Medical Science

To Cite:
Bielawska K, Auguścik WJ, Tlak A. Human papillomavirus (HPV) infection – risk factors, mechanisms of carcinogenesis and modern prevention strategies for cervical cancer - a narrative review. *Medical Science* 2025; 29: e223ms3736
doi: https://doi.org/10.54905/disssi.v29i166.e223ms3736

Authors' Affiliation:
[1]Medical University of Warsaw, 61 Żwirki i Wigury Street, 02-091 Warsaw, Poland
[2]Medical University of Łódź, 4 Tadeusza Kościuszki Street, 90-419 Łódź, Poland

***Corresponding Author:**
Katarzyna Bielawska,
Medical University of Warsaw, 61 Żwirki i Wigury Street, 02-091 Warsaw, Poland,
E-mail: bielawskka@wp.pl

Orcid:
Katarzyna Bielawska 0009-0001-7381-8415
Wiktoria Julia Auguścik 0009-0009-6670-1604
Aleksandra Tlak 0009-0008-2506-5309

Peer-Review History
Received: 12 July 2025
Reviewed & Revised: 25/July/2025 to 21/November/2025
Accepted: 30 November 2025
Published: 9 December 2025

Peer-review Method
External peer-review was done through double-blind method.

Medical Science
pISSN 2321–7359; eISSN 2321–7367

DISCOVERY SCIENTIFIC SOCIETY

Human papillomavirus (HPV) infection – risk factors, mechanisms of carcinogenesis and modern prevention strategies for cervical cancer - a narrative review

Katarzyna Bielawska[1]*, Wiktoria Julia Auguścik[1], Aleksandra Tlak[2]

ABSTRACT

Cervical cancer is one of the most common cancers worldwide, despite the availability of preventive and diagnostic methods. Progression to cancerous changes takes a long time. Infection with human papillomavirus (HPV) plays a crucial role, but not the only one. Recent studies increasingly show that multiple viral, immunological, hormonal, and microbiological factors influence the course and progression of the infection. This review aims to summarize current knowledge on the mechanisms of HPV-dependent carcinogenesis, with particular emphasis on the role of vaginal microflora, environmental factors, and modern methods of prevention and diagnosis. This review draws on 17 scientific papers published between 2011 and 2025, found through searches in PubMed, Scopus, and Google Scholar. Disruption of the vaginal microbiome may contribute to the persistence of HPV infection. A decrease in Lactobacillus bacteria and an increase in anaerobic bacteria lead to inflammation and oxidative stress, which may facilitate the integration of viral DNA into the host genome. There is increasing emphasis on new diagnostic methods. Testing for epigenetic methylation markers (e.g., CADM1, MAL) may improve the specificity of molecular tests. HPV vaccination is known to be a preventive measure against CIN2+ lesions and cervical cancer. Restoring normal vaginal microflora through the use of probiotics may be an important element in the prevention and treatment of HPV infections in the future. The most effective way to decrease incidence may be to combine comprehensive vaccination programs with modern, integrated screening strategies.

Keywords: cervical cancer, epigenetic biomarkers, human papillomavirus (HPV), microbiota, prevention

1. INTRODUCTION

The most common sexually transmitted disease is HPV infection (Schiffman et al., 2011). Over 80% of sexually active women and men come into contact with the virus during their lifetime. In most cases, HPV infection regresses spontaneously within 12-24 month. Only a small percentage of infections persist, leading to precancerous changes (Vink et al., 2013). The main factor in cervical intraepithelial neoplasia

(CIN) and invasive cancer is persistent infection with HPV-16 and HPV-18 (Schiffman et al., 2011).

The development of cervical cancer follows several stages. According to research (Vink et al., 2013), the average time for CIN2/3 lesions to progress to cervical cancer is approximately 23 years, but HPV-16 infection shows a tendency toward faster progression. The development of cervical cancer is a complex process involving many factors. These include environmental, immunological, and viral factors. The type of HPV virus and the integration of its genetic material into the host genome are of significant importance in the development of carcinogenesis (Schiffman et al., 2011; Luhn et al., 2013; Bowden et al., 2023).

