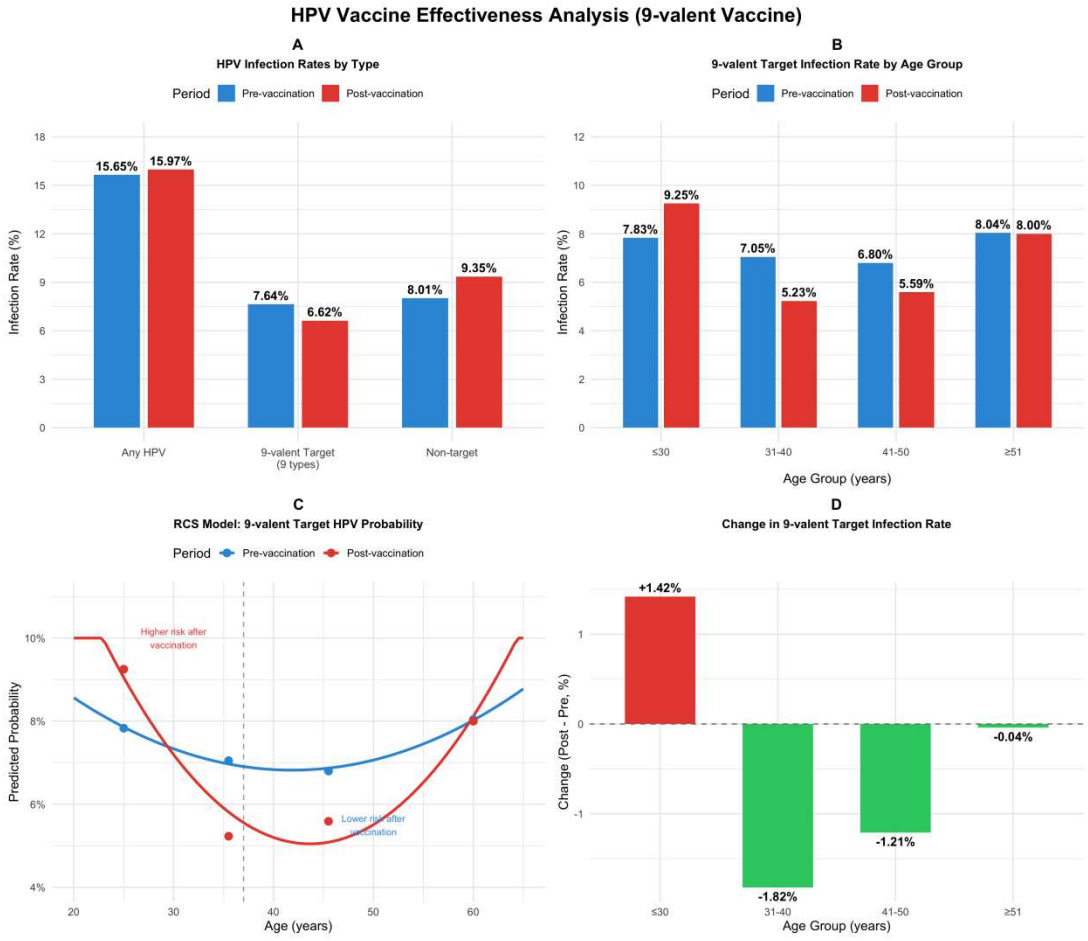


Figure 4. Nine-valent vaccine target types analysis

(A) Overall infection rates for any HPV, nine-valent vaccine target types (9 types: 6,11,16,18,31,33,45,52,58), and non-target types in pre-vaccination (blue) and post-vaccination (red) periods.

(B) Age-specific infection rates of nine-valent target types across four age groups.

(C) Restricted cubic spline (RCS) models showing predicted probability of nine-valent target HPV

infection by age. Shaded areas represent 95% confidence intervals.
(D) Changes in nine-valent target infection rates (post minus pre) by age group. Positive values indicate increased risk after vaccination; negative values indicate decreased risk.

Discussion

In this 11-year study of 29,041 women, vaccination in Fuzhou produced three patterns: (1) a persistent U-shaped age distribution; (2) significant declines in HPV16/18 across all ages, confirming real-world vaccine effectiveness[9]. and (3) marked increases in low-risk and non-vaccine types-especially HPV81, HPV6, HPV44-most pronounced in ⩽30, suggesting possible type replacement warranting surveillance.

Age-specific distribution of HPV infection

The U-shaped prevalence-peaks at ⩽30 (15.32-18.03%) and ⩾51 years (17.79-19.72%), nadir at 31-40 (11.81-14.37%)-matches the bimodal pattern in large surveys[10-12]. The early peak reflects high incidence with rapid clearance after sexual debut[9, 13]. the late peak reflects immunosenescence and accumulated persistent infection [10, 11]. These support age-adapted prevention: vaccination and education for younger women, and risk-based continued screening rather than cessation at 65.

Age-specific patterns of vaccine response

The age-dependent hierarchy of vaccine response was reproducible across both the bivalent and 9-valent vaccines (Figures 3-4). The 31-40 age cohort-occupying the nadir of the U-shaped prevalence curve-consistently derived the largest absolute benefit from both vaccines (bivalent HPV16/18: -1.73%; 9-valent targeted types: -1.82%). This concordance argues against confounding and supports a causal interpretation of the observed reductions.

