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ORIGINAL ARTICLE

Prevalence of HPV Infection During and After the COVID-19 Pandemic in Zhangzhou, Fujian

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ABSTRACT

Background: **We assessed whether HPV prevalence and genotype distribution among women screened in Zhangzhou, Fujian, differed between the COVID-19 pandemic (2020 - 2022) and the post-pandemic period (2023 - 2024).**
Methods: **We analyzed routine HPV DNA testing results from 115,557 women and compared overall prevalence, single-type vs. multiple-type infections, and genotype patterns across age groups and time periods using standard statistical tests.**
Results: **Overall HPV prevalence was higher after the pandemic than during it. The most common high-risk types across the study were HPV52, HPV51, HPV58, HPV16, and HPV39; low-risk HPV81 became prominent in the post-pandemic period. Both single-type and multiple-type infections increased after the pandemic. The ≤ 24-year group had the highest prevalence, and prevalence also rose in women ≥ 55 years.**
Conclusions: **Our large single-center dataset suggests increased HPV detection after the pandemic, with stable dominance of East-Asia-relevant types (notably HPV52/58) and a shift toward higher detection of HPV81 post-pandemic. Because behavioral and vaccination data were not collected, we avoid causal attribution; observed differences may reflect changes in screening attendance, age mix, and community transmission. These findings support targeted screening and vaccination strategies in southern China.**
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INTRODUCTION

Human papillomavirus (HPV) is a small, double-stranded circular DNA virus with a strong affinity for epithelial tissues. It infects the skin as well as mucosal surfaces of the cervix, anus, oral cavity, vagina, vulva, and penis and is implicated in the development of both benign lesions and malignant tumors in these tissues [1]. To date, more than 200 HPV types have been identified and classified based on nucleotide sequence homology [2]. Out of these, 12 are recognized as high-risk oncogenic types by the International Agency for Research on Cancer (IARC). Globally, cervical cancer ranks as the fourth most common cancer among women, with an es-

timated 662,000 new cases and 349,000 related deaths in 2022, accounting for 6.8% of all female cancer diagnoses and 8.1% of cancer-related mortality in women [3]. According to the World Health Organization (WHO), more than 1 million new sexually-transmitted infections (STIs) occur daily worldwide, with HPV being the most prevalent causative agent [4]. In China, the epidemiology of HPV infection exhibits considerable regional variability, influenced by factors such as age, socioeconomic conditions, cultural norms, and population mobility. Among women, HPV infection and its progression to cervical cancer have attracted significant public health concern [5].

The COVID-19 pandemic, which emerged in late 2019, triggered substantial changes in human behavior and healthcare access. Nationwide lockdowns, restrictions on travel, and reduced social interactions during 2020 - 2022 disrupted the prevention and control of STIs and led to a sharp decline in cervical cancer screening. In this context, the present study aimed to assess the impact of the COVID-19 pandemic on HPV prevalence and genotype distribution among women in southern Fujian. By comparing data from the pandemic period (2020 - 2022) and the post-pandemic period (2023 - 2024), our findings provide insights to support the refinement of HPV prevention, control, and screening strategies in the post-pandemic era.

MATERIALS AND METHODS

Study population

A total of 115,557 female participants who underwent HPV DNA testing at Zhangzhou Affiliated Hospital of Fujian Medical University between January 2020 and December 2024 were included in this study. Participant ages ranged from 14 to 99 years, with a median age of 44 years. Among them, 40,590 women sought clinical care for gynecological conditions, while 75,783 underwent routine physical examinations. For individuals tested more than once during the study period, only the initial screening result was included; duplicate entries were excluded from analysis. This study was approved by the Institutional Medical Ethics Review Committee of Zhangzhou Affiliated Hospital of Fujian Medical University, China (approval no. 2025LWB265).

Participant categories (clarification) were the following: "Clinical patients" were women attending gynecology clinics for evaluation or management of gynecologic symptoms (e.g., abnormal bleeding or discharge, pelvic pain), abnormal screening results, or follow-up of known gynecologic conditions. "Healthy women" were attendees of routine health examinations without documented gynecologic disease. These definitions are now used consistently throughout the manuscript.

Data presentation note: Extremely detailed geno-type-level tables are not retained in the main text; key summaries are presented here. The full de-identified datasets are available from the corresponding author upon reasonable request.

