Original Article

SAGE Open Medicine

SAGE Open Medicine
Volume 13: 1–7

DOI: 10.1177/20503121251320522
journals.sagepub.com/home/smo
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Prevalence and genotype distribution of human papillomavirus infection among Kurdish people, Iran

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Abstract

Background and aim: Human papillomavirus is a common sexually transmitted infection and a health concern, being the major cause of cervical cancer and genital warts. The aim of this study was to investigate the prevalence and genotype distribution of human papillomavirus in the Kurdish population.

Methods and materials: A total of 2650 samples from individuals attending central laboratories in Sanandaj, the center of Kurdistan Province in north-west Iran, were tested for human papillomavirus infection. Cervical samples were collected from women using a cervical brush, while semen samples were collected from men. Cervical samples were stored in a vial with cell preservation solution, while semen samples were stored at room temperature until complete liquefaction of the semen. Human papillomavirus DNA from positive samples was extracted using a high-purity viral nucleic acid kit, and human papillomavirus genotyping was performed using the ZYTOVISION human papillomavirus Chip1.0 genotyping system according to the manufacturer's instructions.

Results: Of the 2610 samples screened, 655 were found positive for human papillomavirus and included in the human papillomavirus molecular typing analysis. The overall prevalence of human papillomavirus in the study samples was 25.1%. Of the 655 positive samples, 645 (98.5%) were women and 10 (1.5%) were men. The mean age was 35.95 years (women: 36.06 years and men: 28.81 years). The most common genotypes identified were types 6 (30.1%), 16(14.4%), and 54(9.0%). Of those affected, 387 (59.1%) had a single human papillomavirus infection and 268 (40.9%) had multiple infections.

Conclusion: This study showed that the prevalence of human papillomavirus among Kurdish women is relatively high. The findings highlight the importance of targeted human papillomavirus vaccination programs and screening strategies, particularly in younger age groups, to reduce the burden of human papillomavirus-related disease in this setting. Future studies should investigate the impact of cultural and behavioral factors on human papillomavirus transmission and associated health outcomes in this population.

Keywords

Human papilloma virus, molecular typing, type distribution, prevalence

Date received: 12 November 2024; accepted: 29 January 2025

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Introduction

Human papillomavirus (HPV) is a DNA virus that infects the cells of the body's squamous epithelium, leading to conditions such as skin warts, anogenital warts, and various types of cancer.[1] With over 200 types identified, only a few poses significant health risks. The high-risk genotypes (HRGs), including HPV-16, HPV-18, and others, can potentially lead to serious complications, while the low-risk genotypes (LRGs) pose a lower risk.[2] HPV is transmitted mainly through contact with infected genitalia, mucous membranes, or body fluids, often during sexual activity.[3] HPV as a health problem in the world has several risk factors such as age (adolescents and young adults), multiple sexual partners, weakened immune systems, damaged skin, and personal contact.[4] Among the health problems associated with HPV, cervical cancer is a major threat, particularly in Asia, Africa, and Latin America, where it is responsible for a significant number of deaths.[5] Persistent infection with high-risk HPV types is responsible for the majority of cervical cancers.[6] To address these challenges, six licensed HPV vaccines are currently available, all of which provide protection against HPV-16 and HPV-18, which are responsible for the majority of cervical cancers worldwide.[7,8]

The prevalence of HPV in Iranian population is high. In a large study conducted on 12,076 Iranian women in 2021, the prevalence of HPV was reported to be 38.7%.[9] Despite the global significance of HPV-related diseases, little research has been conducted in the Kurdish population, an area at high risk for HPV.[5,10] In some studies using the carrageenan as a secondary preventive strategy for HPV are outlined,[11] while there are no any evidence for using the carrageenan in our culture. It is clear that the most important strategy for the prevention of HPV-related diseases in both men and women is vaccination.[12] Until the time of this study, there is no specific national program for widespread vaccination against HPV in Iran and Kurdistan. Therefore, this study aims to investigate the prevalence and genotypic distribution of HPV in the Kurdish population. The results will contribute to early screening efforts, inform decisions regarding universal vaccination, and help select appropriate vaccines to prevent cervical cancer in the Kurdish population.

