

Praca poglądowa/Review paper

Cervical cancer in the Polish population- preventive actions review

Sebastian Łażniak¹, Michalin Kliber¹, Sylwester Król¹, Paweł Jagodziński¹¹ Zakład Fizyki Medycznej, Wielkopolskie Centrum Onkologii, Poznań, Polska

Abstract

Cervical cancer (CC) remains one of the primary health concerns for women worldwide, with its prevalence in Poland posing a significant health challenge. Despite advancements in preventive treatments, this disease persists as one of the leading causes of morbidity and mortality among women. In Poland, its impact extends beyond individual health and patient quality of life, also affecting the healthcare system and the nation's economy.

Keywords: Cervical Cancer; Cervical Dysplasia; Human Papillomavirus (HPV); Thermocoagulation; Cervical Intraepithelial Neoplasia

Adres do korespondencji

Sebastian Łażniak

Wielkopolskie Centrum Onkologii, ul. Garbary 15, 61-866 Poznań, Polska

Telefon. +48 61 885 0700

e-mail: sebastian_lazniak@interia.pl

Introduction

Cervical cancer (CC) is a malignant tumor that develops in the cells lining the cervix- the lower part of the uterus that connects to the vagina. It is the sixth most common malignancy in women under the age of 45, with chronic infection with the human papillomavirus (HPV) being the primary cause. Poland has one of the highest incidence and mortality rates for cervical cancer in Europe (Bray in., 2024). Table 1 presents the number of cases from 2015 to 2021 (*Raporty | Krajowy Rejestr Nowotworów*, b.d.). From 2015 to 2018, the incidence rates showed a proportional decline. However, there was an increase in cases from 2018 to 2019, followed by a significant decrease from 2019 to 2020. Since 2020, an upward trend in incidence has been observed again. By 2017, thanks to increased awareness of prevention and a higher number of screening tests performed in Poland, the incidence of cervical cancer had decreased. In 2019, during the COVID-19 pandemic, it is estimated that not all cases of cervical cancer were captured Table 2 outlines the mortality rates from 2015 to 2021. Since 2017, there has been a noticeable downward trend in cervical cancer deaths, stabilizing at around 1,400 deaths annually. In the case of cervical cancer mortality, we can also observe a trend between the years 2019 and 2021, where the number of deaths decreased. This may suggest that not all cases were recorded due to the COVID-19 pandemic (Didkowska i in., 2022).

The pandemic imposed temporary restrictions on scheduled therapies, delayed diagnoses, and routine cancer screening tests. As shown in several previous international studies, lower wealth in cancer screening programs has increased the incidence of advanced-stage cancers, mortality, and years of life lost (Didkowska i in., 2022). Despite the availability of widespread preventive measures and screening tests in the Polish healthcare market, these statistics remain alarming (*Raporty | Krajowy Rejestr Nowotworów*, b.d.).

Cervical cancer can be classified into several types, depending on the kind of cells from which it originates. The

most common type is squamous cell carcinoma, accounting for about 70-90% of all cases. It develops from the flat cells lining the cervix. Adenocarcinoma constitutes approximately 10-25% of cases and originates from glandular cells in the cervical canal. Mixed carcinoma (adenosquamous carcinoma) exhibits features of both aforementioned types. Rare types include small cell carcinoma, sarcoma, and clear cell carcinoma. Cervical cancer can be cured if diagnosed and treated in its early stages. Recognizing symptoms and seeking medical advice to address any issues is a critical step. Women should consult a doctor if they notice: Unusual bleeding between periods, after menopause, or after sexual intercourse, increased or foul-smelling vaginal discharge, symptoms such as the persistent back, leg, or pelvic pain, weight loss, fatigue, and loss of appetite, vaginal discomfort, swelling of the legs (Nowicki I in., 2017).