Sample collection and HPV genotyping

Cervical exfoliated cell samples were collected from each participant using a disposable cytobrush. The specimens were placed in a specialized preservation buffer solution and kept at 2 - 8°C, with processing completed within 48 hours. Prior to DNA extraction, samples were thoroughly vortexed. Genomic DNA was extracted using a commercial nucleic acid extraction reagent according to the manufacturer's instructions. HPV genotyping was performed using the HPV37 Genotyping Kit (Chaozhou Kaipu Biochemical Co., Ltd., Chaozhou, China). PCR amplification was carried out on a Cobas Z480 fluorescent quantitative PCR instrument, and hybridization and signal detection were done using an HHM-3 flow-through hybridization system. This genotyping system can simultaneously detect 37 HPV types, including 18 intermediate/high-risk types (HPV16, 18, 26, 31, 33, 35, 39, 45, 51, 52, 53, 56, 58, 59, 66, 68, 73, 82) and 19 low-risk types (HPV6, 11, 34, 40, 42, 43, 44, 54, 55, 57, 61, 67, 69, 70, 71, 72, 81, 83, 84). All procedures were performed in accordance with the kit's instruction manual and standard laboratory protocols.

Statistical analysis

HPV test data were compiled and organized using Microsoft Excel. Participants were stratified by sample source (clinical patients vs. physical examination attendees), age group (≤ 24, 25 - 34, 35 - 44, 45 - 54, ≥ 55 years), and time period (pandemic: 2020 - 2022; post-pandemic: 2023 - 2024). The proportion of HPV-positive cases and corresponding 95% confidence intervals (CIs) were calculated for each subgroup. Differences in HPV prevalence between groups were assessed using the chi-squared (χ^2) test. Paired χ^2 tests were used to compare the distribution of HPV genotypes between groups. When expected cell counts in contingency tables were ≤ 5 or when p-values approached 0.05, Fisher's exact test was applied to ensure statistical robustness. All statistical tests were two-sided, and a p-value < 0.05 was considered statistically significant. Analyses were conducted using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA).

RESULTS

HPV prevalence before and after the pandemic

A total of 115,557 women were included in this study, out of whom 19,504 tested positive for HPV DNA, resulting in an overall infection rate of 16.9% (95% CI: 16.7 - 17.1). During the COVID-19 pandemic period (2020 - 2022), 56,018 women were tested, and 7,801 (13.9%, 95% CI: 13.6 - 14.2) were HPV-positive. In the post-pandemic period (2023 - 2024), 59,539 women were tested, and 11,703 (19.7%, 95% CI: 19.3 - 20.0) tested positive. This increase in infection rate was statistically significant ($\chi^2 = 675.494$, $p < 0.01$) (Table 1).

Table 1. Prevalence of HPV infection and genotypes during and after the pandemic in this study.