Material and methods

Study design, setting, and participants

This was a cross-sectional prevalence study conducted in the Kurdish population, Kurdistan Province, in northwestern Iran from September 2020 to February 2023. Cervical specimens from 2650 individuals attending the central laboratories in Sanandaj, the center of Kurdistan Province, were screened for HPV infection. Of the 2610 specimens, 655 were screened positive for HPV infection and submitted for HPV molecular typing. Inclusion criteria were (1) age range from 18 to 75 years; (2) all participating patients were selected based on the diagnosis of a specialist physician to perform an HPV test and refer to laboratories and diagnostic centers in Sanandaj; (3) no history of sexual life or vaginal medication in the past 1 week. All procedures were performed in accordance with relevant guidelines and standard operating procedures.

Ethical consideration

The proposal of this study was reviewed and approved by the ethics committee of Kurdistan University of Medical Sciences (ethics code: IR.MUK.REC.1399.247). Written informed consent was obtained from each participant prior to data collection and sample collection. Anonymity of the participants was considered in all stages of data collection.

Data collection

The demographic data of each participant were recorded in a checklist, and a unique code was allocated to each participant before the data gathering. This special code was written on the checklist and samples. The cervical samples were taken by gynecologists using a cervical brush. Vaginal secretions were wiped with a cotton swab, and the cervical brush, rotated 4–5 times, was inserted into the cervix to obtain a sufficient amount of cervical epithelial cells. The brush was then placed in a vial containing cell preservation solution (Sigma). Cervical samples were collected at room temperature and sent immediately to the PCR departments of the Cellular and Molecular Research Center. For men, semen samples were collected in a private room and kept at room temperature until complete liquefaction. After liquefaction, the semen samples were sent to the PCR departments of the Cellular and Molecular Research Centre for PCR. Approximately, 1 mL of each sample was collected for HPV DNA extraction. All extracted samples were stored at −20°C until use.

DNA extraction

Cell-containing samples (cervical smears and semen) were eluted; the eluate was transferred to a 1.5 mL centrifuge tube, centrifuged at 13,000 rpm for 10 min, the supernatant was discarded, and DNA was then extracted from the cell pellets using a high-purity viral nucleic acid kit (Roche Diagnostics GmbH, Mannheim, Germany) according to the manufacturer's instructions.

HPV DNA genotyping

The DNA genomic is first extracted from the samples using the kit E.Z.N.A DNA isolation (product of the US Omega Bio-tek Company). The quantity and quality of the extracted DNA is investigated by the use of Thermo Fisher Scientific (NanoDrop Technologies, Wilmington, DE, USA). HPV

Table 1. Human papilloma virus genotypes distribution frequency.

HPV type	*N* (%)	HPV type	*N* (%)	HPV type	*N* (%)	HPV type	*N* (%)
HPV-6	197 (30.1)	HPV-43	15 (2.3)	HPV-58	21 (3.2)	HPV-81	8 (1.2)
HPV-11	51 (7.8)	HPV-44	12 (1.8)	HPV-59	12 (1.8)	HPV-82	10 (1.5)
HPV-16	94 (14.4)	HPV-45	21 (3.2)	HPV-60	3 (0.5)	HPV-83	5 (0.8)
HPV-18	52 (7.9)	HPV-49	2 (0.3)	HPV-61	10 (1.5)	HPV-84	33 (5.0)
HPV-31	16 (2.4)	HPV-51	19 (2.9)	HPV-62	31 (4.7)	HPV-86	2 (0.3)
HPV-33	17 (2.6)	HPV-52	12 (1.8)	HPV-66	20 (3.1)	HPV-90	30 (4.9)
HPV-34	19 (2.9)	HPV-53	32 (4.9)	HPV-67	19 (2.9)	HPV-91	47 (7.2)
HPV-35	47 (7.2)	HPV-54	59 (9.0)	HPV-68	53 (8.1)	HPV-92	1 (0.2)
HPV-39	19 (2.9)	HPV-55	7 (1.1)	HPV-70	11 (1.7)		
HPV-40	13 (2.0)	HPV-56	43 (6.6)	HPV-73	14 (2.1)		
HPV-42	26 (4.0)	HPV-57	2 (0.3)	HPV-78	1 (0.2)		

genotyping was performed for all positives according to the manufacturer's instructions. ZYTOVISION HPV Chip1.0 is used for the qualitative detection and genotyping of HPV. This kit is designed to detect 41 different genotypes of HPV at the same time.