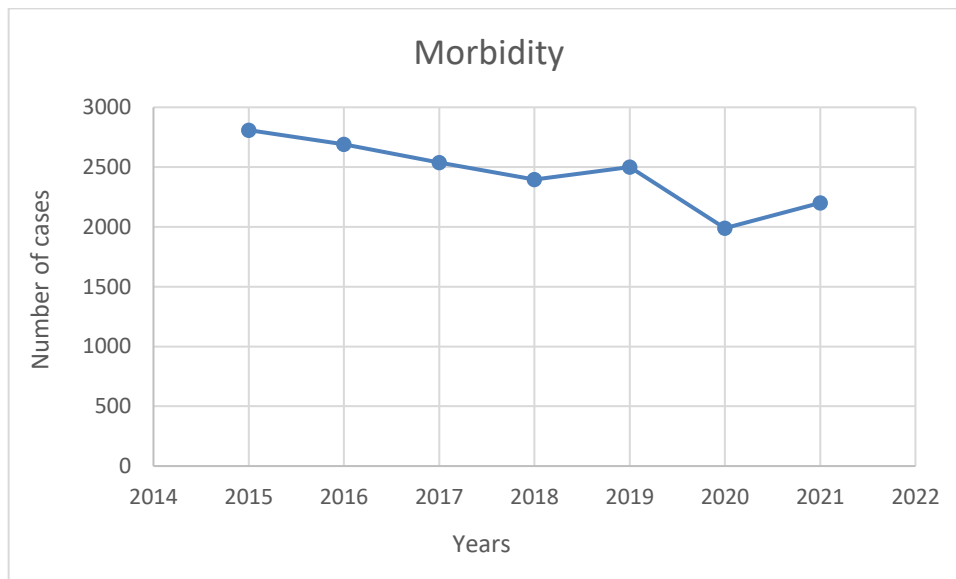


Figure 1. The table presents cervical cancer incidence data in women from 2015 to 2021. The results are illustrated in a chart based on the National Cancer Registry.

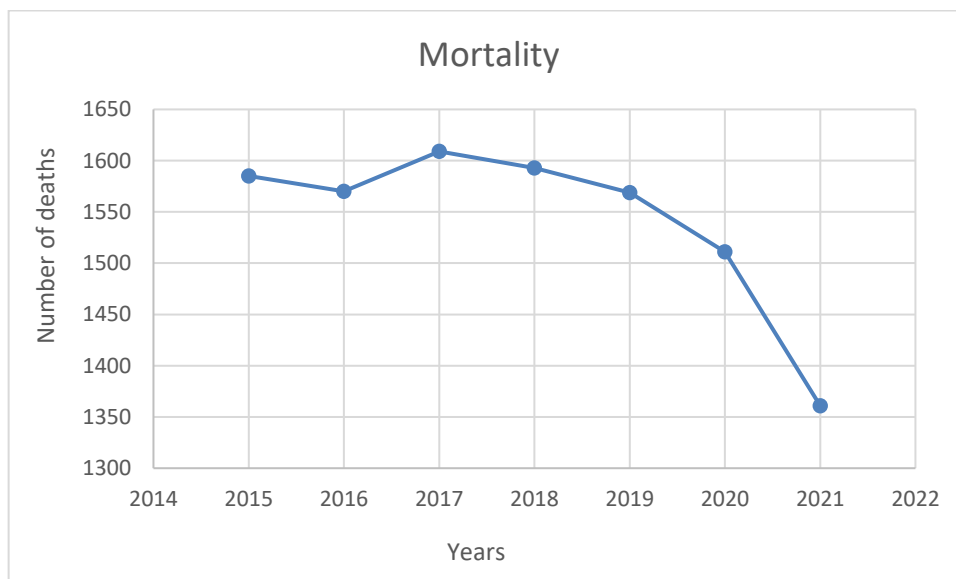


Figure 2. The graph includes data on the number of deaths of women in RSM in 2015-2021. The results were presented based on data from the National Cancer Registry.

Pathogenesis

Cervical cancer develops as a result of a prolonged process involving multistep cellular changes in the cervix, primarily triggered by infection with human papillomavirus (HPV). HPV is a small DNA virus consisting of a spherical genome, which is made of about 8000 base pairs surrounded by an icosahedral viral capsid made of two proteins L1 and L2 (Robert Jach, Magda Dumin, i in., b.d.). HPV infection is the leading risk factor for cervical cancer, and approximately 99% of cases are linked to chronic infection with high-risk HPV strains, particularly types 16 and 18 (Burd, 2003).

HPV is mainly transmitted through sexual contact, and infections can be asymptomatic, often resolving spontaneously due to immune system responses. In cases where the infection is not cleared, chronic infection can occur, leading to cellular changes in the epithelial cells of the cervix. HPV targets the epithelial cells of the cervical transformation zone, where squamous and glandular cells meet. This zone is especially susceptible to HPV infection. High-risk HPV strains produce E6 and E7 oncoproteins, which disrupt normal cellular regulation by inactivating tumor suppressors proteins like p53 and pRb (Revathidevi i in., 2021). This leads to disturbances in apoptosis (programmed cell death) and uncontrolled cell proliferation. Chronic HPV infection can cause cellular changes leading to cervical intraepithelial neoplasia (CIN). CIN is a continuous process of carcinogenesis that begins with low-grade dysplasia and ends with invasive cancer. There are three grades:

CIN 1 (low-grade): mild dysplastic changes, often resolving on their own.