Persistent HPV infection is likely promoting vaginal and cervical dysbiosis. The predominance of anaerobic bacteria over Lactobacillus species hinders virus clearance, encouraging chronic inflammation (Alimena et al., 2022; Lebeau et al., 2022; Mancilla et al., 2024). The literature emphasizes the role of coexisting factors such as smoking, multiple births, and long-term use of hormonal contraceptives, which may exacerbate the process of cellular transformation during HPV infection (Luhn et al., 2013).

Introduction of HPV vaccination programs and development of modern diagnostic methods have had a significant impact on cervical cancer prevention. Molecular HPV DNA testing is highly sensitive but lacks specificity, which may lead to overdiagnosis of transient or clinically insignificant lesions (Swid and Monaco, 2022).

The purpose of this review is to summarize current evidence on the natural course of HPV infection, with particular emphasis on viral, environmental, and host factors that influence the progression of intraepithelial lesions to cervical cancer.

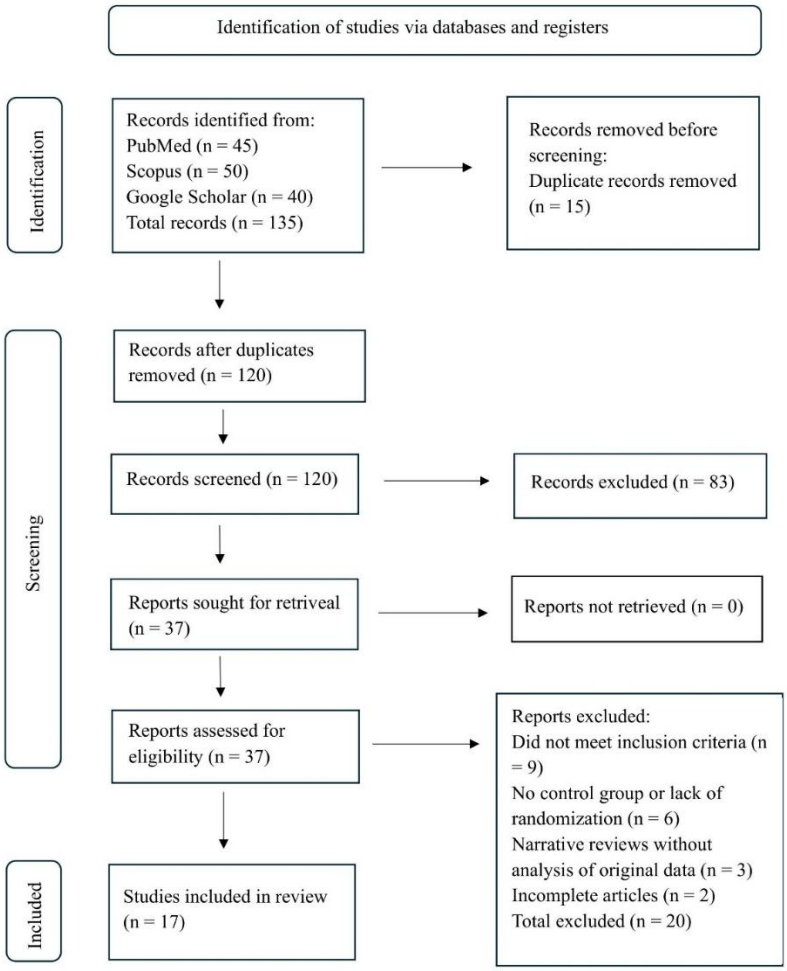

Figure 1. PRISMA flow diagram showing the selection process of studies included in the review.

2. REVIEW METHODS

In this review, we analyzed studies found in PubMed, Scopus, and Google Scholar. This review included an analysis of studies on human papillomavirus (HPV) infections and their association with cervical intraepithelial neoplasia (CIN). Particular attention focused on factors influencing the persistence of infection, such as the vaginal microbiome and the host immune response. We applied

keywords including HPV, human papillomavirus, cervical cancer, vaginal microbiome, epigenetic biomarkers, methylation, screening, and vaccination to locate relevant articles. We mainly analyzed publications from 2011–2025. We only added older works when they were needed to supplement the context. We included only English-language studies that underwent peer review. Altogether, we identified 135 papers: 45 in PubMed, 50 in Scopus, and 40 in Google Scholar. After removing 15 duplicates, we screened 120 records and assessed 37 full-text articles for eligibility - the final analysis comprised 17 studies. Figure 1 presents the study selection process. The primary sources of bias in this review may include the limited number of randomized controlled trials, heterogeneous study designs, and small sample sizes, which could reduce the representativeness of the findings.

