

NATIONAL HUMAN PAPILLOMAVIRUS (HPV)

SURVEILLANCE & MONITORING REPORT 2025

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ISSN: 3083-3221 (Online)

This publication and associated data are available at
<https://www.kirby.unsw.edu.au/research/reports/national-human-papillomavirus-hpv-surveillance-and-monitoring-report-2025> and www.cdc.gov.au

Suggested citation:

Machalek DA, Dyer CEF, Boyd L, Smith MA, Whop LJ, Mitchell LE, Hendry A, Hossain FB, Balgovind P, Aung HL, Asselin J, Brotherton JM. (2026). *National Human Papillomavirus (HPV) Surveillance and Monitoring Report 2025*. Kirby Institute, UNSW, Sydney. Published April 7, 2026.

Publication design by Design Design.

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NATIONAL HUMAN PAPILLOMAVIRUS (HPV) SURVEILLANCE & MONITORING REPORT

2025

Co-led by The Kirby Institute and The University of Melbourne in collaboration with the NHMRC Centre for Research Excellence in Cervical Cancer Control (C4 CRE), National Centre for Immunisation Research and Surveillance, and Burnet Institute.

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Funding:

Australian Centre for Disease Control: HPV Surveillance and Monitoring Plan Reporting Project (Health/E23-328293).

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ABBREVIATIONS

ABS	Australian Bureau of Statistics
ACCESS	The Australian Collaboration for Coordinated Enhanced Sentinel Surveillance of Sexually Transmissible Infections and Blood-Borne Viruses
AoRRP	Adult-onset recurrent respiratory papillomatosis
AIHW	Australian Institute of Health and Welfare
AIR	Australian Immunisation Register
AIS	Adenocarcinoma in situ
APSU	Australian Paediatric Surveillance Unit
ASR	Age-standardised rate
ASGS	Australian Statistical Geography Standard
ATAGI	Australian Technical Advisory Group on Immunisation
CINI/CINII/CINIII	Cervical intraepithelial neoplasia (Grade I/II/III)
CIN NOS	Cervical intraepithelial neoplasia, not otherwise specified
GBMSM	Gay, bisexual and other men who have sex with men
HGA	High-grade cervical abnormality
HPV	Human papillomavirus
ICD	International Classification of Diseases
IRSD	Index of Relative Socio-Economic Disadvantage
JoRRP	Juvenile-onset recurrent respiratory papillomatosis
NCIRS	National Centre for Immunisation Research and Surveillance
NCSP	National Cervical Screening Program
NCSR	National Cancer Screening Register
NHMD	National Hospital Morbidity Database
PLIDA	Person level integrated data asset
SCC	Squamous cell carcinoma
SEIFA	Socio-Economic Indexes for Areas
SES	Socio-economic status
WHO	World Health Organization

Standard abbreviations for Australia's eight States and Territories: NSW, Vic, Qld, NT, SA, WA, Tas, ACT

ABOUT THIS REPORT

This is the National Human Papillomavirus (HPV) Surveillance and Monitoring Report. It evaluates progress against the objectives and indicators outlined in the HPV Surveillance and Monitoring Plan 2023 (The Plan) and systematically monitors the implementation and performance of the National HPV Vaccination Program.

The report's main goal is to monitor and assess HPV vaccination coverage and trends in HPV-related diseases among females and males. It (the report) evaluates the the National HPV vaccination program's equity and impact across all population groups, including Aboriginal and Torres Strait Islander communities. The aim is to identify gaps and challenges to ensure the program provides equitable benefits for HPV prevention nationwide.

These objectives and indicators are designed to complement—and, where relevant, align with—existing national strategies and monitoring efforts related to HPV.