Variables	Total (n = 115,557)		During the pandemic (n = 56,018)		Post-pandemic (n = 59,539)		χ^2	p-value
	No.	Prevalence % (95% CI)	No.	Prevalence % (95% CI)	No.	Prevalence % (95% CI)		
Any type	19,504	16.9 (16.7 - 17.1)	7,801	13.9 (13.6 - 14.2)	11,703	19.7 (19.3 - 20.0)	675.494	< 0.001
Single	14,036	12.1 (12.0 - 12.3)	5,782	10.3 (10.1 - 10.6)	8,254	13.9 (13.6 - 14.1)	339.235	< 0.001
Multiple	5,468	4.7 (4.6 - 4.9)	2,019	3.6 (3.4 - 3.8)	3,449	5.8 (5.6 - 6.0)	306.692	< 0.001
HPV52	3,751	3.2 (3.1 - 3.3)	1,574	2.8 (2.7 - 2.9)	2,177	3.7 (3.5 - 3.8)	65.87	< 0.001
HPV51	1,930	1.7 (1.6 - 1.7)	571	1.0 (0.9 - 1.1)	1,359	2.3 (2.2 - 2.4)	280.444	< 0.001
HPV58 *	1,835	1.6 (1.5 - 1.7)	921	1.6 (1.5 - 1.7)	914	1.5 (1.4 - 1.6)	2.194	0.139
HPV16 *	1,684	1.5 (1.4 - 1.5)	820	1.5 (1.4 - 1.6)	864	1.4 (1.4 - 1.5)	0.032	0.857
HPV39	1,590	1.4 (1.3 - 1.4)	671	1.2 (1.1 - 1.3)	919	1.5 (1.4 - 1.6)	25.418	< 0.001
HPV81	1,451	1.3 (1.2 - 1.3)	510	0.9 (0.8 - 1.0)	941	1.6 (1.5 - 1.7)	104.513	< 0.001
HPV53	1,367	1.2 (1.1 - 1.2)	750	1.3 (1.2 - 1.4)	617	1.0 (1.0 - 1.1)	22.602	< 0.001
HPV61	1,363	1.2 (1.1 - 1.2)	506	0.9 (0.8 - 1.0)	857	1.4 (1.3 - 1.5)	71.17	< 0.001
HPV54	1,046	0.9 (0.9 - 1.0)	441	0.8 (0.7 - 0.9)	605	1.0 (0.9 - 1.1)	16.858	< 0.001
HPV68	944	0.8 (0.8 - 0.9)	324	0.6 (0.5 - 0.6)	620	1.0 (1.0 - 1.1)	76.346	< 0.001
HPV42	868	0.8 (0.7 - 0.8)	130	0.2 (0.2 - 0.3)	738	1.2 (1.2 - 1.3)	392.948	< 0.001
HPV18	778	0.7 (0.6 - 0.7)	416	0.7 (0.7 - 0.8)	362	0.6 (0.5 - 0.7)	7.821	0.005
HPV70	761	0.7 (0.6 - 0.7)	290	0.5 (0.5 - 0.6)	471	0.8 (0.7 - 0.9)	32.974	< 0.001
HPV84	743	0.6 (0.6 - 0.7)	187	0.3 (0.3 - 0.4)	556	0.9 (0.9 - 1.0)	162.657	< 0.001
HPV44	703	0.6 (0.6 - 0.7)	186	0.3 (0.3 - 0.4)	517	0.9 (0.8 - 0.9)	137.291	< 0.001

*** indicates no statistical significance between the pandemic group and the post-pandemic group. This table only shows the genotypes with the top 15 prevalence rates. For complete data, please contact the corresponding author.**

Among all participants, 14,036 (12.1%, 95% CI: 12.0 - 12.3) had single-type HPV infections. During the pandemic, 5,782 cases (10.3%, 95% CI: 10.1 - 10.6) were identified, compared to 8,254 cases (13.9%, 95% CI: 13.6 - 14.1) in the post-pandemic period - a significant increase (χ^2 = 339.235, $p < 0.01$). In addition, 5,468 women (4.7%, 95% CI: 4.6 - 4.9) were infected with multiple HPV genotypes. This included 2,019 cases (3.6%, 95% CI: 3.4 - 3.8) during the pandemic and 3,449 cases (5.8%, 95% CI: 5.6 - 6.0) post-pandemic, indicating a statistically significant rise in multiple-type infections (χ^2 = 306.692, $p < 0.01$).

HPV prevalence in clinical patients vs. health check-up populations

Out of the total study cohort, 39,255 women were hospital patients (outpatients or inpatients), while 76,302 were attendees of routine health examinations. HPV DNA was detected in 9,126 patient samples (23.2%, 95% CI: 22.8 - 23.7) and in 10,378 samples from the health check-up population (13.6%, 95% CI: 13.4 - 13.8), showing a significant difference in infection rates

Table 2. Prevalence of HPV in patients and healthy individuals in this study.

