Bandala-Jacques *et al. BMC Cancer* (2025) 25:1441
https://doi.org/10.1186/s12885-025-14904-4

BMC Cancer

SYSTEMATIC REVIEW **Open Access**

Oropharyngeal cancer risk groups in the United States: a meta-analysis and SEER analysis

Antonio Bandala-Jacques[1], Ian J. Saldanha[1,2] and Gypsyamber D'Souza[1*]

Abstract

Background Incidence of oropharyngeal cancer (OPC) in the United States (US) has increased significantly due to human papillomavirus infections. This study characterized OPC incidence rates (IRs) by subgroups to identify high-risk populations for future screening efforts.

Methods We conducted a comprehensive analysis comprising a systematic review and meta-analysis of studies published since 2000 reporting OPC IRs in the US, supplemented by a primary analysis of data from the Surveillance, Epidemiology, and End Results (SEER) program. From each source, we extracted cases and person-years to calculate IRs per 100,000 person-years (PY). Data from 11 studies selected for meta-analysis were analyzed using random-effects models. We stratified analyses by sex, age, race/ethnicity, and HIV status, as available.

Results The meta-analysis found an overall OPC IR of 8.2 per 100,000 PY. Men had four times the incidence of OPC than women (13.4 and 3.6 per 100,000 PY, respectively). People living with HIV (PLWH) had the highest IR (27.6 per 100,000 PY), particularly men LWH (35.2 per 100,000 PY, compared to 12.4 in women LWH). SEER data confirmed sex differences, with low incidence in men < 50 years and in women of all ages, and a peak IR among men 60–79 years (32.4 per 100,000 PY). Among men aged 60–79, IRs were higher among White, Black, and American Indian/Alaskan Native (25.7–39.6 per 100,000 PY) than Hispanic/Latino or Asian American/Pacific Islander men (8.8–16.7 per 100,000 PY).

Conclusions Men aged 50–79 years and PLWH are at highest risk for OPC, with IRs comparable with or exceeding those of other HPV-associated cancers in the US. Any future screening efforts should prioritize these groups while excluding low-risk populations, such as women and individuals < 50 years.

Registration: PROSPERO CRD42024588671.

Keywords Oropharyngeal cancer, Human papillomavirus, HPV, incidence rate

*Correspondence:
Gypsyamber D'Souza
gdsouza2@jhu.edu
[1]Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, 615 N. Wolfe Street. Baltimore, Baltimore 21205, MD, USA
[2]Department of Health Policy and Management, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, USA

Background

The incidence of oropharyngeal cancer (OPC) in the United States (US) has increased in recent decades, driven by an increase in OPC caused by human papillomavirus (HPV). Decreasing tobacco use [1] and increasing oral sex practices [2] have changed the epidemiology of these cancers, and most OPCs in the US are now caused by HPV. The incidence of OPC has increased by 1.6% per year for more than 25 years, most notably among middle-aged and older-aged men [3, 4]. Indeed, the number of newly diagnosed OPCs per year in the US has surpassed the number of newly diagnosed cervical cancers, making it the most common HPV-associated cancer in the US, with an average of 22,000 cases diagnosed every year, 71% among men [5]. Recent US data from 2011 to 2019 suggest that in people under 45 years, most sexually active men (87–89%) and women (85–89%) performed oral sex in the past year [6]. Due to HPV vaccination uptake, initial reductions in OPC incidence have been observed among 20–45 year olds [7], but not among older ages, where most OPCs occur, as most people over 40 in the US have not received the HPV vaccine. We expect HPV-OPC incidence rates to decrease in the future as the age-cohorts that have received HPV vaccination enter ages where more OPC occurs, although modeling studies suggest overall OPC rates will not decrease before 2045 [8, 9].

Currently, there are no US Food and Drug Administration (FDA)-approved methods for screening for OPC, and most cases are diagnosed in late stages [10]. Primary HPV screening is now a standard method for another HPV-associated cancer (cervical cancer), where it has been shown to effectively identify those at increased risk [11, 12]. The utility of using HPV biomarkers for OPC screening is less clear. HPV16 E6 antibodies [13] and persistent oncogenic oral HPV infection [14] have been detected years before HPV-related OPC, suggesting that they may be useful for identifying individuals at increased risk of HPV-related OPC (HPV-OPC). However, it is unclear whether screening for HPV-OPC is appropriate, because there is no known treatment for healthy, biomarker-positive individuals beyond enhanced surveillance. Furthermore, there is no clear evidence regarding the benefits and harms of HPV-OPC screening in terms of morbidity or mortality [15].

Given the increasing incidence of OPC [4], and the high morbidity associated with OPC treatment [16], there is clear motivation to develop effective screening tests for OPC. If screening tests were determined to have high sensitivity and specificity and the benefits of early detection from screening were proven, we would need to understand who to screen. An analysis of risk groups for another HPV-associated cancer, anal cancer, reported incidence by subgroups to explore groups at high risk [17]. That analysis revealed anal cancer risk differences by age, sex, sexual orientation, HIV status, and genital cancer history. When anal cancer screening effectiveness was recently demonstrated in the ANCHOR randomized trial, the published data regarding risk groups helped inform who should be targeted for screening [18, 19]. The description of incidence rates (IRs) of OPC by subgroup could similarly help identify populations at low-, medium-, and high-risk for OPC to inform which groups would and would not be appropriate to consider for OPC screening. Therefore, we designed a comprehensive analysis to describe the IRs of OPC among subgroups to characterize risk groups with a higher OPC incidence. This analysis included a systematic review and meta-analysis as well as a primary analysis of data from the Surveillance, Epidemiology, and End Results (SEER) program.

Methods

Methods overview We performed a systematic review and meta-analysis of the literature. While this meta-analysis contained key data, it was limited by the subgroups that each study reported and by the limited number of studies for some subgroups. Therefore, we performed a complementary analysis by leveraging an additional data source (SEER) to estimate OPC incidence for additional subgroups not available in the meta-analysis data.

Systematic review with meta-analysis

Eligibility criteria We included studies published since January 1, 2000 that reported IRs of OPC in US populations, as well as the corresponding number of person-years and cases. We defined OPC according to the site and histology ICD-O-3 codes adopted by the Centers for Diseases Control and Prevention [20]. We excluded studies that combined incidence rates of oropharyngeal and oral cavity cancer.