CIN 2 (moderate-grade): more significant changes that may progress to advanced dysplasia.

CIN 3 (high-grade): severe changes with a high risk of progressing to invasive cervical cancer if untreated.

A critical step in the development of cervical cancer is the integration of HPV DNA into the host cell genome. High-risk HPV types, such as 16 and 18, can incorporate their genetic material into cervical cells. This leads to the persistent expression of oncoproteins E6 and E7, promoting genomic instability and increasing susceptibility to mutations (Burd, 2003)

If untreated, high-grade neoplasia can progress to invasive cancer, a process that may take several years. Squamous cell carcinoma is the most common form of cervical cancer (70–90% of cases), originating from the squamous epithelial cells of the cervix. Adenocarcinoma, arising from glandular cells, accounts for 10–25% of cases.

HPV is adept at evading the immune system, allowing persistent infection. Altered cervical cells infected with HPV may go undetected by immune surveillance, enabling continued growth and progression toward cancer.

Invasive cervical cancer occurs when cancer cells break through the basement membrane of the epithelium and begin to invade surrounding tissues, such as the uterus, vagina, bladder, and regional lymph nodes. Symptoms of invasive cancer include abnormal bleeding, pelvic pain, and urinary problems.

Stages of clinical advancement

Cervical cancer typically progresses through several stages, which can be described as follows. Precursors and Precancerous Changes:

Dysplasia: Early changes in the cervical cells that can be detected through a Pap smear or HPV testing. Dysplasia is classified into different grades (low-grade, moderate-grade, and high-grade)

based on the depth and extent of the changes (Piątek Szymon i in., b.d.).

Lichen Planus: Changes in the mucous membrane of the cervix that may result from viral infections such as HPV.

Early Cancer:

Carcinoma in Situ: A stage where cancerous cells are confined to the upper layer of the cervical mucosa and have not penetrated deeper tissues.

Invasive Cervical Cancer: Cancerous cells begin to invade beyond the basement membrane and develop into deeper layers of the cervix. It can be classified into different stages depending on the depth of invasion and spread to nearby structures (Piątek Szymon i in., b.d.).

Advanced Cancer:

Stage	Invasion and spread
<i>I</i>	The cancer is confined to the cervix
<i>IA</i>	microscopic invasion
<i>B</i>	Visible invasion
<i>II</i>	the cancer extends to nearby structures such as the lower part of the vagina and/or the surrounding connective tissue but has not spread beyond the pelvis
<i>III</i>	The cancer spreads to the pelvic walls and/or the lower part of the vagina. It may obstruct the ureters and symptoms related to kidney function
<i>IV</i>	The cancer extends beyond the pelvis to distant organs, such as the bladder, rectum, liver, or lungs (Lepka i in., 2019) (Kozakiewicz, b.d.)

Table 1. The table shows the invasiveness of CC in relation to its stage.

At a given stage, we can identify clinical symptoms:

Symptoms	Stage
<i>May not present any symptoms and is often detected through routine screening</i>	Early
<i>Pain and Discomfort: pain in the lower abdomen, pain during intercourse, or a general feeling of discomfort.</i>	Later
<i>Abnormal Bleeding: Bleeding between menstrual periods, after intercourse, or after menopause.</i>	Later/advanced
<i>Discharge: Vaginal discharge that may have an unpleasant odor and be bloody or watery</i>	Later/advanced
<i>Urinary and Bowel Issues: problems with urination or bowel movements, as well as swelling of the legs.</i>	Advanced

Table 2. The table shows clinical symptoms at a given stage.

Risk Factors

Human Papillomavirus

Based on scientific publications regarding cervical cancer (CC) in the Polish population, several main risk factors associated with this disease can be identified (Andrzej i in., 2008). The most common of these is infection with the human papillomavirus (HPV) (Stefanek & Durka, 2014). HPV belongs to the genus of sexually transmitted papillomaviruses (Mađry i in., 2009). HPV can be categorized into low-risk or high-risk strains depending on their oncogenic potential. Low-risk strains may be asymptomatic or cause genital warts, while high-risk strains are oncogenic and can lead to dysplasia or cervical cancer. There are over 200 identified HPV strains, with types 16 and 18 responsible for 70% of all cervical cancer cases (Olejniak Anita, b.d.). Type 16 accounts for 50% of squamous cell carcinomas and up to 60% of all cervical cancers, while type 18 causes approximately 20% of cervical adenocarcinomas (Bebyn i in., 2022). After HPV infection, chronic inflammation can occur, allowing the virus to persist in the body and gradually cause cellular changes in the cervical epithelium, potentially leading to precancerous lesions (Bedell i in., 2020). These changes can include cellular abnormalities, dysplasia, carcinoma in situ, and cancer. The virus can damage the DNA of cervical epithelial cells by integrating its genetic material into the host cells. This damage can lead to uncontrolled cell growth and cancer development (Bowden i in., 2023). Effective preventive measures, such as vaccination against HPV and regular screening, are crucial for reducing the risk of cervical cancer and its progression.