3. RESULTS

HPV infection and mechanisms of cervical carcinogenesis

The development of cervical cancer is a multifaceted process. In most cases, HPV infection is resolved spontaneously within 12-24 months. However, persistent infection with HPV16 and HPV18 can lead to CIN3 lesions and cervical cancer (Schiffman et al., 2011; Vink et al., 2013). Neoplastic changes develop because of the deactivation of the p53 and pRb tumor suppressors by the viral oncoproteins E6 and E7. This phenomenon results in cell cycle dysregulation, genomic instability, and the survival of cells with damaged DNA. At a later stage, gene expression deregulation and permanent activation of oncoproteins occur because of viral DNA integration into the host genome (Nedjai et al., 2018).

The integration of viral DNA into the host genome leads to epigenetic changes, such as abnormal methylation of the CADM1, MAL, DAPK1, and RARB suppressor genes. Recent studies show that increased methylation of viral genes (L1, L2) promotes persistent infection and facilitates the integration of HPV DNA into the host genome (Bowden et al., 2023).

Immunological and hormonal factors influence the persistence and progression of HPV infection. HPV can effectively modulate the host's immune response. The E6 and E7 oncoproteins inhibit the expression of type I and II interferons, weakening the activation of cytotoxic T lymphocytes (CD8+) and NK cells. As a result, infected cells evade immune elimination, and chronic infection triggers persistent inflammation (Luhn et al., 2013). Chronic HPV infection leads to inflammation of the cervical epithelium and increased concentrations of proinflammatory cytokines (IL-6, IL-8, and TNF-α).

Smoking, long-term use of hormonal contraceptives, and multiple births are further factors that increase the risk of persistent HPV infection and, consequently, neoplastic changes (Table 1) (Bowden et al., 2023).

Table 1. Primary risk factors of persistent HPV infection and CIN progression

Category	Risk factor	Proposed mechanism	References
Viral	HPV type (16, 18)	High oncogenic potential, E6/E7 oncoproteins inactivate p53 and pRb,	Schiffman et al., 2015; Vink et al., 2021.
Hormonal	Long-term hormonal contraception	Altered cervical microenvironment, enhanced proliferation,	Macios and Nowakowski, 2022;
Environmental	Smoking, multiparity,	Oxidative stress, epithelial microtrauma,	Bowden et al., 2023.

The mechanisms described facilitate the progression of CIN lesions by stimulating proliferation signaling pathways, altering DNA methylation, and inhibiting local immune responses. The cervical microenvironment, particularly the vaginal microbiota, further influences infection outcomes. Its role in HPV persistence and the progression of precancerous lesions is discussed in detail later in this article (Alimena et al., 2022; Lebeau et al., 2022).

Vaginal microbiota in HPV persistence and cervical carcinogenesis

Recent studies confirm the relationship between vaginal and cervical dysbiosis and an increased risk of HPV infection persistence and the development of cancerous lesions (Alimena et al., 2022; Lebeau et al., 2022). Normal microflora is predominantly dependent on Lactobacillus bacteria, which maintain an acidic pH (3.5–4.5) through the synthesis of lactic acid, hydrogen peroxide (H_2O_2), and

bacteriocins, which prevent the occurrence of pathogenic microorganisms and increase the resistance of mucous membranes. It is a natural immune barrier that protects against HPV infections and other sexually transmitted pathogens (Alimena et al., 2022).

Dysbiosis leads to a decrease in Lactobacillus bacteria and an increase in the number of anaerobic bacteria such as Gardnerella vaginalis, Atopobium vaginae, Prevotella spp., Sneathia spp., Megasphaera spp., and Mobiluncus spp. (Lebeau et al., 2022). Reduced lactic acid production leads to a higher vaginal pH and increased activity of proteolytic enzymes and cytotoxic metabolites, which damage the epithelium and compromise its barrier function.