Information sources and search strategy We searched Medline, PubMed Central (PMC), and BookShelf via PubMed on September 20, 2024. Our search query (Additional file 1) included controlled-vocabular and free-text terms related to IR, OPC, and US.

Study selection Two reviewers independently screened all titles for inclusion in the abstract stage of review, and then independently screened the abstracts of all relevant titles. Next, the two reviewers independently screened potentially eligible full-text articles, including supplemental data. Articles that presented IRs only within figures were excluded. At all stages, discrepancies were resolved through discussion.

Data extraction One reviewer extracted the data, and a second reviewer independently confirmed the extracted data. We extracted data pertaining to the study characteristics, years over which the IRs were calculated, IR per 100,000 person-years, number of OPC cases, and person-years of follow-up. We extracted the overall IR and IRs stratified by sex, race, HIV status, and age, as available.

For studies that reported IRs but omitted person-time and/or the number of cases used in their calculations, we emailed the authors to request information. Because some studies used the same data source for calculations (mainly multiple articles using SEER data), we included only one article from those that overlapped in time and population, prioritizing articles that reported data for the maximum person-time.

Risk of bias We assessed the risk of bias in each included study using a modified version of the tool by Hoy et al. [21] Because we are not aware of specific tools for assessing risk of bias in studies of IRs, we modified the tool by adding two items related to follow-up duration and losses to follow-up. We then classified each study into low, medium, or high risk of bias. Two independent reviewers independently assessed the risk of bias in each included study and resolved discrepancies through discussion.

Statistical analysis All statistical analyses were performed using R, Version 4.3.2 (R Core Team 2023, Vienna, Austria) via RStudio, Ocean Storm release (Posit Team 2023, Boston, USA). For the meta-analysis, we used the metafor package [22]. To calculate the pooled estimates, we used random-effects models using the Hartung-Knapp-Sidik-Jonkman modification [23]. We assessed heterogeneity by calculating the I-squared statistic and Cochran's Q (test for heterogeneity).

Registration and reporting We registered this systematic review in PROSPERO (registration number CRD42024588671). This systematic review adhered to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA).

SEER data analysis 2011–2022

We obtained data from SEER-17, which includes incident cancers diagnosed in 17 registries up to 2022. We used the SEER*Stat Software to extract OPC cases and person-years by age group, sex, race, and ethnicity. We extracted annual data from 2011 to 2022. We defined OPC using ICD-O-3 codes for site and histology. We aggregated cases and person-years for the study period and calculated IRs per 100,000 person-years overall and by sex, age, and race strata using RStudio. We defined risk groups for OPC as low, medium, or high based on incidence rates of cancers that are currently screened for in the United States, and by the distribution of our data. We chose an incidence rate of 24 per 100,00 person-years as the lower limit for "high-risk" in order to not artificially separate race and age groups close to 25 cases per 100,000 person-years. Similarly, We chose an incidence rate below 10 per 100,000 person-years as "low risk", as delineated by similar groups under this threshold in our data.

Results

Systematic review and meta-analysis

A PRISMA flowchart of study identification for the systematic review is presented in additional file 2. Our search identified 2,210 records, 61 of which were screened in full text. The most common reasons for the exclusion of full-text articles were that IRs were only reported in figures ($n = 13$), person time and/or events were unobtainable ($n = 13$), and the studies reported overlapping SEER data with another study ($n = 11$). The detailed list of exclusions is presented in additional file 3. We finally included 11 studies in the systematic review [4, 24–33].

The 11 included studies reported cases of OPC diagnosed between 1990 and 2017 using data from SEER, Medicaid, and Veterans Affairs. Five studies reported overall IRs [24, 26, 28, 29, 31] and another four studies reported stratified rates that could be combined for overall estimates [4, 25, 27, 33]. A subset of studies reported IRs stratified by sex ($n = 4$) [4, 27, 29, 33], race or ethnicity ($n = 3$) [25, 32, 33], and HIV status ($n = 3$) [27, 28, 30]. Therefore, studies could each contribute to one or more meta-analyses.

The meta-analyzed IRs of OPC are shown in Fig. 1 for the overall population (1A) as well as specific estimates for men (1B), women (1C), and people living with HIV (PLWH) (1D). The overall IR of OPC in the US was 8.2 cases per 100,000 person-years (PY) (95% confidence interval [CI] 4.1, 12.3; 9 studies). When stratified by sex, the incidence was almost 4-fold higher among men (13.4 per 100,000 PY, 95% CI 3.8, 22.9; 4 studies) than among women (3.6 per 100,000 PY, 95% CI 0.6, 6.5; 4 studies). The highest IR for any group was for PLWH, for whom the IR was 27.6 per 100,000 PY (95% CI 21.6, 33.5; 3 studies). Both men and women LWH had elevated OPC incidence, although the IR was especially high among men LWH (35.2 per 100,000 PY), three times higher than among women LWH (12.4 per 100,000 PY) (Fig. 2).

We next explored differences in OPC incidence according to sex, race, and ethnicity (Fig. 2). OPC IRs were highest among White men (16.9 per 100,000 PY, 95% CI 8.7, 25.2; 3 studies) and Black men (14.7 per 100,000 PY, 95% CI 0.0, 41.5; 3 studies). Incidence was lower for men who were Hispanic or Latino (4.9 per 100,000 PY, 95% CI 0.0, 14.0; 3 studies) or Asian American or Pacific