Sex life

As we know, HPV is sexually transmitted, which is why sex life is the most important risk factor. Multiple sexual partners and early onset of sexual activity are considered high-risk factors for developing CC due to the sexually transmitted nature of HPV (Mak i in., 2004). High sexual activity increases exposure to various HPV types, including high-risk oncogenic strains. Each new sexual partner increases the risk of infection and prolonged exposure, heightening the risk of inflammation (Baltzer i in., 2017). In addition, a partner who has sexual intercourse with other partners, unprotected sexual intercourse, or polygamy are also among the risk factors (Ngoma & Autier, 2019).

Hormonal Contraception

Steroidal contraceptive hormones, especially in the context of oral contraception, may influence the development of cervical cancer, particularly when combined with human papillomavirus (HPV) infection. The proposed mechanism suggests that these hormones may promote the integration of HPV DNA into the host cell genome, which can increase the risk of carcinogenesis (Gadducci i in., 2020). Steroidal contraceptive hormones may also promote the integration of HPV DNA into the host genome and may bind to specific HPV DNA sequences in the transcriptional regulatory regions, thus increasing or inhibiting the transcription of various genes and potentially modulating cell apoptosis (Webster i in., 2001) (Marks i in., 2011). Estrogen and progesterone increase the level of apoptosis induced by HPV16 E2 and E7 proteins, and therefore, these hormones may protect cells from malignant transformation (de Villiers, 2003). Epidemiological data indicate that the use of hormonal contraception is associated with a 1.5 to 3.3 times higher relative risk of developing cervical cancer. This increased risk may result from the interaction between hormones and the regulatory sequences of the HPV virus, potentially affecting the expression of viral oncogenes. Additionally, hormones may inhibit apoptosis (programmed cell death), which facilitates uncontrolled growth of HPV-infected cells (Gadducci i in., 2020). Despite these potential mechanisms, it is important to understand that the risk associated with oral contraception is relative and can be modulated by other factors, such as the duration of contraceptive use, the presence of HPV infection, and other cervical cancer risk factors, such as smoking or the immune status of the individual (Gadducci i in., 2020). Long-term use of oral contraceptives may correlate with the risk of developing CC (Bovo i in., 2023). Taking contraceptive pills for more than 5 years may increase the risk of cervical cancer development (Medard M & Ostrowska, 2007). The use of combined oral contraceptives is associated with an increased risk of invasive cervical cancer. The relative risk for individuals

currently using oral contraceptives increases with the duration of use: using them for 5 or more years is associated with approximately a twofold increase in risk. The increased risk of cervical cancer associated with the use of combined oral contraceptives decreases over time after discontinuation. Interestingly, in cases of other types of cancer, contraception reduces the risk of their incidence (Gadducci i in., 2020).

Smoking

Smoking is associated with an increased risk of developing cervical cancer. Chemicals in cigarette smoke (Su i in., 2018), can damage the DNA of cervical cells and increase susceptibility to HPV infection, especially among long-term smokers (Bravi i in., 2014). The cervical mucus of smokers contains detectable amounts of cigarette components and their metabolites, such as benzo[a]pyrene (BaP), nicotine, and nitrosamines derived from nicotine, including 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (Fonseca-Moutinho, 2011). Regulation of HPV genome amplification by BaP may increase the risk of viral DNA integration into the host genome, which is a critical step in the development of cervical cancer. Long-term in vivo exposure to nicotine can lead to sustained cell proliferation, inhibition of apoptosis, and stimulation of vascular endothelial growth factor, resulting in increased microvessel density (Bravi i in., 2014). Quitting smoking was associated with a twofold reduction in risk. Giving up the habit provides significant benefits in protecting against cancer (Roura i in., 2014).

Education and knowledge

Individuals with low educational attainment may have limited knowledge about CC, its risk factors, and the importance of screening tests (Pasławska et al., 2014). They may be less aware of the need for Pap smears or HPV testing and may be less likely to utilize healthcare services or have difficulty accessing medical care (Kłapa, n.d.). Low education levels can lead to risky health behaviors, such as an unhealthy diet or improper sexual practices. Another risk factor is the low vaccination rate against HPV in Poland (Wysocki et al., 2012). According to the Ministry of Health, 35,932 boys and girls were vaccinated in 2021, and this number increased to 57,830 in 2022 (Bulletins, reports, epidemiological information, n.d.). Despite the availability of vaccines, low public awareness, concerns about vaccine safety, and distrust of vaccination programs contribute to low vaccination rates, which increases the risk of infection.