Variables	Total (n = 115,557)		Patients (n = 39,255)		Healthy women (n = 76,302)		χ^2	p-value
	No.	Prevalence % (95% CI)	No.	Prevalence % (95% CI)	No.	Prevalence % (95% CI)		
Any type	19,504	16.9 (16.7 - 17.1)	9,126	23.2 (22.8 - 23.7)	10,378	13.6 (13.4 - 13.8)	1,719.321	< 0.001
Single	14,036	12.1 (12.0 - 12.3)	6,299	16.0 (15.7 - 16.4)	7,737	10.1 (9.9 - 10.4)	847.369	< 0.001
Multiple	5,468	4.7 (4.6 - 4.9)	2,827	7.2 (6.9 - 7.5)	2,641	3.4 (3.3 - 3.6)	804.428	< 0.001
HPV52	3,751	3.2 (3.1 - 3.3)	1,779	4.5 (4.3 - 4.7)	1,972	2.6 (2.5 - 2.7)	312.999	< 0.001
HPV51	1,930	1.7 (1.6 - 1.7)	857	2.2 (2.0 - 2.3)	1,073	1.4 (1.3 - 1.5)	95.264	< 0.001
HPV58	1,835	1.6 (1.5 - 1.7)	987	2.5 (2.4 - 2.7)	848	1.1 (1.0 - 1.2)	326.464	< 0.001
HPV16	1,684	1.5 (1.4 - 1.5)	974	2.5 (2.3 - 2.6)	710	0.9 (0.9 - 1.0)	434.03	< 0.001
HPV39	1,590	1.4 (1.3 - 1.4)	716	1.8 (1.7 - 2.0)	874	1.1 (1.1 - 1.2)	87.939	< 0.001
HPV81	1,451	1.3 (1.2 - 1.3)	664	1.7 (1.6 - 1.8)	787	1.0 (1.0 - 1.1)	91.084	< 0.001
HPV53	1,367	1.2 (1.1 - 1.2)	686	1.7 (1.6 - 1.9)	681	0.9 (0.8 - 1.0)	162.108	< 0.001
HPV61	1,363	1.2 (1.1 - 1.2)	629	1.6 (1.5 - 1.7)	734	1.0 (0.9 - 1.0)	91.192	< 0.001
HPV54	1,046	0.9 (0.9 - 1.0)	490	1.2 (1.1 - 1.4)	556	0.7 (0.7 - 0.8)	78.006	< 0.001
HPV68	944	0.8 (0.8 - 0.9)	446	1.1 (1.0 - 1.2)	498	0.7 (0.6 - 0.7)	74.782	< 0.001
HPV42	868	0.8 (0.7 - 0.8)	352	0.9 (0.8 - 1.0)	516	0.7 (0.6 - 0.7)	16.895	< 0.001
HPV18	778	0.7 (0.6 - 0.7)	428	1.0 (1.0 - 1.2)	350	0.5 (0.4 - 0.5)	154.623	< 0.001
HPV70	761	0.7 (0.6 - 0.7)	315	0.8 (0.7 - 0.9)	446	0.6 (0.5 - 0.6)	18.816	< 0.001
HPV84	743	0.6 (0.6 - 0.7)	355	0.9 (0.6 - 0.8)	388	0.5 (0.4 - 0.5)	63.574	< 0.001
HPV44	703	0.6 (0.6 - 0.7)	291	0.7 (0.7 - 0.8)	412	0.5 (0.5 - 0.6)	17.379	< 0.001

This table only shows the genotypes with the top 15 prevalence rates. For complete data, please contact the corresponding author.

between the two groups (χ^2 = 1719.321, $p < 0.01$). The rate of single-type infection was higher among clinical patients than in the health check-up group (16.0% vs. 10.1%), as was the rate of multiple-type infections (7.2% vs. 3.4%) (both $p < 0.01$). In terms of genotype distribution, the five most prevalent HPV types among hospital patients were HPV52, HPV58, HPV16, HPV51, and HPV39. For the health check-up population, the top five were HPV52, HPV51, HPV39, HPV58, and HPV81. Each of these genotypes showed significantly different prevalence between the two groups ($p < 0.01$ for all comparisons).

Genotypic distribution of HPV during vs. after the pandemic

A total of 37 HPV genotypes were detected in the study population, including 18 intermediate/high-risk types and 19 low-risk types. Across all time periods, the five most prevalent intermediate/high-risk types were HPV 52, HPV51, HPV58, HPV16, and HPV39. The most common low-risk genotypes were HPV81, HPV61, HPV54, HPV42, and HPV70. During the pandemic, the top five genotypes were HPV52, HPV58, HPV16, HPV 53, and HPV39. In the post-pandemic period, the distribution shifted to HPV52, HPV51, HPV81, HPV39,

Table 3. The infection rates and genotype distributions of HPV in age-specifc groups.