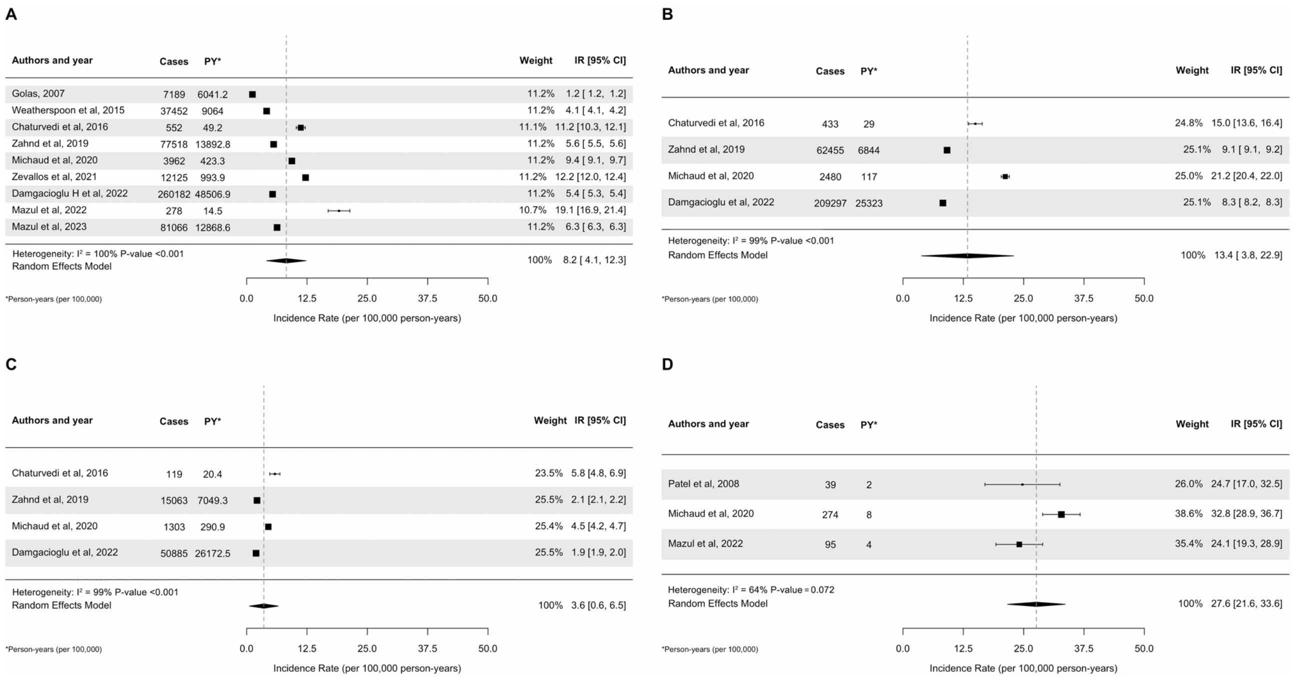

Fig. 1 Forest plots of meta-analyzed incidence rates of oropharyngeal cancer in the overall population (**A**), men (**B**), women (**C**), and people living with HIV (**D**)

Islanders (2.0 per 100,000 PY, 95% CI 0.0, 6.8; 2 studies), and women of all races/ethnicities (IRs ranging from 0.7 to 3.6 per 100,000 PY; 4 studies in total).

Of the 11 studies, we rated seven as low risk of bias and four as moderate or high risk (Additional file 4). Moderate and high risk of bias ratings were mostly related to study populations that did not reflect our target population. This included one Medicaid [27] and two US veteran [26, 28] studies because their study populations may not reflect our target population, and one study [31] because its definition of OPC was narrower than ours. We rated the two studies among US veterans at a moderate risk of bias for sex-stratified estimates but a high risk of bias for the overall OPC estimate, given the higher proportion of men in their population than the overall US population. When assessing the heterogeneity of the included studies, all but one of the pooled estimates had I-squared estimates ranging from 98 to 100%, indicating that the variability across study estimates was predominantly due to heterogeneity rather than chance. The results of formal tests (Q-tests) were statistically significant. The estimates for PLWH showed less heterogeneity (I-squared = 64%, $p = 0.072$).

SEER analysis

The SEER dataset contains 56,352 incident OPC cases diagnosed between 2011 and 2022 from 17 US cancer registries. The IR for OPC in the SEER dataset was 5.5 per 100,000 PY overall, 9.1 per 100,000 PY for men, and 1.8 per 100,000 PY for women (Fig. 3). When compared with sex-stratified estimates from the meta-analysis, inferences were consistent, and we were able to calculate age-stratified IRs by sex using the SEER data (Fig. 3). Regardless of sex, we found low incidence rates for those under 50 years of age, which increased with age until they peaked at 60–79 years and then declined (Additional file 5).

Next, we explored racial differences in OPC incidence using SEER data. OPC incidence by race followed the same pattern as the meta-analyzed data. For each race or ethnicity, men had a higher IR than women did. For example, among White individuals, IRs were 14.2 for men and 2.7 for women, and among Black individuals, 6.1 for men and 1.3 for women. Additionally, SEER had data for American Indian/Alaska Native individuals (which the meta-analysis did not) and showed an IR of 4.5 per 100,000 PY overall (7.7 in men and 1.3 in women).

We then explored differences by age and sex among each race group (Fig. 4, Additional File 6) and found consistently low rates of OPC (< 10 per 100,000 PY) among individuals under 50 years of age, regardless of sex or race. OPC incidence was elevated (≥ 24 per 100,000 PY, "high risk") among 60-79-year old men who were White (39.6 per 100,000 PY), Black (25.7 per 100,000 PY), or American Indian/Alaska Native (32.7 per 100,000 PY). Among 50-59-year olds, only White men were also in the "high risk" category (28.6 per 100,000 PY, Fig. 4). Rates were also increased, although not as high (10 - <24 per 100,000 PY, "medium risk"), among many men 50–59 and over 80 years old (Fig. 4). This included "medium