Preventive Measures in Poland

In Poland, various preventive measures are in place to reduce the incidence of cervical cancer. These include both vaccination programs against the human papillomavirus (HPV) and screening tests such as Pap smears or HPV tests.

Vaccination Programs

On June 1, 2023, the Ministry of Health introduced a nationwide HPV vaccination program that complements the free vaccination program for children and adolescents (*Szczepionka przeciw HPV*, b.d.). This program targets both girls and boys aged 9-15 and provides two free doses of the vaccine. Currently, three types of vaccines are available on the market. Cervarix: a bivalent vaccine targeting HPV types 16 and 18. Gardasil: a quadrivalent vaccine targeting HPV types 6, 11, 16, and 18. Gardasil 9: a nine-valent vaccine targeting HPV types 6, 11, 16, 18, 31, 33, 45, 52, and 58. (*Szczepionka przeciw HPV*, b.d.) All three vaccines are administered to prevent precancerous lesions of the genital organs and rectum (Trojańczyk, 2012). Promotional and educational activities are conducted to increase public awareness about the benefits of vaccination and the importance of protecting against cervical cancer.

Screening Tests

In Poland, several screening programs are available for the early detection of HPV (human papillomavirus) and cervical cancer. The main tests include cytology (Pap smear) and HPV testing. Cytology (Pap test or cytological smear) detecting precancerous changes and early forms of cervical

cancer. The test involves collecting a sample of cells from the surface of the cervix and examining them microscopically (Tuchowska i in., 2013). It allows the detection of abnormal cells that may indicate precancerous or cancerous changes. It is a rapid test available in pharmacies for patients to do themselves. It is recommended that women aged 25–59 have a cytological test every 3 years (Meggiolaro i in., 2016). This test is part of the National Cancer Control Program and is available free of charge within the healthcare system.

HPV Test (test for human papillomavirus) detecting HPV infection can lead to the development of cervical cancer. The test involves collecting a sample from the cervix and testing for high-risk types of HPV (e.g., HPV 16, HPV 18), while in men, the sample is taken from the penis or urethra (*Urologia Polska 2008/61/Supl. 1 - Diagnostyka zakażeń wirusem brodawczaka ludzkiego (HPV) skóry i błony śluzowej narządów płciowych męskich*, b.d.). It is a rapid test available in pharmacies for patients to do themselves. It should be noted that self-performance may reduce the sensitivity of the test, therefore it is recommended to use higher sensitivity tests for this type of trial (Nowak-Markwitz & Spaczyński, 2015). It was noted that the independent performance of the HPV test resulted in the screening of women who had never performed it before (Gravitt i in., 2011). HPV infection does not always lead to cancer, but its presence increases the risk of developing it. In some countries, the HPV test replaces cytology as a screening method for women over 30 (Janiszewska i in., 2015). In Poland, cytology is still more common, and the HPV test is often used as a follow-up for unclear cytology results. This test is available in Poland, but it is not covered by insurance. It can be performed privately.

Colposcopy is a supplementary test after abnormal cytology or HPV test results. Colposcopy involves examining the cervix with a colposcope, a specialized microscope that allows a detailed inspection of the cervical surface and the detection of any abnormalities. This is not a routine test and is usually done after abnormal cytology or HPV test results. It is available in Poland within the healthcare system as a follow-up test (Burness i in., 2020)

Cervical Biopsy confirming the diagnosis of cervical cancer or precancerous changes. A tissue sample is taken from the cervix during a biopsy for further histopathological examination. This invasive test confirms a diagnosis after abnormal results from other tests. A biopsy is performed in Poland as part of the diagnostic process following abnormal test results (Janiszewska i in., 2015)

Public health organizations, foundations, medical facilities, and the media run educational and informational campaigns about cervical cancer, its risk factors, preventive methods, and available treatment options. These efforts aim to increase public awareness about the importance of regular screenings and HPV vaccinations. Additionally, psychosocial support programs are available for women affected by cervical cancer and their families, offering emotional support, psychological counseling, and information about treatment options.

Education and Awareness

Public education and the availability of vaccination and screening programs are crucial for effective prevention (Nowakowski i in., 2013). Efforts include raising awareness about the importance of regular screenings and the benefits of vaccination against HPV. Support programs offer emotional and psychological support to affected women and their families (Pacewicz i in., 2012).