Biotinylated consensus primers (SPF10) were used to amplify a 65-bp region within the L1 ORF of several HPV types and based on reverse line-blot hybridization. A poly(dT) tail was enzymatically added to the 3′ end of each of 41 oligonucleotides specific for 41 different HPV types and capable of identification by detection of part of the L1 region of HPV by SPF-10 primers. Amplification of the human major histocompatibility complex class II gene was used to monitor sample quality and efficiency of DNA extraction from equal volumes.

Statistical analysis

Data were summarized using descriptive statistics such as mean, standard deviation, frequencies, and percentages. Chi-squared test and Fisher's exact test were used to assess the association of categorical variables. The normality assumption of age was tested by Kolmogorov–Smirnov, which was not met. Differences in the mean age of patients were compared using the Mann–Whitney *U* test. A p-value < 0.05 was considered statistically significant. Data analysis was performed using IBM SPSS statistical software (version 23).

Results

Prevalence of HPV

Of the 2610 specimens screened, 655 were found positive for HPV and included in the HPV molecular typing analysis. The overall prevalence of HPV in the study samples was 25.1%. Of the 655 positive samples, 645 (98.5%) were female, and 10 (1.5%) were male. The mean age of the positive cases was 35.95 years (women: 36.06 years and men: 28.81 years). Of the women, 587 (89.71%) were of reproductive age (⩽49 years).

HPV genotype distribution

A total of 41 HPV genotypes were detected in the 655 participants. The most commonly detected genotypes were types 6, 16, 54, 68, and 11, with frequencies of 197 (30.1%), 94 (14.4%), 59 (9%), 53 (8.1%), and 51 (7.8%) individuals, respectively (Table 1). Of the participants, 299 (45.6%) tested positive for high-risk HPV types, indicating a significant risk factor for developing cervical cancer and other related diseases. In addition, 159 (24.3%) participants showed the presence of probable risk types, indicating a moderate risk factor. On the other hand, 356 (54.4%) participants had low-risk HPV types, which are generally associated with a lower risk of serious health complications (Table 1). The highest number of participants (151 individuals, 23.05%) were in the 30–34-year-age group, while the lowest number was observed in the 65+ age group with only four individuals (Table 2).

Interestingly, the occurrence of superinfection, defined as the co-occurrence of multiple HPV types, was observed in 237 (36.2%) participants (Table 3). Furthermore, an analysis of HPV infections by age group revealed that the majority of HPV-positive cases were found in women of reproductive age (⩽49 years), accounting for 587 (89.61%) participants. Conversely, only 68 (10.38%) women aged over 49 years were identified as HPV positive.

Age-specific HPV prevalence

Among the five prevalent HPV genotypes in this study, genotypes 6, 16, and 54 had the highest age-specific prevalence rates. Specifically, in the age group 30–34 years, genotype six was prevalent in 50 individuals (25.4%), genotype 16 in 22 individuals (23.4%), and genotype 54 in 19 individuals (32.2%). For genotypes 11 and 18, the age-specific prevalence rates were highest in the age groups 25–29 years and 40–44 years, with 14 individuals (25.4%) and 11 individuals (21.2%), respectively.

Due to the smaller number of samples available for ages less than or equal to 19 years and greater than or

Table 2. Frequency of human papilloma virus genotypes across different age groups.