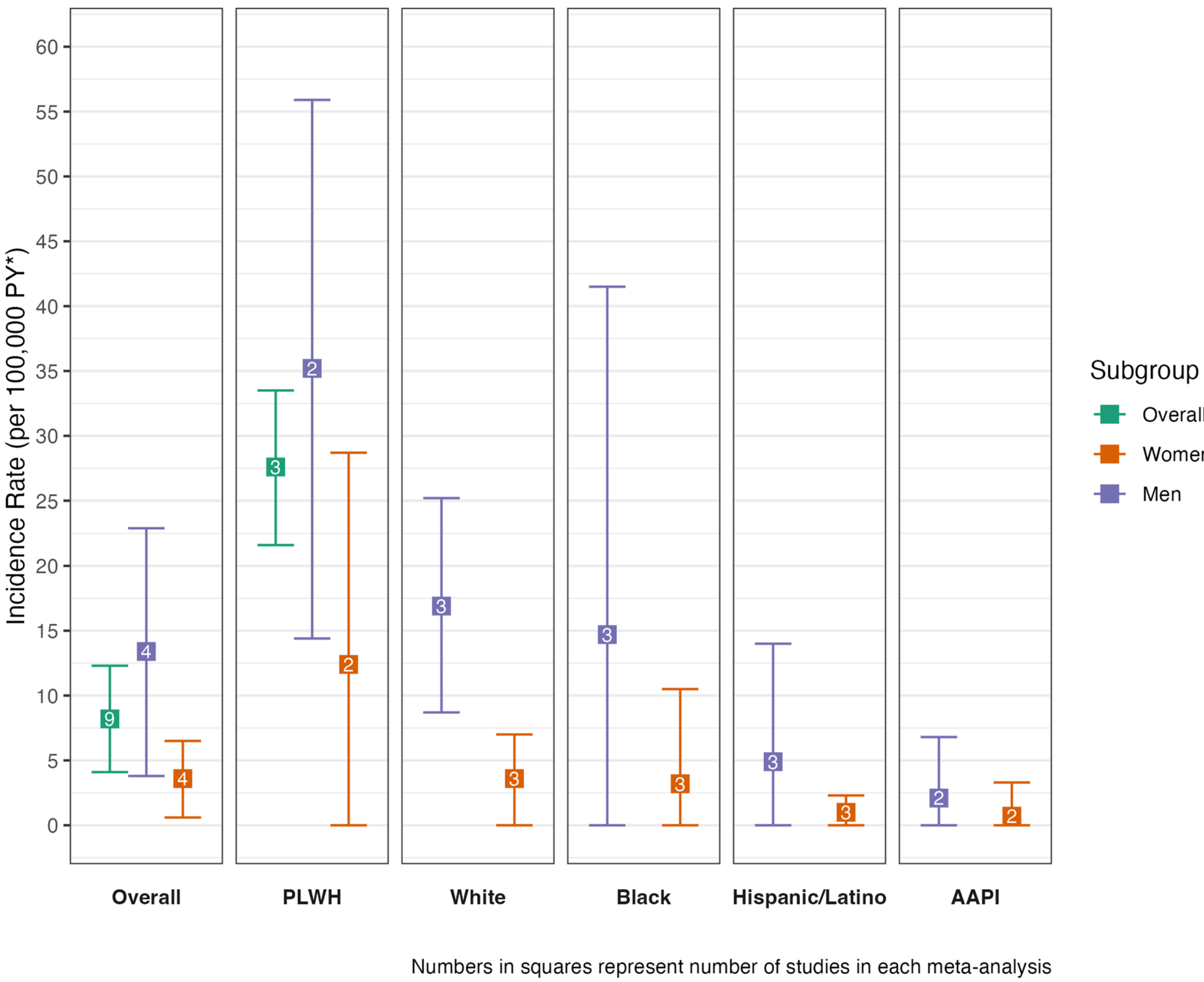

Fig. 2 Meta-analyzed incidence rates of oropharyngeal cancer by subgroup. Abbreviations: AAPI = Asian Americans or Pacific Islanders; PLWH = persons living with HIV; PY = person-years. Number of studies contributing to each estimate is indicated in squares

risk" for White, Black, Hispanic/Latino, and American Indian/Alaska Native men over 80 (range 12.6–20.0 per 100,000 PY), Black and American Indian/Alaska Native men 50–59 (16.4–17.3 per 100,000 PY), and Hispanic/Latino men 60–79 years old (16.7 per 100,000 PY). Asian American and Pacific Islander (AAPI) men of all ages had IRs lower than 10 per 100,000 PY. Women of all race and age groups had IRs < 10 per 100,000 PY, with the highest incidence in White women 60–79 years old (7.0 per 100,000 PY).

Discussion

Using nationally representative data from the US, our analysis identified high-, medium-, and low-risk populations for OPC, which can inform future OPC screening efforts. Low-risk groups included all individuals under 50 years of age and women in the general population, regardless of age, suggesting that these groups should not be considered for OPC screening. Several high-risk groups were identified, including men aged ≥ 50 years and men living with HIV. The OPC incidence among men of all ages in our study (9.1 per 100,000 PY) was higher than the IR for other HPV-associated cancers in the US, including cervical cancer in women [34] (7.6 per 100,000 PY) and anal cancer [5] (1.6 for men and 2.4 for women per 100,000 PY).

Our systematic review and meta-analysis found clear and consistent data showing that OPC incidence is almost 4-fold higher in men than in women, consistent with previous studies [3, 35]. Previous research has shown that the higher OPC risk among men is only partially explained by a higher number of oral sexual partners [36]. Studies suggest that higher OPC risk among men may be due to a higher risk of HPV acquisition when performing oral sex on a woman than on a man [37], and/or lower immune response to HPV among men [38],