Abnormal vaginal microflora promotes HPV infection by inducing a local inflammatory response. There is an increase in proinflammatory cytokines (IL-1β, IL-6, IL-8, TNF-α), activation of the NF-κB pathway, and recruitment of neutrophils and macrophages to the site of infection. Chronic inflammation leads to the formation of reactive oxygen species (ROS), oxidative stress, and DNA abnormalities in host cells. These factors facilitate the integration of viral DNA into the host genome, promoting the development of neoplastic changes. (Lebeau et al., 2022).

HPV infection can also affect the composition of the vaginal microflora. Studies have shown that infection inhibits the expression of host antimicrobial peptides, including SLPI and β-defensins, which are a source of amino acids for Lactobacillus species. This bidirectional relationship forms a self-perpetuating cycle—HPV infection induces dysbiosis, and dysbiosis, in turn, sustains the infection (Lebeau et al., 2022).

Studies involving premenopausal women have shown that CIN3 progression is associated with microbiota characterized by increased diversity (community type IV). Female patients with spontaneous regression of HPV infection were found to have vaginal microflora rich in Lactobacillus bacteria (community type I) (Banila et al., 2025). The results suggest that microflora testing may in future serve as a marker for assessing the risk of persistent HPV infection and the progression of precancerous lesions. Studies conducted among Latin women reported an association between the presence of bacterial vaginosis and an increased risk of HPV infection, regardless of the patients' age and immune status (Mancilla et al., 2024). Moreover, particular bacterial species can secrete enzymes that break down the extracellular matrix and modulate the expression of cell receptors used by HPV to penetrate the epithelium.

Modulation of the vaginal microflora through the use of probiotics rich in Lactobacillus crispatus or L. rhamnosus bacteria seems to be another method that can support the treatment of HPV infection. Proper biocenosis can reduce inflammation and improve CIN treatment outcomes (Alimena et al., 2022; Mancilla et al., 2024). Preliminary clinical studies suggest that an intervention like this may increase the likelihood of eliminating the virus and preventing its recurrence.

Screening strategies for cervical cancer

The basis for secondary prevention of cervical cancer is still screening, which enables early detection and effective treatment of precancerous lesions. Conventional cytology (the Papanicolaou test), developed in the mid-20th century, remains the foundation of cervical cancer screening. Since its introduction, this method has played a crucial role in lowering both the incidence and mortality of cervical cancer worldwide (Schiffman et al., 2011). The later development of liquid-based cytology (LBC) improved the quality of the material collected. It enabled the use of the same sample for molecular testing, including HPV detection (Table 2) (Swid and Monaco, 2022).

Table 2. Summary of various screening tests for cervical cancer

Method	Principle	Advantages	Limitations	Recommended interval
Liquid-based cytology (LBC)	Automated preparation, and HPV testing on the same sample.	Better sample quality, fewer artifacts.	Higher cost, specialized equipment.	Every 3 years, Nedjai et al., 2018.
HPV DNA test	Detection of high-risk viral genetic material.	High sensitivity, early detection.	Low specificity (transient infections).	Every 5 years. Schiffman et al., 2011.
Epigenetic assays	DNA methylation of host/viral genes.	Potential high specificity for relevant infections.	Still in research phase,	In validation, Nedjai et al., 2018.

The discovery of the link between HPV infection and cervical cancer was a breakthrough in prevention, leading to the development of molecular tests that detect the genetic material of high-risk HPV. These tests are more sensitive and have a higher negative predictive value in detecting CIN2+ lesions than cytology. Thanks to the introduction of these tests, it is possible to extend the time between screening tests to 5 years (Rebolj et al., 2024). It noted that the specificity of the tests described is lower, which can lead to the detection of clinically insignificant lesions and transient infections (Macios and Nowakowski, 2022).

Current recommendations suggest that tests detecting viral genetic material should form the basis of screening. According to the 2020 guidelines of the American Cancer Society (ACS), HPV screening should be performed every 5 years starting at age 25. An alternative may be cytology every 3 years or a combination of both methods (cytology plus HPV) every 5 years (Swid and Monaco, 2022).