These include:

- Annual Cervical Cancer Elimination Progress Reports
- Annual Immunisation Coverage Reports
- AIHW National Cervical Screening Program Monitoring Reports
- National Blood-borne Viruses and Sexually Transmissible Infections Annual Surveillance Reports
- National Immunisation Strategy for Australia
- National Preventive Health Strategy 2021–2030
- Australian Cancer Plan
- National Aboriginal and Torres Strait Islander Health Plan
- National Blood-borne Viruses and Sexually Transmissible Infections Strategies and associated Monitoring Plan
- National Agreement on Closing the Gap

The report is organised around the key surveillance objectives defined in The Plan. Each monitoring objective is introduced with a rationale that explains its significance in assessing the impact of the HPV vaccination program. Within each objective, the report presents progress through the specific indicators outlined in the Plan, providing status updates and a summary figure that illustrates key trends. As this is the first report, trends are presented in the context of broader historical patterns, where available, since the introduction of the National HPV Vaccination Program in 2007. Visuals are complemented by detailed tables and explanatory text, offering further insights into the data. Each section concludes with a discussion of the identified gaps, limitations, and considerations for

interpreting the data. A separate methodology section outlines the data sources, indicator definitions, and analytical methods employed in the report.

This first report establishes a foundation for ongoing, annual monitoring of the National HPV Vaccination Program. It supports consistent reporting, coordinated action, and evidence-informed policy and program development to strengthen Australia's response to HPV and accelerate progress toward the elimination of HPV-related diseases.

Terminology

This report uses the term 'participants' when referring to data collected under the National Cervical Screening Program.

When data are reported using the categories 'female' or 'male' (e.g., Australian Bureau of Statistics [ABS] estimated resident populations, cancer registries), we retain these terms in tables, figures, and when explicitly referring to source data that include children or adolescents, not only adults.

We acknowledge that some people with a cervix or a penis may not identify with these terms.

The term Indigenous is used when referring to data collected to ascertain a person's Indigenous status; otherwise, we use Aboriginal and Torres Strait Islander peoples.

II. NATIONAL HPV VACCINATION PROGRAM: MILESTONES AND STATUS OF ELIGIBLE COHORTS

In 2007, Australia became the first country to introduce a national, publicly funded, school-based HPV vaccination program through the National Immunisation Program, offering three doses of the quadrivalent vaccine to girls aged 12–13 years. Between 2007 and 2009, the program expanded to include females aged 12–26 through both school and community-based initiatives. The routine program has since continued annually for 12–13-year-old girls. In 2013, the program was extended to include boys, with a two-year catch-up program offered to those aged 14–15, making Australia the first country to vaccinate all children in the target age group. In mid-2017, in line with other vaccines in the National Immunisation Program, funded catch-up vaccine eligibility was extended to individuals up to 19 years old. In 2018, the schedule was revised to two doses, administered six months apart, in line with World Health Organization (WHO) recommendations. The quadrivalent vaccine was also replaced with the