	≤ 24 years n (%)	25 - 34 n (%)	35 - 44 n (%)	45 - 54 n (%)	≥ 55 n (%)	χ^2	p-value
Sample size	2,281	23,134	31,924	37,943	20,275	-	-
Any type	713 (31.3) [a]	3,610 (15.6) [b]	4,866 (15.2) [b]	5,977 (15.8) [b]	4,338 (21.4) [c]	753.038	< 0.001
Single	402 (17.6) [a]	2,690 (11.6) [b]	3,733 (11.7) [b]	4,504 (11.9) [b]	2,707 (13.4) [c]	106.397	< 0.001
Multiple	311 (13.6) [a]	920 (4.0) [b]	1,133 (3.5) [b]	1,473 (3.9) [b]	1,631 (8.0) [c]	1,083.641	< 0.001
HPV16	100 (4.4) [a]	301 (1.3) [b]	382 (1.2) [b]	466 (1.2) [b]	435 (2.1) [c]	235.838	< 0.001
HPV18	56 (2.5) [a]	165 (0.7) [b, c]	157 (0.5) [d]	214 (0.6) [c, d]	186 (0.9) [b]	149.407	< 0.001
HPV26	2 (0.1) [a]	3 (0.0) [a]	9 (0.0) [a]	10 (0.0) [a]	13 (0.1) [a]	11.884	0.018
HPV31	18 (0.8) [a]	82 (0.4) [b]	114 (0.4) [b]	116 (0.3) [b]	142 (0.7) [a]	64.170	< 0.001
HPV33	24 (1.1) [a]	85 (0.4) [b]	94 (0.3) [b]	160 (0.4) [b]	173 (0.9) [a]	109.658	< 0.001
HPV35	13 (0.6) [a]	33 (0.1) [b]	48 (0.2) [b]	73 (0.2) [b]	98 (0.5) [a]	87.309	< 0.001
HPV39	75 (3.3) [a]	316 (1.4) [b]	396 (1.2) [b]	437 (1.2) [b]	366 (1.8) [c]	107.375	< 0.001
HPV45	10 (0.4) [a, b]	32 (0.1) [c]	42 (0.1) [c]	80 (0.2) [b, c]	68 (0.3) [a]	37.093	< 0.001
HPV51	103 (4.5) [a]	405 (1.8) [b, c]	480 (1.5) [c, d]	521 (1.4) [d]	421 (2.1) [b]	159.525	< 0.001
HPV52	154 (6.8) [a]	704 (3.0) [b]	886 (2.8) [b]	1,070 (2.8) [b]	937 (4.6) [c]	258.850	< 0.001
HPV53	44 (1.9) [a]	229 (1.0) [b]	318 (1.0) [b]	423 (1.1) [b]	353 (1.7) [a]	83.301	< 0.001
HPV56	28 (1.2) [a]	106 (0.5) [b]	127 (0.4) [b]	188 (0.5) [b]	188 (0.9) [a]	90.832	< 0.001
HPV58	94 (4.1) [a]	358 (1.5) [b]	395 (1.2) [c]	500 (1.3) [b, c]	488 (2.4) [d]	223.750	< 0.001
HPV59	35 (1.5) [a]	141 (0.6) [b, c]	158 (0.5) [b, c]	189 (0.5) [c]	138 (0.7) [b]	48.904	< 0.001
HPV66	25 (1.1) [a]	77 (0.3) [b]	86 (0.3) [b]	110 (0.3) [b]	103 (0.5) [c]	61.476	< 0.001

Each superscript letter (a, b, c, d) denotes a subset of age categories whose column proportions show no significant difference from each other at the 0.05 level, based on post-hoc pairwise comparisons with Bonferroni correction.
This table only shows the genotypes with the top 15 prevalence rates. For complete data, please contact the corresponding author.