Age groups (*n*)	HPV-6 (%)	HPV-11 (%)	HPV-16 (%)	HPV-18 (%)	HPV-54 (%)
⩽19 (4)	3 (1.5)	0	0	0	1 (1.7)
20–24 (44)	24 (12.2)	9 (17.6)	5 (5.3)	3 (5.8)	5 (8.5)
25–29 (118)	39 (19.8)	14 (27.5)	18 (19.1)	6 (11.5)	8 (13.6)
30–34 (151)	50 (25.4)	6 (11.8)	22 (23.4)	9 (17.3)	19 (32.2)
35–39 (133)	37 (18.8)	9(17.6)	20 (21.3)	9 (17.3)	10 (16.9)
40–44 (98)	23 (11.7)	5 (9.8)	10 (10.6)	11 (21.2)	6 (10.2)
45–49 (39)	7 (3.6)	2 (3.9)	8 (8.5)	2 (3.8)	4(6.8)
50–54 (42)	5 (2.5)	1 (2)	6 (6.4)	8 (15.4)	4 (6.8)
55–59 (16)	5 (2.5)	0	1 (1.1)	2 (3.8)	2 (3.4)
60–64 (6)	2 (1)	4 (7.8)	4 (4.3)	2 (3.8)	0
65–69 (2)	0	0	0	0	0
⩾70 (2)	2 (1.0)	1 (2)	0	0	0

Table 3. Association between human papilloma virus infections and age, reproductive age and gender.

Variable	Super infection		*p*-Value	High risk types		*p*-Value	HPV-6		*p*-Value
	Yes, *n* (%)	No, *n* (%)		Yes, *n* (%)	No, *n* (%)		Yes, *n* (%)	No, *n* (%)	
Gender									
Male	4 (40.0)	6 (60.0)	0.75*	2 (20.0)	8 (80.0)	0.12*	7 (70.0)	3 (30.0)	0.01*
Female	233 (36.1)	412 (63.9)		297 (46.1)	348 (53.9)		190 (29.45)	455 (70.5)	
Reproductive age									
Yes	216 (43.1)	285 (56.9)	<0.001**	263 (52.5)	238 (47.5)	<0.001**	183 (36.5)	318 (63.5)	<0.001**
No	21 (13.6)	133 (86.4)		36 (23.4)	118 (76.6)		14 (9.1)	140 (90.9)	
Age, mean	35.51	36.37	0.26£	37.03	35.24	.014£	34.03	36.93	<0.001£

*Fisher's exact test.
**Chi-squared test.
£Mann–Whitney *U* test.

equal to 60 years, the frequency of different HPV genotypes was relatively lower. In fact, none of the common HPV genotypes were detected in the age group 65 to 69 years (Table 2).

HPV genotyping risk assessment

As shown in Table 3, 237 (36.2%) of the patients had superinfection. The percentage of cases with superinfection was significantly higher in people of reproductive age ($p < 0.001$). High-risk types were found in 299 (11.4%) patients. In other words, the prevalence of high-risk types among all positive HPV cases was 45.6%. The frequency of high-risk types was significantly higher in people of reproductive age ($p < 0.001$). One of the most important strains examined separately was HPV-6, which was observed in 197 (30.1%) of the positive cases. In addition to the significant relationship between the prevalence of this strain and reproductive age ($p < 0.001$), its frequency was significantly higher in women ($p = 0.01$). In addition, the mean age of patients with HPV-6 was significantly 3 years lower than that of other patients ($p < 0.001$).

Discussion

In the current study conducted in the city of Sanandaj, the average age of women infected with HPV was 36.06 years. The highest number of infected individuals was observed in the age group 30–34 years, with a total of 151 individuals (23.05%). A similar study by Gavrankapetanovic et al.[13] in Bosnia and Herzegovina reported a mean age of 33.38 years for women with HPV. In contrast, Prabhu et al.[14] conducted a study in the United States where the mean age of women with HPV was 23.9 years. This discrepancy may be due to different sexual behaviours observed in different parts of the world.[15,16]

The results showed that the prevalence of HPV in our data was 25.1%. Studies conducted around the world have provided different estimates of the prevalence of this virus. According to a study conducted by Vinodhini et al.[17] on 576,281 women from different regions of the world, the prevalence of infection was reported to be 32.1%. In a systematic review conducted by Colpani et al.[18] in Brazil, the prevalence of cervical HPV among 57,513 individuals was reported to be 25.4%.[18] The prevalence of this infection is

higher in high-risk groups such as female sex workers (FSWs), so much so that in a systematic review of 62 primary studies involving 21,402 FSWs from 33 countries, the pooled HPV prevalence was 42.6%.[19]