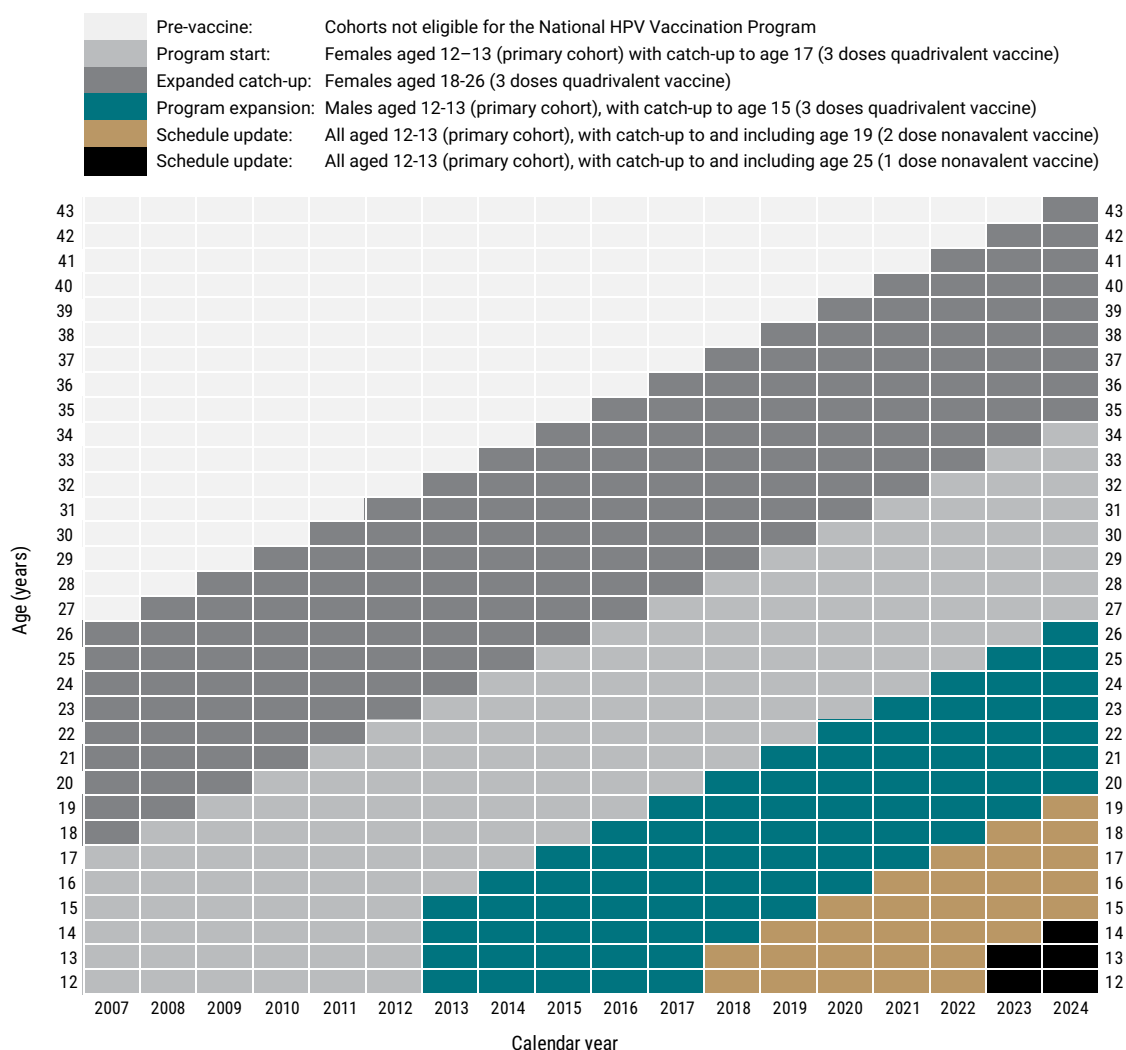
nonavalent vaccine. Most recently, in 2023, the program switched to a single-dose schedule and expanded free vaccination access to everyone up to and including age 25 who was unvaccinated.

The quadrivalent and nonavalent vaccines protect against oncogenic HPV types 16 and 18, which cause 70% of cervical and most non-cervical HPV-related cancers. The nonavalent vaccine targets five additional oncogenic types, accounting for 20% of cervical cancers. The vaccines also protect against HPV 6 and 11, which cause most genital warts.

By the end of 2024, the first cohorts vaccinated through the school-based program (at age 12–13 years) with the three-dose schedule

will have reached age 30 for females and age 24 for males. Those eligible for time-limited catch-up initiatives would have reached age 43 and 26, respectively (see Figure).

Implementation timelines for the National HPV Vaccination Program



Program monitoring

In 2024, national HPV vaccination coverage by age 15 was 79.5% (81.1% among females and 77.9% among males), down from a peak of 85.7% in 2020 (86.6% among females and 84.9% among males) since the program began in 2007. Coverage varied across states and territories and remained lower for Aboriginal and Torres Strait Islander adolescents and those in remote areas, with inequities widening since 2020. Post-COVID declines are likely driven by school absenteeism, challenges in program delivery, staffing constraints, and potential increases in vaccine hesitancy. Tailored strategies to reach adolescents outside of school, increased awareness of catch-up programs, and enhanced demographic reporting – including culturally and linguistically diverse populations and individuals with disabilities – are critical to addressing these gaps.