Future Prospects

The future of combating cervical cancer in Poland relies on the effective implementation and promotion of preventive strategies, the development of modern diagnostic and therapeutic methods, and further scientific research on HPV infection mechanisms and cervical cancer development (Magiera i in., 2024). Implementing integrated healthcare and public education programs can significantly reduce the burden of cervical cancer on Polish society.

Conclusion

Analyzing risk factors and preventive strategies in the context of cervical cancer in Poland is crucial for effectively managing this disease. Implementing effective vaccination programs (Pruski i in., 2023), conducting regular screenings, and educating the public are key to reducing the incidence and mortality associated with cervical cancer (Basta i in., 2019). Continuous scientific research and monitoring the effectiveness of preventive measures are necessary to continually improve strategies for combating this disease. Cervical cancer remains a significant health problem in Poland, and further efforts are needed to improve access to preventive programs, educate the public, and ensure equal access to modern diagnostic and therapeutic methods to reduce the occurrence and impact of this disease in the Polish population.

Bibliography

1. Andrzej, N., Iwona, B., & Ma, M. (2008). Zachowania zdrowotne kobiet w zakresie zapobiegania, wczesnego wykrywania stanów przedrakowych i raka szyjki macicy. *Ginekol Pol.*
2. Baltzer, N., Sundström, K., Nygård, J. F., Dillner, J., & Komorowski, J. (2017). Risk stratification in cervical cancer screening by complete screening history: Applying bioinformatics to a general screening population. *International Journal of Cancer*, 141(1), 200–209. <https://doi.org/10.1002/ijc.30725>
3. Basta, T., Knapp, P., Blecharz, P., Bodnar, L., Gawron, I., Babczyk, D., Piróg, M., Kluz, T., Markowska, A., Horbaczewska, A., & Jach, R. (2019). Current management of cervical cancer in Poland—Analysis of the questionnaire trial for the years 2002-2014 in relation to ASCO 2016 recommendations. *PLoS ONE*, 14(1), e0209901. <https://doi.org/10.1371/journal.pone.0209901>
4. Bebyn, M. G., Śledzińska, P., Wojtysiak, J., Jóźwicki, W., Mierzwa, T., Dziedzic, J., Kowalewski, J., & Lewandowska, M. A. (2022). HPV RNA and DNA testing in Polish women screened for cervical cancer – A single oncological center study. *European Journal of Obstetrics and Gynecology and Reproductive Biology*, 268, 129–134. <https://doi.org/10.1016/j.ejogrb.2021.11.427>
5. Bedell, S. L., Goldstein, L. S., Goldstein, A. R., & Goldstein, A. T. (2020). Cervical Cancer Screening: Past, Present, and Future. *Sexual Medicine Reviews*, 8(1), 28–37. <https://doi.org/10.1016/j.sxmr.2019.09.005>
6. Bovo, A. C., Pedrão, P. G., Guimarães, Y. M., Godoy, L. R., Resende, J. C. P., Longatto-Filho, A., & Reis, R. dos. (2023). Combined Oral Contraceptive Use and the Risk of Cervical Cancer: Literature Review. *RBGO Gynecology & Obstetrics*, 45(12), e818–e824. <https://doi.org/10.1055/s-0043-1776403>