Pap smears remain an important diagnostic method in screening. They enable morphological assessment of cervical epithelial cells and allow for the identification of abnormalities unrelated to HPV, including infections, metaplastic changes, and rare glandular changes. (Swid and Monaco, 2022). In areas where molecular testing remains inaccessible, cytology continues to play a key role because of its low cost and widespread availability (Schiffman et al., 2011)

In order for tests detecting viral genetic material to be reliable in diagnosis, it is necessary to control the quality of the tests performed, validate analytical platforms, and provide appropriate training for physicians. Studies show that the most effective screening method is a combined approach—initial HPV testing followed by cytological verification of positive results. Using both methods reduces the number of unnecessary colposcopies (Rebolj et al., 2024;).

HPV Vaccination in Cervical Cancer Prevention

The most important achievement in primary prevention of cervical cancer was the introduction of HPV vaccination. At the moment, bivalent, quadrivalent, and nonavalent vaccines are available on the commercial market. All vaccines target high-risk HPV types—mainly 16 and 18—which are responsible for approximately 70% of cervical cancer cases. (Schiffman et al., 2011). HPV vaccination substantially lowers the risk of developing high-grade cervical lesions (CIN2+ and CIN3+) and thereby prevents progression to invasive cancer. (Table 3) (Ikeda et al., 2021).

Table 3. Current strategies for cervical cancer prevention

Prevention type	Example actions	Mechanism	Effectiveness
Primary	HPV vaccination (bivalent, quadrivalent, nonavalent)	Prevention of infection with high-risk HPV types (16, 18).	Up to 90–95% CIN2+ reduction Ikeda et al., 2021
Secondary	Screening: cytology, HPV tests, co-testing.	Early detection and treatment of precancerous lesions.	Reduced mortality. Schiffman et al., 2015
Tertiary	Treatment of CIN, microbiota modulation (L. crispatus probiotics),	reduced recurrence and improved immune response.	Under clinical evaluation, Rebolj et al., 2024

A nationwide case–control study from Japan by Ikeda et al., (2021), which included women aged 20–29 years, reported that HPV vaccination prevented more than 90% of CIN2+ lesions. This finding supports the strong association between high vaccination coverage and reduced incidence of cervical precancerous lesions in screening programs.

Schiffman et al., (2011) highlighted that widespread vaccination calls for an update of current cervical screening strategies, since declining rates of high-risk HPV infection among vaccinated women alter both predictive values and optimal screening intervals. Population studies show that HPV infection rates are significantly lower in countries with high vaccination coverage. This also applies to unvaccinated individuals, which is a result of herd immunity (Rebolj et al., 2024). The high efficacy of HPV vaccines has long been proven. However, vaccination rates remain low in many countries due to social, financial, and organizational factors. Achieving high vaccination rates—especially before the onset of sexual activity—is crucial to ensuring maximum protection. However, in several countries, including Poland, vaccination rates remain below the levels recommended by international health authorities (ECDC, 2021).

Nevertheless, HPV vaccination does not replace the need for continued cervical cancer screening. Current guidelines consistently recommend continued participation in screening programs for both vaccinated and unvaccinated women, because the available vaccines do not protect against all oncogenic HPV types (Schiffman et al., 2011).

4. DISCUSSION

The literature clearly confirms the key role of HPV in the pathogenesis of cervical cancer. Increasing evidence suggests that this process is much more complex than previously assumed (Schiffman et al., 2011; Luhn et al., 2013; Vink et al., 2013; Bowden et al., 2023). HPV infection is the leading risk factor for cervical cancer. Still, many interacting factors influence the further development of the disease — both viral and involving the host organism, microbiota, and hormonal balance (Alimena et al., 2022; Mancilla et al., 2024; Lebeau et al., 2022; Banila et al., 2025).