Nationally, 78.1% of the population aged 25–74 years eligible for the National Cervical Screening Program had at least one HPV test within the 5.5-year period to 30 June 2025, meeting the national 2030 target. The proportion exceeded 70% in all age groups except those aged 70–74 years, where it was lowest (64.3%), and peaked among middle-aged adults, reaching approximately 81–82% in those aged 45–54 years. The proportion with at least one HPV test was lowest in the most disadvantaged areas (70.2%) and in very remote regions (70.0%), and higher in major cities (78.6%) and the least disadvantaged areas (87.2%). Overall, the proportion with a HPV test declined slightly compared with the previous period (78.8% for the period 1 January 2019 – 30 June 2024), highlighting the need to understand the drivers of this decline in screening.

Monitoring infection

HPV prevalence has declined by more than 90% among eligible populations following the implementation of the HPV vaccination program. In 2024, HPV16/18 prevalence among National Cervical Screening Program participants aged 25–74 years was very low (1.4% overall; 1.0% among those aged 25–29 years). Among samples from 16–24-year-old females attending chlamydia testing in 2021–22, prevalence was similarly low (1.2%), with fewer than 1 in 50 positive for quadrivalent vaccine types, compared with 1 in 5 before vaccination. Vaccinated females had a substantially lower prevalence (<0.5%) than same-age unvaccinated females (5.4%). Aboriginal and Torres Strait Islander screening participants in vaccine-eligible age cohorts also showed very low prevalence of HPV16/18, confirming high HPV vaccine program impact. Overall, oncogenic HPV prevalence remained higher among Aboriginal and Torres Strait Islander screening

participants than in the national population (12.1% vs 7.3%, respectively), driven mainly by non-16/18 oncogenic HPV types. This likely reflects the younger age structure of the Aboriginal and Torres Strait Islander population, historical underscreening, and higher prevalence of risk factors for persistence of HPV infection, highlighting ongoing inequities.

Among rectal samples from males aged ≥16 years undergoing testing for sexually transmitted infections in 2021–22 – a population indicative of gay, bisexual, and other men who have sex with men – anal oncogenic HPV prevalence was high (66.4%), with more than one in five carrying HPV16/18 (22.2%), one-third positive for additional nonavalent types (33.5%), and more than half carrying other oncogenic types not covered by current vaccines (51.1%). Prevalence was lowest in 16–20-year-old males (35.3%), who would have been age-eligible for HPV vaccination in early adolescence, and increased steadily with age, reaching 77.8% in men aged 60 years or older. Most males in the overall sample were unvaccinated or had unknown status (93.5%), reflecting that many were too old to be eligible for free school-based vaccination when the program commenced. Ongoing surveillance will be critical as cohorts eligible for school-based vaccination age, to monitor population-level impacts of HPV programs on anal HPV infection in this group.

Monitoring non-cancer endpoints

Genital warts diagnoses at sexual health clinics have decreased significantly since the introduction of HPV vaccination. By 2024, females aged 21–24 had the lowest diagnosis rate (0.7%), down from 13.8% in 2006, with peak diagnoses moving to older age groups (35–39 and 40 and above). Among heterosexual males aged 21–24 years, new diagnoses dropped from 17.0% to 1.2%, while among gay, bisexual, and other men who have sex with men in the same age group, rates declined from 10.0% to 1.1%. Geographic and socioeconomic disparities narrowed, with prevalence rates falling to ≤2.2% across all regions and showing minimal variation by 2024. There were also notable declines among Aboriginal and Torres Strait Islander peoples, with no new diagnoses among 21–24-year-olds in 2024, compared to 7–9% in 2006.