Based on our study, 299 (45.6%) of the total identified genotypes were classified as HRGs, while 356 (54.4%) were classified as LRGs. In a similar study conducted by Jamdar et al.[20] in Karaj in 2018, 10.3% of the identified genotypes were HRGs, while 89.7% were LRGs. However, in another study conducted by Thapa et al.[21] in 2018 in Nepal, approximately 57.3% of the identified genotypes were HRGs, while 42.7% were LRGs. It is worth noting that different reports on the prevalence of HRGs and LRGs have been observed in other studies, likely due to geographical differences in sample selection.[22–24]

Among the 41 genotypes investigated in our study, the most common genotypes were 6, 16, and 54, with frequencies of 197 (30.1%), 94 (14.4%), and 59 (9%), respectively. These genotypes have also been reported as the most common LRGs and HRGs in other studies, including HPV 6 and HPV 16.[25–27] In contrast to some studies in other regions of the world where HPV 18 was reported as the second most common HRG,[28,29] our study found HPV 59 to be the second most common HRG.

Individuals with HRGs had a significantly higher prevalence of multiple infections, while single infections were more common in individuals aged 45–54 years. In contrast, the study by Chen et al.[30] showed a higher rate of single infections in individuals aged 25–55 years. The highest age-specific HPV prevalence for the most common genotypes (6 and 16) was between 30 and 34 years of age. In the Iranian study by Kesheh et al.,[31] the 30–45 age group had one of the highest prevalence rates for HPV 6. However, the study by Dunne et al.[32] showed a different pattern, with the highest prevalence of types 6 and 16 in individuals aged 14–19 years and 20–24 years, respectively. In our study, a significant association was observed between HRGs and the age group 15–24 years. Similarly, the studies by Baussano et al.[33] and Trama et al.[34] showed that HRGs were significantly higher in individuals younger than 25 and 29 years, respectively.

It is clear that the high prevalence of HPV infection is one of the consequences of increasing risky sexual behaviors in society.[35] In addition, the high prevalence of this infection may be one of the major challenges for the health care system due to the increased risk of developing HPV-related cancers.[35,36] in such a situation, more attention needs to be paid to the issue of HPV vaccination as well as the early detection and timely treatment of HPV-related cancers. In such a situation, more attention needs to be paid to the issue of HPV vaccination, as well as to the early detection and timely treatment of HPV-related cancers.

This study has several limitations and some strengths that need to be noted. First limitation is that the results of the study could not be generalized to the total population of Kurdistan province because of the lack of sample size calculation and also, we did not select the samples randomly from the population and study patients were those who were referred by doctors to the laboratories. The second limitation is the lower samples from the male population, so we could not estimate the prevalence of HPV in even study population. Despite these limitations, this study is considered the first study in Kurdistan that was conducted to determine the genotypes of papillomavirus. The second strength of the study is that viral genotyping of all samples was performed using the same method and in the same laboratory. Further population-based studies with a larger sample size of both men and women in our regions are needed in order to generalize the results.

Conclusion

In conclusion, this study provides valuable insights into the prevalence and genotype distribution of HPV in the Kurdish population. The results indicate that HPV infection is prevalent in women of reproductive age, with the highest number of cases observed in the 30–34-year age group. Furthermore, high-risk HPV genotypes were found in a significant proportion of the infected population, suggesting a risk factor for the development of cervical cancer and related diseases. The most common genotypes identified were types 6, 16, and 54. Interestingly, the occurrence of superinfection, defined as the co-occurrence of multiple HPV types, was observed in more than a third of the participants. The study also showed a significant association between HPV types and the age group 15–24 years. These findings highlight the importance of targeted HPV vaccination programs and screening strategies, particularly in younger age groups, to reduce the burden of HPV-related disease in the Kurdish population. According to our data and high prevalence of HPV, we recommend interventional program specifically vaccination in national level.

Acknowledgements

All authors would like to thank the study patients and laboratories of Sanandaj for collaboration in this research.