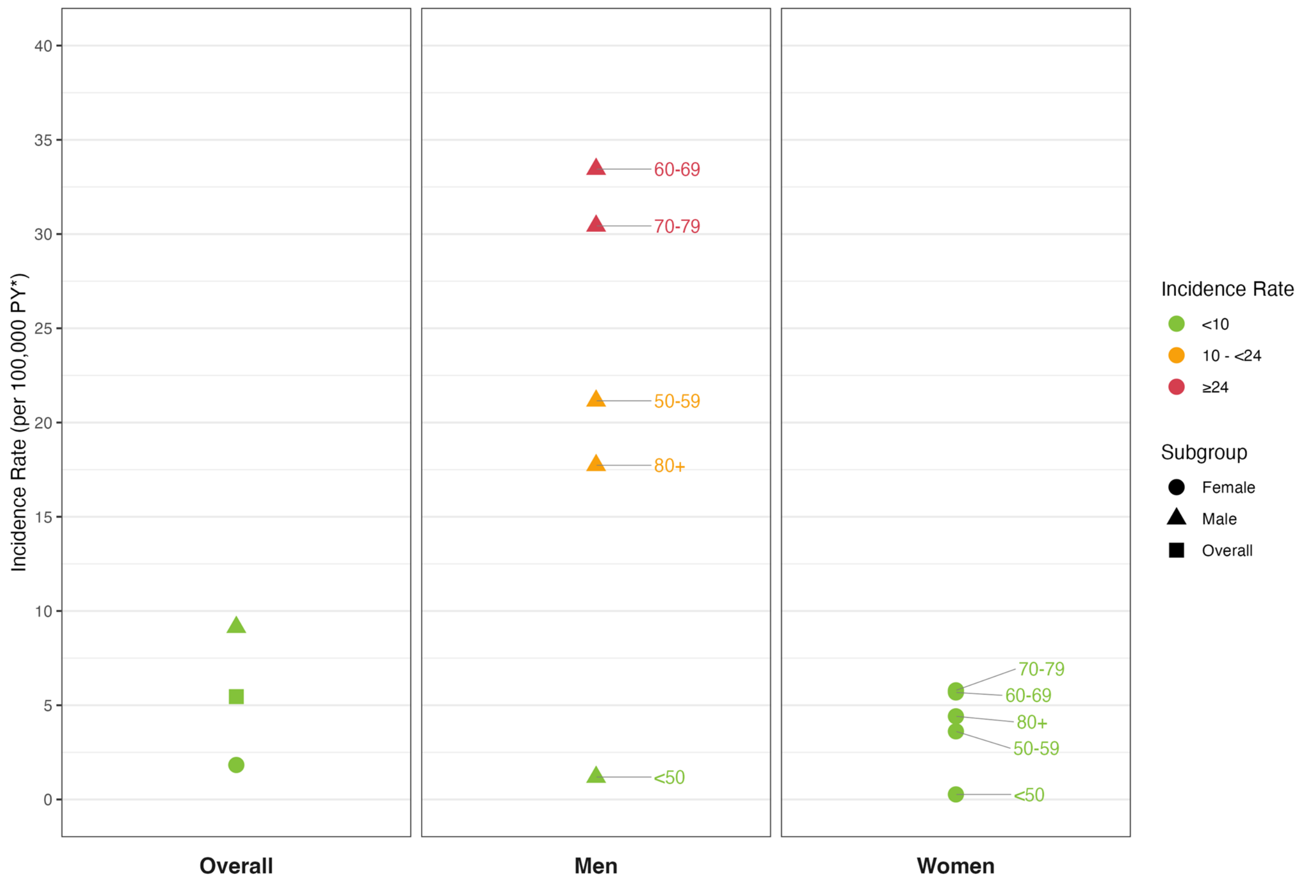

Fig. 3 SEER incidence rate of oropharyngeal cancer by sex and age

thereby decreasing oral HPV clearance. Since HPV vaccination for men was introduced in the US in 2009 and recommended for those aged 9–26 years, most men in the US currently aged 40 years or older are unvaccinated and remain at risk for HPV-associated diseases [4].

Our comprehensive analysis is the first to delineate clear OPC risk groups, utilizing sex-, age-, and race-stratified risk. Among both men and women, OPC incidence is low until the age of 50 years, peaks between ages 60 and 79 years, and decreases slightly after age 80 years. We found that men 50–79 years of age had the highest OPC risk in all general population groups, with an incidence of 27.3 per 100,000 PY. When restricting to men 60–79 years of age, OPC incidence was higher at 32.4 per 100,000 PY; however, screening at that age would miss many cases because half of the current OPC cases are diagnosed before age 62 [5]. The cancer incidence levels observed among 50-79-year old men are levels at which cancer screening might be considered feasible, although they are lower than the incidence of some other commonly screened cancer among men of this age (lung, colorectal, and advanced prostate cancer incidence are each > 50 per 100,000 PY for men aged 50–64 years and > 100 among men aged 65–74 years) [39].

Our analysis found that there were differences in OPC incidence according to race and ethnicity. Among men over 50, White men had the highest OPC incidence, followed by American Indian/Alaska Native and Black men (33.5, 24.9, and 20.8 per 100,000 PY, respectively), similar to several previous studies [32, 40]. Research suggests that differences in oral sexual behavior by race may explain racial differences observed in oral HPV infection and thus the differences in HPV-OPC incidence [41]. As behaviors change, these differences in incidence can also change, so caution should be taken in targeting risk categories by race. HPV now causes the majority of OPC in the US among all racial groups [42]. While there are differences in exact IRs by race, overall recommendations of OPC risk levels (high, medium, low) are similar across race groups and do not warrant race-based screening. The higher IR of OPC among PLWH in this study is consistent with the higher risks of many other cancers in PLWH compared with people without HIV [43]. This includes a higher risk of other HPV-related cancers, such as anal [17], penile [44], vulvar [45], and cervical cancers [46]. This higher risk has led to recommendations for increased cancer screening for cervical and anal cancers for those living with HIV [18, 47].