Notifications and hospitalisations indicate a substantial decline in juvenile-onset recurrent respiratory papillomatosis (JoRRP). Between 2011 and 2023, 22 cases were reported among children aged 0–14 years, with 17 (77%) occurring before 2018. Incidence remained low throughout the period, at ≤ 0.2 cases per 100,000 children, with most cases occurring in children of unvaccinated or overseas-born mothers; only two cases were reported among Aboriginal and Torres Strait Islander children. Declining hospitalisation episodes and Australian Paediatric Surveillance Unit notifications are consistent with the population-level impact of HPV vaccination, including the 2007–2009 catch-up program, which reduced HPV 6 and 11 infections in young females and subsequent vertical transmission to their infants.

High-grade cervical abnormalities have declined markedly in vaccine-eligible cohorts. Compared with pre-vaccine cohorts, by 2017 declines had reached 70% among participants aged <20 years, 50% among those aged 20–24 years, and 19% among those aged 25–29 years, with peak detection shifting to older age groups. The transition to HPV-based screening temporarily increased observed detection rates due to higher test sensitivity, and these rates are expected to continue fluctuating due to the extension of the recommended screening interval from 2 to 5 years. Vaccine impact remains evident, particularly in younger cohorts, and gradually extends to older cohorts. Aboriginal and Torres Strait Islander participants experienced higher high-grade detection rates, reflecting historical disparities in disease burden and underscreening. Expanded vaccine eligibility and cervical screening self-collection strategies have improved access, with provision of culturally safe care and accurate data collection remaining essential priorities.

Between 2018 and 2023, the proportion of high-grade cervical abnormality attributable to HPV16/18 declined substantially, from 43% to 23%, while lesions caused by other oncogenic types increased from 52% to 66%, and mixed infections rose from 5% to 11%. The largest reductions in HPV16/18-only lesions occurred in people under 40 years, reflecting the impact of vaccination, with non-16 and 18 types now accounting for the majority of remaining lesions in younger age groups. Ongoing monitoring will require more detailed differentiation of non-16 and 18 types, especially as nonavalent vaccine-eligible cohorts will become eligible for screening from approximately 2030.

Monitoring cancer endpoints

From 2001 to 2020, cervical cancer incidence in Australia remained relatively stable, with age-standardised rates of 7.5 per 100,000 in 2001, 7.2 in 2016, and 7.5 in 2020 (standardised to the 2001 Australian population¹). Among females aged 25–74 years, incidence ranged from 9.9 to 11.8 per 100,000, and this group accounted for most deaths, with mortality declining from 3.2 in 2001 to 2.0 per 100,000 in 2020. Following the introduction of primary HPV-based screening, incidence increased modestly from 2018, particularly among females aged ≥ 35 years, while rates among females aged ≥ 75 years declined steadily between 2001 and 2020. Females aged 25–29 years experienced a temporary plateau in incidence between 2008 and 2013, followed by a decline, and rates remained very low in those under 20 years. Squamous cell carcinoma (SCC) incidence and mortality generally declined over the entire observation period; adenocarcinoma incidence rose modestly, but mortality remained low, and adenosquamous carcinoma remained rare. These trends reflect the combined effects of vaccination and changes to the screening program, highlighting the need for continued monitoring to assess long-term impacts on cervical cancer outcomes. Among Aboriginal and Torres Strait Islander females, 2016–2020 incidence and mortality rates were higher across most cervical cancer types, reflecting historical inequities in screening.

Other HPV-related cancers show varied trends. Anal SCC incidence increased steadily in females from 0.8 to 1.7 per 100,000 between 2001 and 2020, and modestly in males from 0.7 to 0.9. Oropharyngeal SCC incidence rose markedly in males from 3.6 to 7.5 per 100,000 over the same period, particularly in those aged ≥ 50 years, while female incidence increased slightly from 1.0 to 1.5. Mortality trends generally reflected these incidence patterns, with anal SCC deaths rising in females from 0.2 to 0.4 per 100,000 and modestly in males, and oropharyngeal SCC deaths increasing in males from 1.4 to 1.8 per 100,000. In contrast, vulvar, vaginal, and penile SCC incidence remained relatively stable (2020: vulvar 2.0, vaginal 0.3, penile 0.9 per 100,000), with low and stable mortality over the period. Disparities were most pronounced among older adults, Aboriginal and Torres Strait Islander peoples, and by sex, particularly for anal and oropharyngeal cancers.