7. Bowden, S. J., Doulgeraki, T., Bouras, E., Markozannes, G., Athanasiou, A., Grout-Smith, H., Kechagias, K. S., Ellis, L. B., Zuber, V., Chadeau-Hyam, M., Flanagan, J. M., Tsilidis, K. K., Kalliala, I., & Kyrgiou, M. (2023). Risk factors for human papillomavirus infection, cervical intraepithelial neoplasia and cervical cancer: An umbrella review and follow-up Mendelian randomisation studies. *BMC Medicine*, 21, 274. <https://doi.org/10.1186/s12916-023-02965-w>
8. Bravi, F., Parazzini, F., Cipriani, S., Chiaffarino, F., Ricci, E., Chiantera, V., Viganò, P., & La Vecchia, C. (2014). Tobacco smoking and risk of endometriosis: A systematic review and meta-analysis. *BMJ Open*, 4(12), e006325. <https://doi.org/10.1136/bmjopen-2014-006325>
9. Bray, F., Laversanne, M., Sung, H., Ferlay, J., Siegel, R. L., Soerjomataram, I., & Jemal, A. (2024). Global cancer statistics 2022: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. *CA: A Cancer Journal for Clinicians*, 74(3), 229–263. <https://doi.org/10.3322/caac.21834>
10. Burd, E. M. (2003). Human papillomavirus and cervical cancer. *Clinical Microbiology Reviews*, 16(1), 1–17. <https://doi.org/10.1128/CMR.16.1.1-17.2003>
11. Burness, J. V., Schroeder, J. M., & Warren, J. B. (2020). Cervical Colposcopy: Indications and Risk Assessment. *American Family Physician*, 102(1), 39–48.
12. de Villiers, E.-M. (2003). Relationship between steroid hormone contraceptives and HPV, cervical intraepithelial neoplasia and cervical carcinoma. *International Journal of Cancer*, 103(6), 705–708. <https://doi.org/10.1002/ijc.10868>
13. Didkowska, J., Wojciechowska, U., Michalek, I. M., & Caetano dos Santos, F. L. (2022). Cancer incidence and mortality in Poland in 2019. *Scientific Reports*, 12, 10875. <https://doi.org/10.1038/s41598-022-14779-6>
14. Fonseca-Moutinho, J. A. (2011). Smoking and Cervical Cancer. *International Scholarly Research Notices*, 2011(1), 847684. <https://doi.org/10.5402/2011/847684>
15. Gadducci, A., Cosio, S., & Fruzzetti, F. (2020). Estro-progestin Contraceptives and Risk of Cervical Cancer: A Debated Issue. *Anticancer Research*, 40(11), 5995–6002. <https://doi.org/10.21873/anticancer.14620>
16. Janiszewska, M., Kulik, T., Dziedzic, M., & Żołnierczuk-Kieliszek, D. (2015). *Śródnabłonkowa neoplazja raka szyjki macicy – diagnoza, profilaktyka*.
17. Kozakiewicz, B. (b.d.). Malignant neoplasms of genitals. *Nowa Medycyna*. Pobrano 17 wrzesień 2024, z <https://www.czytelniamedyczna.pl/1512,nowotwory-zoliwe-narzdu-rodnego.html>
18. Lepka, P., Jedryka, M., Misiek, M., Matkowski, R., & Zalewski, K. (2019). An update of the cervical cancer staging system as of 2019. *Current Gynecologic Oncology*, 17, 10–18. <https://doi.org/10.15557/CGO.2019.0002>
19. Mađry, E., Gibas-Dorna, M., Adamczak-Ratajczak, A., & Mađry, R. (2009). HPV - Multiple face virus. *Family Medicine and Primary Care Review*, 11, 702–704.
20. Magiera, K., Rybak, J., Magiera, B., Grabarczyk, A., & Grabowska-Szczurek, M. (2024). Rak szyjki macicy – czynniki ryzyka i nowe możliwości profilaktyki w Polsce. *Medycyna Środowiskowa*, 26(3–4), 109–113. <https://doi.org/10.26444/ms/178419>