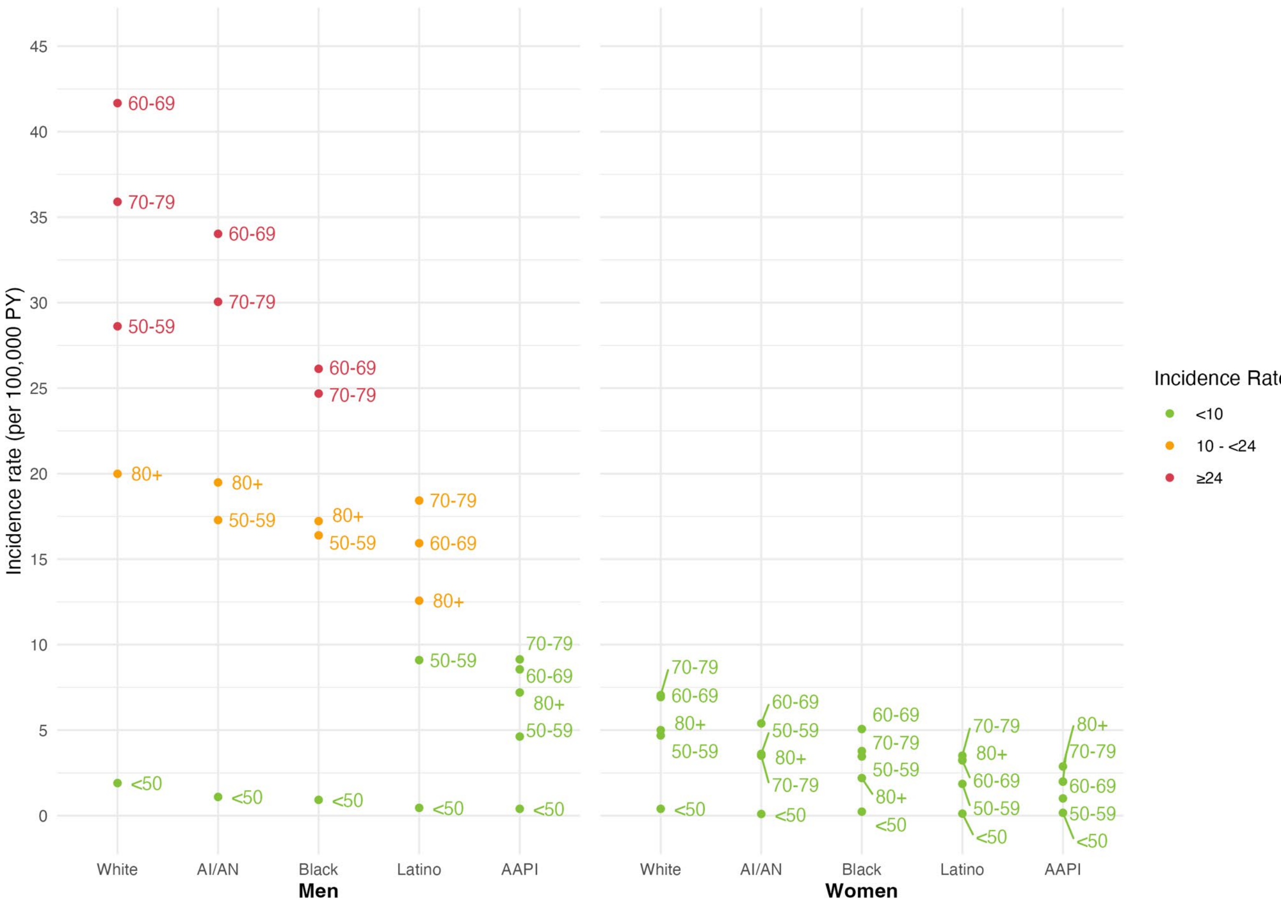

Fig. 4 SEER incidence rate of oropharyngeal cancer by sex and age, when further stratified by race and ethnicity. Abbreviations: AI/AN = American Indian/Alaskan Native; AAPI = Asian Americans or Pacific Islanders; PY = person-years

The estimates of OPC incidence in our study were slightly higher in the meta-analysis than in the SEER analysis (IR of 13.4 vs. 9.1 per 100,000 PY for men, IR of 3.6 vs. 1.8 cases per 100,000 PY for women). This is likely explained by the non-representative ages of the populations included in some of the studies in the meta-analysis, such as older populations in the study using data from Medicaid. Conclusions about risk groups were similar in the meta-analysis and SEER analysis, although the SEER data are a better reflection of the US population risk levels for each group. In the meta-analysis, high I-squared estimates suggested heterogeneity among studies, and the pooled estimates reflected this variation. Studies have used distinct source populations that vary dramatically in size, contributing to heterogeneity across estimates. However, the similar risk groupings identified by both the meta-analysis and the SEER analysis reinforce the conclusions about which risk groups are at increased risk, although the exact estimates may vary.

This study had several strengths and limitations. We used two robust and complementary epidemiologic analyses to determine the current OPC risk in the US. SEER analysis enabled well-powered stratified risk estimates in the general US population. The meta-analysis provided combined data from multiple recent US studies, allowing more precision than single studies, and included some data on PLWH not available for the SEER analysis. However, the meta-analysis was limited by the modest number of studies and high heterogeneity. Some subgroups, such as men and women with a history of anogenital cancer, may have elevated OPC risk but did not have data available to inform this analysis. Furthermore, SEER*Stat does not report HPV tumor status, so we were unable to calculate the proportion of OPC that are caused by HPV, though most OPCs in the US are HPV-related [42].

The systematic review methods have certain limitations. First, we searched using a major platform (PubMed) and may have missed any studies not available through PubMed. Second, our initial screening involved title and subsequent abstract screening. As such, our screening may have missed studies that reported OPC incidence data but whose titles were noninformative. Furthermore, SEER*Stat does not report HIV serostatus, so we were unable to compare the conclusions from our meta-analysis for people living with HIV.

To our knowledge, this systematic review and meta-analysis is one of the first to comprehensively summarize OPC incidence and report sex-, age-, and race-stratified risks. Currently, there are no approved OPC screening methods available. However, given the rising incidence of OPC, it is important to understand who should be screened if studies establish that screening tests lead to early detection with improved outcomes. Synthesizing multiple data sources, we identified that men 50–79 years old and men living with HIV are at high risk of OPC. Our analysis identified possible targets for future HPV-OPC screening strategies.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12885-025-14904-4.

Supplementary Material 1

Acknowledgements

We would like to thank Darien Weatherspoon, Jaimie Shing, and other authors not referred to by name for providing additional data for our study beyond what was published in their studies, helping to inform sex-specific meta-analysis estimates. Our appreciation extends to all the authors, collaborators, and participants involved in the studies from which we obtained the data.

Author contributions

GD conceptualized the study. AB and GD obtained the data. AB performed the analysis. AB, GD and IS analyzed and interpreted the data. AB and GD drafted the manuscript. AB, GD, and IS critically reviewed the manuscript. All authors approve the final version of the manuscript.