Authors' contributions

The concept was designed by authors BN and KR; authors SN, FS, PB, AD, SM, and FZ were involved in data collection; SN, BN, and KR were involved in documentation for the study; the initial manuscript was drafted by KR and BN; KR and SN coordinated, supervised, and critically reviewed the manuscript for intellectual content. All authors approved the final manuscript and agree upon being accountable for all aspects of the work.

Availability of supporting data

Please contact the corresponding author for data requests.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

Ethical considerations

The proposal of this study was assessed and approved by the ethics committee of Kurdistan University of Medical Sciences (Ethic code: IR.MUK.REC.1399.247). In addition, written informed consent was obtained from each patient before the data gathering.

Informed consent

Written informed consent was obtained from all subjects before the study.

Consent for publication

Written informed consent was obtained from each patient for publication of this research.

Trial registration

Not applicable.

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References

1. Burd EM and Dean CL. Human papillomavirus. *Microbiol Spectr* 2016; 4(4): 177–195.
2. Dunne EF and Park IU. HPV and HPV-associated diseases. *Infect Dis Clin North Am* 2013; 27: 765–778.
3. Brianti P, De Flammineis E and Mercuri SR. Review of HPV-related diseases and cancers. *New Microbiol* 2017; 40: 80–85.
4. Shigeishi H and Sugiyama M. Risk factors for oral human papillomavirus infection in healthy individuals: a systematic review and meta-analysis. *J Clin Med Res* 2016; 8: 721.
5. De Sanjosé S, Diaz M, Castellsagué X, et al. Worldwide prevalence and genotype distribution of cervical human papillomavirus DNA in women with normal cytology: a meta-analysis. *Lancet Infect Dis* 2007; 7: 453–459.
6. Gebrie A. Disease progression role as well as the diagnostic and prognostic value of microRNA-21 in patients with cervical cancer: a systematic review and meta-analysis. *PLoS One* 2022; 17: e0268480.
7. Cheng L, Wang Y and Du J. Human papillomavirus vaccines: an updated review. *Vaccines* 2020; 8: 391.
8. Markowitz LE and Schiller JT. Human papillomavirus vaccines. *J Infect Dis* 2021; 224: S367–S378.
9. Bitarafan F, Hekmat MR, Khodaeian M, et al. Prevalence and genotype distribution of human papillomavirus infection among 12,076 Iranian women. *Int J Infect Dis* 2021; 111: 295–302.
10. Aghaeipour F, Salehiniya H and Abbaszadeh H. Prevalence of human papillomavirus (HPV) in oral mucosal lesions in Iran: a systematic review and meta-analysis. *J Med Virol* 2021; 93: 6089–6099.
11. Calagna G, Maranto M, Paola C, et al. "Secondary prevention" against female HPV infection: literature review of the role of carrageenan. *Expert Rev Anti Infect Ther* 2020; 18: 865–874.
12. Bosco L, Serra N, Fasciana T, et al. Potential impact of a nonvalent anti HPV vaccine in Italian men with and without clinical manifestations. *Sci Rep* 2021; 11: 4096.
13. Gavrankapetanovic F, Sljivo A, Dadic I, et al. Epidemiological aspects of age and genotypical occurrence of HPV infection among females of Canton Sarajevo over a 10-year period. *Mater Sociomed* 2022; 34: 260.
14. Prabhu VS, Bansal N, Liu Z, et al. HPV vaccination uptake and administration from 2006 to 2016 in a commercially insured population of the United States. *BMC Public Health* 2021; 21: 1–9.
15. Liu G, Hariri S, Bradley H, et al. Trends and patterns of sexual behaviors among adolescents and adults aged 14 to 59 years, United States. *Sex Transm Dis* 2015; 42: 20–26.
16. Moon JW. Why are world religions so concerned with sexual behavior? *Curr Opin Psychol* 2021; 40: 15–19.
17. Vinodhini K, Shanmughapriya S, Das BC, et al. Prevalence and risk factors of HPV infection among women from various provinces of the world. *Arch Gynecol Obstet* 2012; 285: 771–777.
18. Colpani V, Soares Falcetta F, Bacelo Bidinotto A, et al. Prevalence of human papillomavirus (HPV) in Brazil: a systematic review and meta-analysis. *PLoS One* 2020; 15: e0229154.
19. Farahmand M, Moghoofei M, Dorost A, et al. Prevalence and genotype distribution of genital human papillomavirus infection in female sex workers in the world: a systematic review and meta-analysis. *BMC Public Health* 2020; 20: 1–14.
20. Jamdar F, Farzaneh F, Navidpour F, et al. Prevalence of human papillomavirus infection among Iranian women using COBAS HPV DNA testing. *Infect Agent Cancer* 2018; 13: 1–5.
21. Thapa N, Maharjan M, Shrestha G, et al. Prevalence and type-specific distribution of human papillomavirus infection among women in mid-western rural, Nepal-A population-based study. *BMC Infect Dis* 2018; 18: 1–8.
22. Arbyn M, Xu L, Verdoodt F, et al. Genotyping for human papillomavirus types 16 and 18 in women with minor cervical lesions: a systematic review and meta-analysis. *Ann Intern Med* 2017; 166: 118–127.
23. Cox JT, Castle PE, Behrens CM, et al. Comparison of cervical cancer screening strategies incorporating different combinations of cytology, HPV testing, and genotyping for HPV 16/18: results from the ATHENA HPV study. *Am J Obstet Gynecol* 2013; 208: 184.e181–184.e111.
24. Tsakogiannis D, Gartzonika C, Levidiotou-Stefanou S, et al. Molecular approaches for HPV genotyping and HPV-DNA physical status. *Expert Rev Mol Med* 2017; 19: e1.
25. Jentschke M, Soergel P and Hillemanns P. Importance of HPV genotyping for the screening, therapy and management