Differences in the rate of progression from HPV infection to invasive cancer are an essential topic for researchers. Median time to cervical cancer development from high-grade intraepithelial neoplasia (CIN2/3) is approximately 23 years, confirming the long-term and multi-stage nature of carcinogenesis (Vink et al., 2013). At the same time, population data suggest that HPV-16 infections progress more rapidly, emphasizing the importance of the virus genotype as a factor determining disease dynamics (Schiffman et al., 2011; Vink et al., 2013). However, many retrospective studies have reported wide variation in the timing of lesion progression and regression, likely reflecting differences in study populations and diagnostic approaches. There is a lack of prospective studies with long follow-up periods that would allow the natural course of infection to be precisely determined, depending on the type of virus and the patient's immune status.

Much of the current research focuses on the epigenetic and immunological mechanisms of cancer transformation. The introduction of conventional cytology into screening has significantly reduced mortality from cervical cancer, but it is characterized by low sensitivity in detecting early lesions. This fact highlights the need to introduce additional methods into screening strategies, such as molecular tests or epigenetic marker analysis (Nedjai et al., 2018; Bowden et al., 2023). Studies show that HPV genetic material detection also has limited application because it has low specificity, which can result in overdiagnosis of transient infections (Rebolj et al., 2024).

The dominance of anaerobic bacteria over Lactobacillus bacteria leads to chronic inflammation and an increased frequency of viral DNA integration into the host genome. It remains unproven whether dysbiosis is the cause or effect of HPV infection. Studies indicate a bidirectional relationship between abnormal vaginal microflora and HPV infection (Alimena et al., 2022; Lebeau et al., 2022; Rebolj et al., 2024). A more detailed analysis of this relationship could complement the treatment strategy for HPV infection through the use of probiotics that would support the elimination of the virus. This area requires randomized trials to confirm the effectiveness of the described therapeutic intervention..

Primary prevention through HPV vaccination is another area of dynamic development. Studies conducted in Japan confirm that vaccination is more than 90% effective in preventing CIN2+ lesions (Ikeda et al., 2021). A similar decline in cervical cancer incidence is being seen worldwide in countries with high vaccination coverage. The introduction of universal HPV vaccination programs have led to a change in the epidemiological picture of infections. There is a reduction in the incidence of HPV 16 and 18 infections among vaccinated individuals, while other types of the virus not covered by vaccination are becoming more common. The lower prevalence of HPV-16 and HPV-18 infections may reduce the predictive value of molecular tests for cervical cancer. This aspect should inform the planning of future screening programs.

The main barriers include a lack of adequate health education and low public awareness. In light of research findings from other countries (Schiffman et al., 2011; Ikeda et al., 2021; Rebolj et al., 2024), increasing vaccination coverage should become one of the key objectives of public health policy.

5. CONCLUSION

HPV infection is the primary factor in the development of cervical cancer, and its course depends on the interaction of viral, immunological, and microbiological aspects. Effective prevention should combine HPV vaccination with modern screening strategies, which will reduce morbidity in the coming years.

Acknowledgments

The authors have no acknowledgments to disclose.

Author Contribution

K.B. – conceptualization, literature review, manuscript drafting, and preparation of tables and figures.
W.J.A. – data analysis, verification of scientific content, and critical revision of the manuscript for important intellectual content.
A.T. – supervision, methodological guidance, and final approval of the version to be published.
All authors read and approved the final version of the manuscript.

Informed consent

Not applicable.

Ethical approval

Not applicable. This article does not contain any studies with human participants or animals performed by any of the authors.

Funding

This research did not receive any external funding like specific grant from funding agencies in the public, commercial, or nonprofit sectors.

Conflict of interest

The authors declare that they have no conflicts of interests, competing financial interests or personal relationships that could have influenced the work reported in this paper.

Data and materials availability

All data associated with this study will be available based on reasonable request to the Corresponding Author.