Funding

Gypsyamber D'Souza was supported by the National Institute of Dental and Craniofacial Research (grant R35DE026631). The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Data availability

The SEER data used in this analysis was obtained from SEER and is available at https://seer.cancer.gov/seerstat/ for all users that acknowledge the agreements and limitations and are authorized by SEER to use the data. The prompt for data extraction used for the systematic review and meta-analysis is available in the supplementary material.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Received: 16 June 2025 / Accepted: 20 August 2025

Published online: 30 September 2025

References

1. Cornelius ME, Loretan CG, Wang TW, Jamal A, Homa DM. Tobacco product use among adults - United states, 2020. MMWR Morb Mortal Wkly Rep. 2022;71(11):397–405. https://doi.org/10.15585/mmwr.mm7111a1.
2. D'Souza G, Cullen K, Bowie J, Thorpe R, Fakhry C. Differences in oral sexual behaviors by gender, age, and race explain observed differences in prevalence of oral human papillomavirus infection. PLoS One. 2014;9(1):e86023. https://doi.org/10.1371/journal.pone.0086023.
3. Zumsteg ZS, Luu M, Rosenberg PS, et al. Global epidemiologic patterns of oropharyngeal cancer incidence trends. J Natl Cancer Inst. 2023;115(12):1544–54. https://doi.org/10.1093/jnci/djad169.
4. Damgacioglu H, Sonawane K, Zhu Y, et al. Oropharyngeal cancer incidence and mortality trends in all 50 States in the US, 2001–2017. JAMA Otolaryngol Head Neck Surg. 2022;148(2):155–65. https://doi.org/10.1001/jamaoto.2021.3567.
5. Surveillance Research Program, National Cancer Institute. SEER*Explorer: An interactive website for SEER cancer statistics. April 17, 2024. Accessed December 16. 2024. https://seer.cancer.gov/statistics-network/explorer/index.html
6. Katz DA, Copen CE, Haderxhanaj LT, et al. Changes in oral and anal sex with Opposite-Sex partners among sexually active females and males aged 15 to 44 years in the United States: national survey of family growth, 2011–2019. Sex Transm Dis. 2023;50(11):713–9. https://doi.org/10.1097/OLQ.0000000000001867.
7. Guo F, Chang M, Scholl M, McKinnon B, Berenson AB. Trends in oropharyngeal cancer incidence among adult men and women in the United States from 2001 to 2018. Front Oncol. 2022;12: 926555. https://doi.org/10.3389/fonc.2022.926555.
8. Wu J, Xiao F, Zheng Y, Lin Y, Wang HL. Worldwide trend in human papillomavirus-attributable cancer incidence rates between 1990 and 2012 and bayesian projection to 2030. Cancer. 2021;127(17):3172–82. https://doi.org/10.1002/cncr.33628.
9. Zhang Y, Fakhry C, D'Souza G. Projected association of human papillomavirus vaccination with oropharynx cancer incidence in the US, 2020–2045. JAMA Oncol. 2021;7(10):e212907. https://doi.org/10.1001/jamaoncol.2021.2907.
10. Timbang MR, Sim MW, Bewley AF, Farwell DG, Mantravadi A, Moore MG. HPV-related oropharyngeal cancer: a review on burden of the disease and opportunities for prevention and early detection. Hum Vaccin Immunother. 2019;15(7–8):1920–8. https://doi.org/10.1080/21645515.2019.1600985.
11. Melnikow J, Henderson JT, Burda BU, Senger CA, Durbin S, Weyrich MS. Screening for cervical cancer with high-risk human papillomavirus testing: updated evidence report and systematic review for the US preventive services task force. JAMA. 2018;320(7):687–705. https://doi.org/10.1001/jama.2018.10400.
12. Fontham ETH, Wolf AMD, Church TR, et al. Cervical cancer screening for individuals at average risk: 2020 guideline update from the American Cancer Society. CA Cancer J Clin. 2020;70(5):321–46. https://doi.org/10.3322/caac.21628.
13. Kreimer AR, Ferreiro-Iglesias A, Nygard M, et al. Timing of HPV16-E6 antibody seroconversion before OPSCC: findings from the HPVC3 consortium. Ann Oncol. 2019;30(8):1335–43. https://doi.org/10.1093/annonc/mdz138.
14. D'Souza G, Clemens G, Strickler HD, et al. Long-term persistence of oral HPV over 7 years of follow-up. JNCI Cancer Spectr. 2020;4(5):pkaa047. https://doi.org/10.1093/jncics/pkaa047.
15. Day AT, Fakhry C, Tiro JA, Dahlstrom KR, Sturgis EM. Considerations in human Papillomavirus-associated oropharyngeal cancer screening: a review. JAMA Otolaryngol Head Neck Surg. 2020;146(7):656–64. https://doi.org/10.1001/jamaoto.2019.4811.
16. You EL, Henry M, Zeitouni AG. Human papillomavirus-associated oropharyngeal cancer: review of current evidence and management. Curr Oncol. 2019;26(2):119–23. https://doi.org/10.3747/co.26.4819.
17. Clifford GM, Georges D, Shiels MS, et al. A meta-analysis of anal cancer incidence by risk group: toward a unified anal cancer risk scale. Int J Cancer. 2021;148(1):38–47. https://doi.org/10.1002/ijc.33185.
18. Stier EA, Clarke MA, Deshmukh AA, et al. International anal neoplasia society's consensus guidelines for anal cancer screening. Int J Cancer. 2024;154(10):1694–702. https://doi.org/10.1002/ijc.34850.
19. Palefsky JM, Lee JY, Jay N, et al. Treatment of anal high-grade squamous intraepithelial lesions to prevent anal cancer. N Engl J Med. 2022;386(24):2273–82. https://doi.org/10.1056/NEJMoa2201048.
20. Watson M, Saraiya M, Ahmed F, et al. Using population-based cancer registry data to assess the burden of human papillomavirus-associated cancers in the United States: overview of methods. Cancer. 2008;113(S10):2841–54. https://doi.org/10.1002/cncr.23758.
21. Migliavaca CB, Stein C, Colpani V, Munn Z, Falavigna M. Quality assessment of prevalence studies: a systematic review. J Clin Epidemiol. 2020;127:59–68. https://doi.org/10.1016/j.jclinepi.2020.06.039.