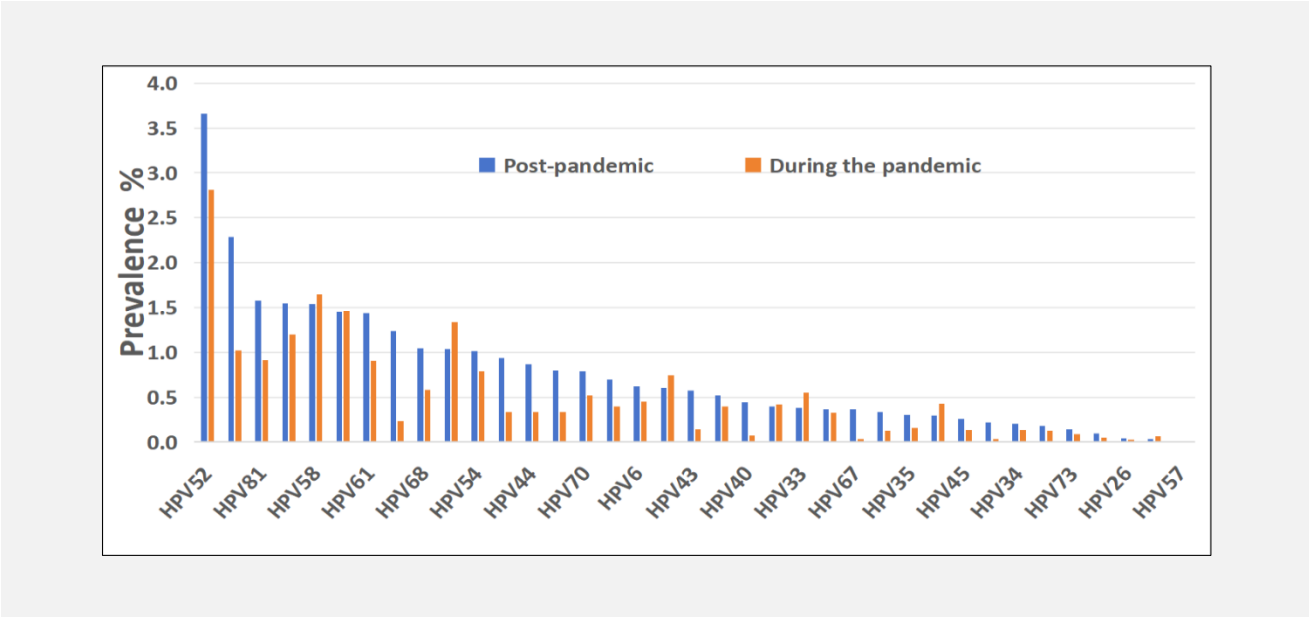

Figure 1. The prevalence of HPV genotypes during and after the pandemic in this study.

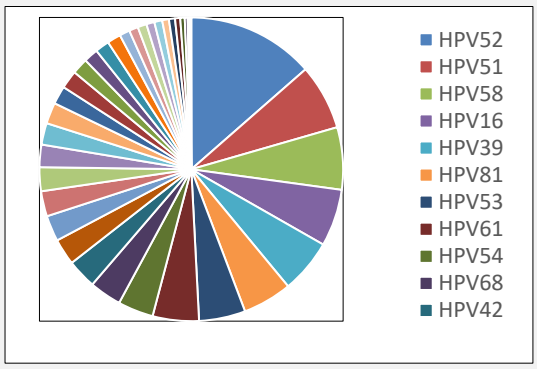

Figure 2. The distribution of HPV genotypes in this study.

and HPV58. Except for HPV58 and HPV16, the prevalence of these dominant genotypes differed significantly between the two periods (Table 1, Figure 1).

HPV prevalence by age group (overall 2020 - 2024)

Participants were stratified into five age groups: ≤ 24 years, 25 - 34 years, 35 - 44 years, 45 - 54 years, and ≥ 55 years. The majority of women were concentrated in the 35 - 44- and 45 - 54-year groups. However, the highest HPV positivity rate was observed in the youngest group (≤ 24 years), with 713 positive cases and a prevalence of 31.3%. The second-highest rate was found in the oldest group (≥ 55 years), with 4,338 positive cases and a prevalence of 21.4%. Notably, the ≤ 24-year group also exhibited the highest proportion of multiple-type infections (13.6%), whereas single-type infections predominated in the other age categories. All differences in infection rates among age groups were statistically significant (Table 2). When genotype distribution was analyzed across all ages, the five most prevalent types were HPV52 (13.56% of positive cases), HPV51 (6.98%), HPV58 (6.63%), HPV16 (6.09%), and HPV39 (5.75%) (Figure 2).

Age-specific HPV prevalence during vs. after the pandemic

In both the pandemic and post-pandemic periods, the ≤ 24-year age group consistently had the highest HPV infection rate: 29.7% during the pandemic and 32.7% post-pandemic, a difference that was not statistically significant. In contrast, the infection rate in the ≥ 55-year group increased significantly from 17.5% to 24.4% between the two periods. All other age groups (25 - 34, 35 - 44, 45 - 54 years) also experienced statistically significant increases in HPV prevalence following the pandemic. Similarly, both single-type and multiple-type infection rates rose in each age group after the pandemic, with the exception of the ≤ 24 group, where the increase was not significant. Detailed statistics for each age group are provided in Table 3.