21. Mak, R., Van Renterghem, L., & Cuvelier, C. (2004). Cervical smears and human papillomavirus typing in sex workers. *Sexually Transmitted Infections*, 80(2), 118–120. <https://doi.org/10.1136/sti.2002.003749>
22. Marks, M., Gravitt, P. E., Gupta, S. B., Liaw, K.-L., Kim, E., Tadesse, A., Phongnarisorn, C., Wootipoom, V., Yuenyao, P., Vipupinyo, C., Rugpao, S., Sriplienchan, S., & Celentano, D. D. (2011). The association of hormonal contraceptive use and HPV prevalence. *International Journal of Cancer*, 128(12), 2962–2970. <https://doi.org/10.1002/ijc.25628>
23. Medard M, L., & Ostrowska, L. (2007). [Combined oral contraception and the risk of reproductive organs cancer in women]. *Ginekologia Polska*, 78(8), 637–641.
24. Meggiolaro, A., Unim, B., Semyonov, L., Miccoli, S., Maffongelli, E., & La Torre, G. (2016). The role of Pap test screening against cervical cancer: A systematic review and meta-analysis. *La Clinica terapeutica*, 167, 124–139. <https://doi.org/10.7417/CT.2016.1942>
25. Ngoma, M., & Autier, P. (2019). Cancer prevention: Cervical cancer. *ecancermedicalscience*, 13, 952. <https://doi.org/10.3332/ecancer.2019.952>
26. Nowakowski, A., Jackowska, T., Oszukowski, P. J., Radowski, S., Wysocki, J., & Zatoński, W. (2013). Profilaktyka raka szyjki macicy – problem interdyscyplinarny. Czy i jak możemy poprawić sytuację w Polsce? *Pediatrica Polska*, 88(4), 340–346. <https://doi.org/10.1016/j.pepo.2013.05.005>
27. Nowicki, A., Farbicka, P., & Anczura, K. (2017). Wiedza uczennic technikum i absolwentek studiów wyższych z zakresu profilaktyki i leczenia raka szyjki macicy. *Pielęgniarstwo Polskie*, 64(2), 246–253. <https://doi.org/10.20883/pielpol.2017.32>
28. Olejnik Anita. (b.d.). *HPV jako czynnik etiologiczny raka szyjki macicy*.
29. Pacewicz, M., Krajewska-Kulak, E., & Krajewska-Ferishah, K. (2012, październik 1). *Profilaktyka raka szyjki macicy—Poziom wiedzy kobiet i mężczyzn. | Palliative Medicine / Medycyna Paliatywna | EBSCOhost*. <https://openurl.ebsco.com/contentitem/gcd:85489954?sid=ebsco:plink:crawler&id=ebsco:gcd:85489954>
30. Piątek Szymon, Bidziński Mariusz, Panek Grzegorz, Wielgoś Mirosław, & Sobiczewski Piotr. (b.d.). *Carcinoma of the cervix uteri—Staging according to FIGO 2018 classification*.
31. Pruski, D., Millert-Kalińska, S., Łagiedo, M., Sikora, J., Jach, R., & Przybylski, M. (2023). Effect of HPV Vaccination on Virus Disappearance in Cervical Samples of a Cohort of HPV-Positive Polish Patients. *Journal of Clinical Medicine*, 12(24), 7592. <https://doi.org/10.3390/jcm12247592>
32. *Raporty | Krajowy Rejestr Nowotworów*. (b.d.). Pobrano 5 czerwiec 2024, z <http://onkologia.org.pl/pl/raporty>
33. Revathidevi, S., Murugan, A. K., Nakaoka, H., Inoue, I., & Munirajan, A. K. (2021). APOBEC: A molecular driver in cervical cancer pathogenesis. *Cancer Letters*, 496, 104–116. <https://doi.org/10.1016/j.canlet.2020.10.004>
34. Robert Jach, Magda Dumin, Małgorzata Radoń-Pokracka, Antoni Basta, Zbigniew Kojs, Kazimierz Karolewski, Małgorzata Klimek, & Paweł Blecharz, Grzegorz Ziętarski, Joanna Dulińska-Litewka. (b.d.). *Zakażenie HPV i jego związki z nowotworami złośliwymi*.
35. Roura, E., Castellsagué, X., Pawlita, M., Travier, N., Waterboer, T., Margall, N., Bosch, F. X., de Sanjosé, S., Dillner, J., Gram, I. T., Tjønneland, A., Munk, C., Pala, V., Palli, D., Khaw, K.-T., Barnabas, R. V., Overvad, K., Clavel-Chapelon, F., Boutron-Ruault, M.-C., ... Riboli, E. (2014). Smoking as a major risk factor for cervical cancer and pre-cancer: Results from the EPIC cohort. *International Journal of Cancer*, 135(2), 453–466. <https://doi.org/10.1002/ijc.28666>
36. Stefanek, A., & Durka, P. (2014). *POZIOM ŚWIADOMOŚCI KOBIET NA TEMAT PROFILAKTYKI RAKA SZYJKI MACICY*.
37. Su, B., Qin, W., Xue, F., Wei, X., Guan, Q., Jiang, W., Wang, S., Xu, M., & Yu, S. (2018). The relation of passive smoking with cervical cancer. *Medicine*, 97(46), e13061. <https://doi.org/10.1097/MD.00000000000013061>
38. *Szczepionka przeciw HPV*. (b.d.). *Szczepienia.Info*. Pobrano 5 czerwiec 2024, z <https://szczepienia.pzh.gov.pl/szczepionki/hpv/>
39. Trojańczyk, M. (2012). *HPV – szczepić czy nie szczepić?*
40. Tuchowska, P., Worach-Kardas, H., & Marcinkowski, J. T. (2013). *Najczęstsze nowotwory złośliwe w Polsce – główne czynniki ryzyka i możliwości optymalizacji działań profilaktycznych*.
41. *Urologia Polska 2008/61/Supl. 1—Diagnostyka zakażeń wirusem brodawczaka ludzkiego (HPV) skóry i błony śluzowej narządów płciowych męskich*. (b.d.). Pobrano 16 wrzesień 2024, z <http://www.urologiapolska.pl/artykul.php?3239>

42. Webster, K., Taylor, A., & Gaston, K. (2001). Oestrogen and progesterone increase the levels of apoptosis induced by the human papillomavirus type 16 E2 and E7 proteins. *The Journal of General Virology*, 82(Pt 1), 201–213. <https://doi.org/10.1099/0022-1317-82-1-201>