In contrast, the youngest (≤30) and oldest (≥51) groups exhibited attenuated or divergent responses. For the bivalent vaccine, the ≤30 group showed the smallest reduction (-1.03%), while the same age group paradoxically increased (+1.42%) in the 9-valent vaccine population. For the ≥51 group, the bivalent vaccine yielded a substantial decline (-1.11%), whereas the 9-valent vaccine showed no measurable change (-0.04%). The blunted response in young women reflects ongoing exposure dynamics: this age group experiences the highest incidence of new infections, and continuous incident cases may offset vaccine-induced reductions at the population

level[14, 15]. The overall crude efficacy differed between the two formulations (bivalent: 48.0% vs. 9-valent: 13.4%), attributable to differences in type composition, baseline prevalence, and phased rollout.

Interpretation of Vaccine-Targeted Genotype Alterations

This study examined genotype-specific changes after bivalent/ nonavalent HPV vaccination. For HPV16/18, vaccination was linked to sustained prevalence reduction. HPV18 declined most, with RR=0.20 in 31-40, and also significantly in 41-50 and ≥51. HPV16 had consistent significant reductions across all older groups (RR 0.48-0.61). HPV33 also declined in 41-50 and ≥51, consistent with bivalent cross-protection [16]. In contrast, HPV52 showed no decline (RR≈1.0) across ages. It is highly prevalent in Asia (1.9-4.0%)[12], and large-scale studies have consistently identified HPV52 as the most common high-risk genotype (1.9% -4.0%)[17]. Lineage B predominates in Asia (89.0% vs. 0%-5.5% in other regions), associated with higher oncogenic potential[18]. Combined with its late inclusion in the nonavalent vaccine and distinct natural history, a population-level declining trend has been difficult to detect within the current observation window.

HPV6 is a low-risk genotype accounting for ~90% of genital wart cases and covered by quadrivalent and nonavalent vaccines[19]. It increased significantly in ≤30 (RR=4.24). Since bivalent vaccines (dominant) do not cover HPV6, the ecological niche from reduced HPV16/18 may be occupied by HPV6 via competition [20]. A recent Xiamen study found HPV6-HPV72 negative correlation (OR<0.01), supporting competition[21].

Persistent High Prevalence of Non-Vaccine-Covered HPV Types 53 and 81

In the present study, HPV81 exhibited a significant upward trend across most age strata (≤30, 41-50, and ≥51 years), with the most pronounced increase among women aged ≤30 years (RR = 3.17, 95% CI: 1.41-7.14). By contrast, HPV53 did not demonstrate a significant upward trend; however, it consistently maintained a relatively high prevalence throughout the study period, warranting continued concern. A Shanghai reported HPV53 (5.8%) and HPV81 (4.6%) detection in cervical specimens[22]. A Caribbean population-based study found HPV53 accounted for 9.1% of high-risk types [23], and a nationwide Chinese survey (2015-2025) ranked HPV53

third (1.67%), behind HPV52 and HPV16[10]. So et al.[24] observed HPV53 as the fourth most common genotype across healthy women and CIN/cancer samples, while a Yunnan study found HPV81 overrepresented in concurrent low/high-risk coinfections[25]. These findings reinforce the need for sustained surveillance and broad-spectrum vaccines.

Study limitations

Limitations: (1) the pre/post design cannot establish causality and lacked individual vaccination status; (2) single-center, though consistent with national data [9, 10]; (3) some age groups need larger samples for rare-type changes.

Conclusions

In conclusion, 11-year data show Fuzhou vaccination effectively reduced HPV16/18, but emergence of low-risk and non-vaccine types-especially HPV81, HPV6-in young women raise type-replacement concerns, underscoring continued surveillance, broader-spectrum vaccines, and age-specific screening.

The authors declare that they have no known competing financial interests or personal relationships that could have influenced the work reported herein.

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Declarations

Ethics approval and consent to participate

This study was approved by the Medical Ethics Committee of Fuzhou University Affiliated Provincial Hospital (Approval No. K2026-08-002). Because anonymized retrospective diagnostic data were used, the requirement for informed consent was waived by the committee. Patients and the public were not involved in the design, conduct, or reporting of this study.

Consent for publication

Not applicable. This manuscript does not contain any individual person's data in any form (including individual details, images, or videos).

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request, subject to approval by the Medical Ethics

Committee of Fuzhou University Affiliated Provincial Hospital.

Competing interests

The authors declare that they have no known competing financial interests or personal relationships that could have influenced the work reported herein.

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Authors' contributions

Pengju Cao and Binbin Zhuang contributed equally to this work. PC and HZ conceptualized the study. BZ and SC performed data curation and formal analysis. YT and YY conducted investigation and data acquisition. XS validated the methodology. SC wrote the original draft. PC, HZ, and XS reviewed and edited the manuscript. HZ and PC supervised the project and acquired funding. All authors read and approved the final manuscript.

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Abbreviations

HPV: Human papillomavirus

HR-HPV: High-risk human papillomavirus

LR-HPV: Low-risk human papillomavirus

RCS: Restricted cubic spline

RR: Rate ratio

CI: Confidence interval

IARC: International Agency for Research on Cancer

WHO: World Health Organization

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