22. Viechtbauer W. Conducting meta-analyses in R with the metafor package. J Stat Softw. 2010;36:1–48. https://doi.org/10.18637/jss.v036.i03.
23. Jackson D, Law M, Rücker G, Schwarzer G. The hartung-knapp modification for random-effects meta-analysis: a useful refinement but are there any residual concerns? Stat Med. 2017;36(25):3923–34. https://doi.org/10.1002/sim.7411.
24. Weatherspoon DJ, Chattopadhyay A, Boroumand S, Garcia I. Oral cavity and oropharyngeal cancer incidence trends and disparities in the United States: 2000–2010. Cancer Epidemiol. 2015;39(4):497–504. https://doi.org/10.1016/j.canep.2015.04.007.
25. Mazul AL, Chidambaram S, Zevallos JP, Massa ST. Disparities in head and neck cancer incidence and trends by race/ethnicity and sex. Head Neck. 2023;45(1):75–84. https://doi.org/10.1002/hed.27209.
26. Zevallos JP, Kramer JR, Sandulache VC, et al. National trends in oropharyngeal cancer incidence and survival within the veterans affairs health care system. Head Neck. 2021;43(1):108–15. https://doi.org/10.1002/hed.26465.
27. Michaud JM, Zhang T, Shireman TI, Lee Y, Wilson IB. Hazard of cervical, oropharyngeal, and anal cancers in HIV-infected and HIV-uninfected medicaid beneficiaries. Cancer Epidemiol Biomarkers Prev. 2020;29(7):1447–57. https://doi.org/10.1158/1055-9965.EPI-20-0281.
28. Mazul AL, Hartman CM, Mowery YM, et al. Risk and incidence of head and neck cancers in veterans living with HIV and matched HIV-negative veterans. Cancer. 2022;128(18):3310–8. https://doi.org/10.1002/cncr.34387.
29. Chaturvedi AK, D'Souza G, Gillison ML, Katki HA. Burden of HPV-positive oropharynx cancers among ever and never smokers in the U.S. population. Oral Oncol. 2016;60:61–7. https://doi.org/10.1016/j.oraloncology.2016.06.006.
30. Patel P, Hanson DL, Sullivan PS, et al. Incidence of types of cancer among HIV-infected persons compared with the general population in the United States, 1992–2003. Ann Intern Med. 2008;148(10):728–36. https://doi.org/10.7326/0003-4819-148-10-200805200-00005.
31. Golas SM. Trends in palatine tonsillar cancer incidence and mortality rates in the united States. Community Dent Oral Epidemiol. 2007;35(2):98–108. https://doi.org/10.1111/j.1600-0528.2007.00299.x.
32. Colevas AD. Population-based evaluation of incidence trends in oropharyngeal cancer focusing on socioeconomic status, sex, and race/ethnicity. Head Neck. 2014;36(1):34–42. https://doi.org/10.1002/hed.23253.
33. Zahnd WE, Rodriguez C, Jenkins WD. Rural-urban differences in human Papillomavirus-associated cancer trends and rates. J Rural Health. 2019;35(2):208–15. https://doi.org/10.1111/jrh.12305.
34. Siegel RL, Miller KD, Jemal A. Cancer statistics, 2019. CA Cancer J Clin. 2019;69(1):7–34. https://doi.org/10.3322/caac.21551.
35. Fan KM, Sprague S, Zhang P, Ariyawardana A, Johnson NW. Rates of oropharyngeal cancer continue to rise steeply amongst Australian men. Oral Dis. 2023;29(5):1959–66. https://doi.org/10.1111/odi.14202.
36. Chancellor JA, Ioannides SJ, Elwood JM. Oral and oropharyngeal cancer and the role of sexual behaviour: a systematic review. Community Dent Oral Epidemiol. 2017;45(1):20–34. https://doi.org/10.1111/cdoe.12255.
37. D'Souza G, Agrawal Y, Halpern J, Bodison S, Gillison ML. Oral sexual behaviors associated with prevalent oral human papillomavirus infection. J Infect Dis. 2009;199(9):1263–9. https://doi.org/10.1086/597755.
38. Windon MJ, Waterboer T, Hillel AT, et al. Sex differences in HPV immunity among adults without cancer. Hum Vaccin Immunother. 2019;15(7–8):1935–41. https://doi.org/10.1080/21645515.2019.1568157.
39. US Preventive Services Task Force. Screening for colorectal cancer: US preventive services task force recommendation statement. JAMA. 2021;325(19):1965–77. https://doi.org/10.1001/jama.2021.6238.
40. Fakhry C, Krapcho M, Eisele DW, D'Souza G. Head and neck squamous cell cancers in the united States are rare and the risk now is higher among white individuals compared with black individuals. Cancer. 2018;124(10):2125–33. https://doi.org/10.1002/cncr.31322.
41. D'Souza G, Westra WH, Wang SJ, et al. Differences in the prevalence of human papillomavirus (HPV) in head and neck squamous cell cancers by sex, race, anatomic tumor site, and HPV detection method. JAMA Oncol. 2017;3(2):169–77. https://doi.org/10.1001/jamaoncol.2016.3067.
42. Scott-Wittenborn N, D'Souza G, Tewari S, et al. Prevalence of human papillomavirus in head and neck cancers at tertiary care centers in the united States over time. Cancer. 2022;128(9):1767–74. https://doi.org/10.1002/cncr.34124.
43. Shiels MS, Engels EA. Evolving epidemiology of HIV-associated malignancies. Curr Opin HIV AIDS. 2017;12(1):6–11. https://doi.org/10.1097/COH.0000000000000327.
44. Amini AP, Brookes TS, Shah H, et al. The association between penile cancer and HIV infection: a literature review. Int J STD AIDS. 2023;34(4):214–28. https://doi.org/10.1177/09564624221148622.
45. Butt JL, Jacobsen JS, Simonds HM, Botha MH. The impact of concurrent HIV-infection on women with vulvar cancer: comparison of clinical characteristics and outcome. Int J STD AIDS. 2024; 9564624241287260. https://doi.org/10.1177/09564624241287260.
46. Stelzle D, Tanaka LF, Lee KK, et al. Estimates of the global burden of cervical cancer associated with HIV. Lancet Glob Health. 2021;9(2):e161–9. https://doi.org/10.1016/S2214-109X(20)30459-9.
47. Center for Diseases Control (CDC). Sexually Transmitted Infections Treatment Guidelines. February 27, 2023. Accessed March 11. 2025. https://www.cdc.gov/std/treatment-guidelines/hpv-cancer.htm

Publisher's note