REFERENCES

1. Alimena S, Davis J, Fichorova RN, Feldman S. The vaginal microbiome: a complex milieu affecting risk of human papillomavirus persistence and cervical cancer. Curr Probl Cancer 2022;46(4):100877. doi:10.1016/j.currproblcancer.2022.100877.
2. Banila C, Ladoukakis E, Scibior-Bentkowska D, Rodrigues Santiago L, Reuter C, Kleeman M, Nedjai B. A longitudinal pilot study in pre-menopausal women links cervicovaginal microbiome to CIN3 progression and recovery. Commun Biol 2025;8(1):883. doi:10.1038/s42003-025-08328-w.
3. Bowden SJ, Doulgeraki T, Bouras E, Markozannes G, Athanasiou A, Grout-Smith H, Kechagias KS, Burney Ellis L, Zuber V, Chadeau-Hyam M, Flanagan JM, Tsilidis KK, Kalliala I, Kyrgiou M. Risk factors for human papillomavirus infection, cervical intraepithelial neoplasia and cervical cancer: an umbrella review and follow-up Mendelian randomisation studies. BMC Med 2023;21(1):274. doi:10.1186/s12916-023-02965-w.
4. European Centre for Disease Prevention and Control (ECDC). Efficacy, effectiveness and safety of HPV vaccination in women with conisation. Stockholm: ECDC; 2021.
5. Ikeda S, Ueda Y, Hara M, Yagi A, Kitamura T, Kitamura Y, Konishi H, Kakizoe T, Sekine M, Enomoto T, Sobue T. Human papillomavirus vaccine to prevent cervical intraepithelial neoplasia in Japan: a nationwide case-control study. Cancer Sci 2021;112(2):839–846. doi:10.1111/cas.14682.
6. Lebeau A, Bruyere D, Roncarati P, Peixoto P, Hervouet E, Cobraiville G, Taminiau B, Masson M, Gallego C, Mazzucchelli G, Smargiasso N, Fleron M, Baiwir D, Hendrick E, Pilard C, Lerho T, Reynders C, Ancion M, Greimers R, Twizere JC, Daube G, Schlecht-Louf G, Bachelerie F, Combes JD, Melin P, Fillet M, Delvenne P, Hubert P, Herfs M. HPV infection alters vaginal microbiome through down-regulating host mucosal innate peptides used by Lactobacilli as amino acid sources. Nat Commun 2022;13(1):1076. doi:10.1038/s41467-022-28724-8.
7. Luhn P, Walker J, Schiffman M, Zuna RE, Dunn ST, Gold MA, Smith K, Mathews C, Allen RA, Zhang R, Wang S, Wentzensen N. The role of co-factors in the progression from human papillomavirus infection to cervical cancer. Gynecol Oncol 2013;128(2):265–270. doi:10.1016/j.ygyno.2012.11.003.
8. Macios A, Nowakowski A. False negative results in cervical cancer screening—risks, reasons and implications for clinical practice and public health. Diagnostics (Basel) 2022;12(6):1508. doi:10.3390/diagnostics12061508.
9. Mancilla V, Jimenez NR, Bishop NS, Flores M, Herbst-Kralovetz MM. The vaginal microbiota, human papillomavirus infection, and cervical carcinogenesis: a

systematic review in the Latina population. J Epidemiol Glob Health 2024;14(2):480–497. doi:10.1007/s44197-024-00201-z.

10. Nedjai B, Reuter C, Ahmad A, Banwait R, Warman R, Carton J, Boer S, Cuzick J, Lorincz AT. Molecular progression to cervical precancer, epigenetic switch or sequential model? Int J Cancer 2018;143(7):1720–1730. doi:10.1002/ijc.31549.
11. Rebolj M, Brentnall AR, Cuschieri K. Predictable changes in the accuracy of human papillomavirus tests after vaccination: review with implications for performance monitoring in cervical screening. Br J Cancer 2024;130:1733–1743. doi:10.1038/s41416-024-02681-z.
12. Schiffman M, Wentzensen N, Wacholder S, Kinney W, Gage JC, Castle PE. Human papillomavirus testing in the prevention of cervical cancer. J Natl Cancer Inst 2011;103(5):368–383. doi:10.1093/jnci/djq562.
13. Swid MA, Monaco SE. Should screening for cervical cancer go to primary human papillomavirus testing and eliminate cytology? Mod Pathol 2022;35(7):858–864. doi:10.1038/s41379-022-01052-4.
14. Vink MA, Bogaards JA, van Kemenade FJ, de Melker HE, Meijer CJLM, Berkhof J. Clinical progression of high-grade cervical intraepithelial neoplasia: estimating the time to preclinical cervical cancer from doubly censored national registry data. Am J Epidemiol 2013;178(7):1161–1169. doi:10.1093/aje/kwt077.