of cervical neoplasias. *Geburtshilfe Frauenheilkd* 2012; 72: 507–512.
26. Tornesello M, Losito S, Benincasa G, et al. Human papillomavirus (HPV) genotypes and HPV16 variants and risk of adenocarcinoma and squamous cell carcinoma of the cervix. *Gynecol Oncol* 2011; 121: 32–42.
27. Zeng Z, Yang H, Li Z, et al. Prevalence and genotype distribution of HPV infection in China: analysis of 51,345 HPV genotyping results from China's largest CAP certified laboratory. *J Cancer* 2016; 7: 1037.
28. Fabiano G, Marcellusi A, Mennini F, et al. Hospital resource utilization from HPV-related diseases in England: a real-world cost analysis. *Eur J Health Econ* 2023; 24: 75–80.
29. Petousis-Harris H. Antigen literature review for the New Zealand national immunization schedule, 2016: human papillomavirus; 2016: 2–52.
30. Chen H-C, Schiffman M, Lin C-Y, et al. Persistence of type-specific human papillomavirus infection and increased long-term risk of cervical cancer. *J Natl Cancer Inst* 2011; 103: 1387–1396.
31. Kesheh MM and Keyvani H. The prevalence of HPV genotypes in Iranian population: an update. *Iran J Pathol* 2019; 14: 197.
32. Dunne EF, Unger ER, Sternberg M, et al. Prevalence of HPV infection among females in the United States. *JAMA* 2007; 297: 813–819.
33. Baussano I, Elfström KM, Lazzarato F, et al. Type-specific human papillomavirus biological features: validated model-based estimates. *PLoS One* 2013; 8: e81171.
34. Trama JP, Trikannad C, Yang JJ, et al. High-risk HPV genotype distribution according to cervical cytology and age. *Open Forum Infect Dis* 2022; 9(11): ofac595.
35. Cook RL, Thompson EL, Kelso NE, et al. Sexual behaviors and other risk factors for oral human papillomavirus infections in young women. *Sex Transm Dis* 2014; 41: 486–492.
36. Vaccarella S, Franceschi S, Herrero R, et al. Sexual behavior, condom use, and human papillomavirus: pooled analysis of the IARC human papillomavirus prevalence surveys. *Cancer Epidemiol Biomarkers Prev* 2006; 15: 326–333.