DISCUSSION

Pre-COVID-19 studies identified marked regional variations in HPV prevalence and genotype distribution across China [6,7]. Northern provinces (e.g., Shanxi: 8.92%; Liaoning: 10.3%) had lower infection rates, while western/central regions (e.g., Sichuan: 12.6 - 15.29%; Henan: 12.09%) showed moderately higher, variable rates. Southern provinces (Guangdong, Fujian, Guangxi) reported the highest national rates (16 - 19.5%). Our study in Zhangzhou (southern Fujian) aligned with this trend, with an overall HPV prevalence of 16.9% during 2020 - 2024. Notably, rates were significantly lower during the pandemic (2020 - 2022) vs. post-pandemic (2023 - 2024) - consistent with central Fujian data [19] - likely due to reduced population mobility and diminished healthcare-seeking/screening behaviors.

HPV genotypes also exhibited regional specificity. Nationally prevalent types included HPV16, 39, 51, 52, 53, 56, 58, with HPV66 more common in northern China [21]. In western regions (Sichuan, Shaanxi), HPV53 ranked top three [14]; central Fujian was dominated by HPV52, 53, 58, 16, 51 [19]. Our southern Fujian findings were broadly consistent, though HPV39 replaced HPV53 in the top five. A 2022 Putian (Fujian) study reported a distinct profile (HPV52, 58, 16, 18, 33) [22], underscoring regional variability. Clinical patients had a significantly higher HPV rate (23.2%) than asymptomatic individuals (13.6%) - consistent with other regions (e.g., central Fujian: 19.0% [19]; Shanghai: 17.92% [23]).

Pandemic-related genotype shifts were observed: during the pandemic, dominant types were HPV52, 58, 16, 53,

39 (all intermediate/high-risk); post-pandemic, distributions shifted to HPV52, 51, 81, 39, 58, with low-risk HPV81 emerging as third most prevalent. These changes may relate to behavioral shifts or healthcare disruptions. HPV52/58 dominance aligns with East/Southeast Asian studies [28,33]. This study did not collect data on sexual behavior or vaccination, so causality cannot be attributed to any specific factor. Therefore, we interpret genotype-specific changes (such as the increased detection rate of HPV81 after the pandemic) with caution.

Age-stratified analysis showed highest rates in the youngest (≤ 24 years: 31.3%) and oldest (≥ 55 years: 21.4%) groups - consistent with prior reports [19,29, 30]. Younger women's higher rates may link to early sexual activity, inconsistent protection, and immature immunity [31]; older women's rates may reflect age-related immune decline and menopausal changes [32]. Underrepresentation of these groups in our sample suggests potential underreporting, emphasizing targeted screening needs.

Across all ages, top genotypes were HPV52, 51, 16, 58, 39. HPV52/58 are prevalent in East Asians and linked to cervical lesions [28]; HPV16, strongly associated with high-grade lesions/cancer [34], requires vigilance for persistent infections. All age groups had lower pandemic-era rates (single/multiple infections), likely due to reduced contact, public health measures (social distancing, masking), and limited routine care access. Redirected COVID-19 resources and restricted non-urgent visits may have also delayed asymptomatic infection detection.

While the temporary decline in HPV incidence may have reduced short-term cervical cancer risk, it also highlights the potential for delayed diagnoses of precancerous lesions and a backlog in screening services. Thus, post-pandemic recovery must include enhanced HPV surveillance and efforts to catch up on missed vaccinations and screenings. Expanding HPV vaccine coverage, particularly by broadening age eligibility, alongside routine cervical cancer screening, is vital to reversing the resurgence in HPV infections. Our study has several limitations. First, the genotyping panel was not exhaustive, and certain rare HPV types were not assessed. Second, fewer women were tested during the pandemic period, which may have introduced selection bias or affected statistical power in comparisons. Third, the absence of histopathological data (e.g., cytology or biopsy results) limits our ability to assess clinical outcomes. Finally, the study lacked follow-up information, and as a retrospective analysis based on laboratory records, it may have missed infections in women who did not seek care. Future prospective studies with comprehensive genotyping, longitudinal follow-up, and integration of clinical outcome data are needed to better characterize HPV epidemiology in the post-pandemic era and to inform precision prevention strategies.

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Data Availability Statement:
The de-identified genotype-level tables and underlying datasets are available from the corresponding author on reasonable request.

Ethical Approval Statement:
This study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by the Ethics Committee of Zhangzhou Affiliated Hospital of Fujian Medical University.

Consent to Participate:
Informed consent was obtained from all individual participants included in the study.

Consent to Publish:
The authors affirm that human research participants provided informed consent for publication of the images in Figures. 1 - 2.

Declaration of Interest:
The authors have no relevant financial or non-financial interests to disclose.

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