¹ Note this is consistent with AIHW cancer incidence monitoring reports but different to the method used in the annual cervical cancer elimination progress reports, which standardises according to the WHO elimination methodology, using the World 2015 female population.

Between 2018 and 2023, among cervical cancers with a routine clinical HPV test result recorded within the five years before diagnosis, HPV16/18-only cases decreased from 78% to 64%, non-16 and 18-only cases increased from 18% to 27%, and mixed infections rose from 4% to 9%. The most substantial changes occurred among people under 30, where HPV16/18-only cases dropped from 83% to 36%, non-16/18-only cases increased from 17% to 52%, and mixed infections grew from 0% to 12%. Compared with pre-vaccine estimates (2005–2015), when 77% of HPV-positive cancers were caused by HPV16/18, these trends probably reflect a substantial decline in HPV16/18-only cancers among younger individuals due to HPV vaccination. HPV typing is not routinely performed for anogenital or oropharyngeal cancers; the limited data available are based on immunohistochemistry staining for p16, a surrogate marker of oncogenic HPV infection. These data are incomplete.

Addressing health disparities among Aboriginal and Torres Strait Islander peoples

While the HPV vaccination program has significantly reduced vaccine-type HPV infections, genital warts, and JoRRP, Aboriginal and Torres Strait Islander peoples continue to bear a disproportionate burden of HPV-related disease. Among women, this is reflected in higher prevalence of non-16/18 oncogenic HPV types, increased rates of high-grade cervical abnormalities, and elevated cervical cancer incidence and mortality. Among men, there is a greater burden of other HPV-related cancers, particularly oropharyngeal cancer. Addressing these persistent health inequities requires culturally safe, community-led approaches that improve access to prevention, screening, and treatment, while also confronting the enduring impacts of colonisation, which has created structural barriers to healthcare and heightened exposure to established risk factors such as tobacco and alcohol.

Data gaps and limitations

Several challenges limit the comprehensive assessment of the HPV vaccination program's impact on all indicators outlined in the Plan:

- **Linkage of outcome data to vaccination status:** Individual vaccination status is not routinely linked to screening, high-grade lesion, or cancer data, limiting assessment of vaccine impact.
- **Screening data gaps limit equity monitoring:** Data gaps exist for priority populations, including Aboriginal and Torres Strait Islander people, LGBTQI+ communities, people with disability, and culturally and linguistically diverse groups.
- **Limited HPV genotyping for monitoring:** Routine data do not include individual HPV genotyping, limiting the ability to measure type-specific vaccine impact and monitor shifts in HPV types over time.
- **Renewal of National Cervical Screening Program (December 2017):** Transition to HPV-based screening affects longitudinal comparisons of detection rates and trends.
- **Delays in cancer incidence data:** National incidence data are delayed by four years, restricting timely trend monitoring and timely response.
- **HPV attribution to cancer:** Routine HPV testing for anogenital cancers and reporting of p16 for head and neck cancers are limited, restricting accurate HPV attribution.
- **Small populations and low counts:** Small numbers in certain age groups and priority populations, and for many cancers, restrict reliable stratified analyses and trend interpretation, with resulting cell suppression applied to protect data confidentiality.
- **Surveillance gaps:** No systematic monitoring exists for immunocompromised populations or non-cervical HPV-related infections. No validated surveillance method yet exists for adult-onset recurrent respiratory papillomatosis.

1.

PROGRAM MONITORING

1. PROGRAM MONITORING

1.1 Monitor HPV vaccination coverage

Justification for this monitoring objective

Effective monitoring of HPV vaccination coverage is crucial for evaluating program performance, identifying gaps, and ensuring equitable progress toward national and global targets. While the WHO recommends 90% coverage in female-only programs for cervical cancer elimination¹, Australia vaccinates all age-eligible adolescents and has set a 90% target by age 15 to maximise impact².

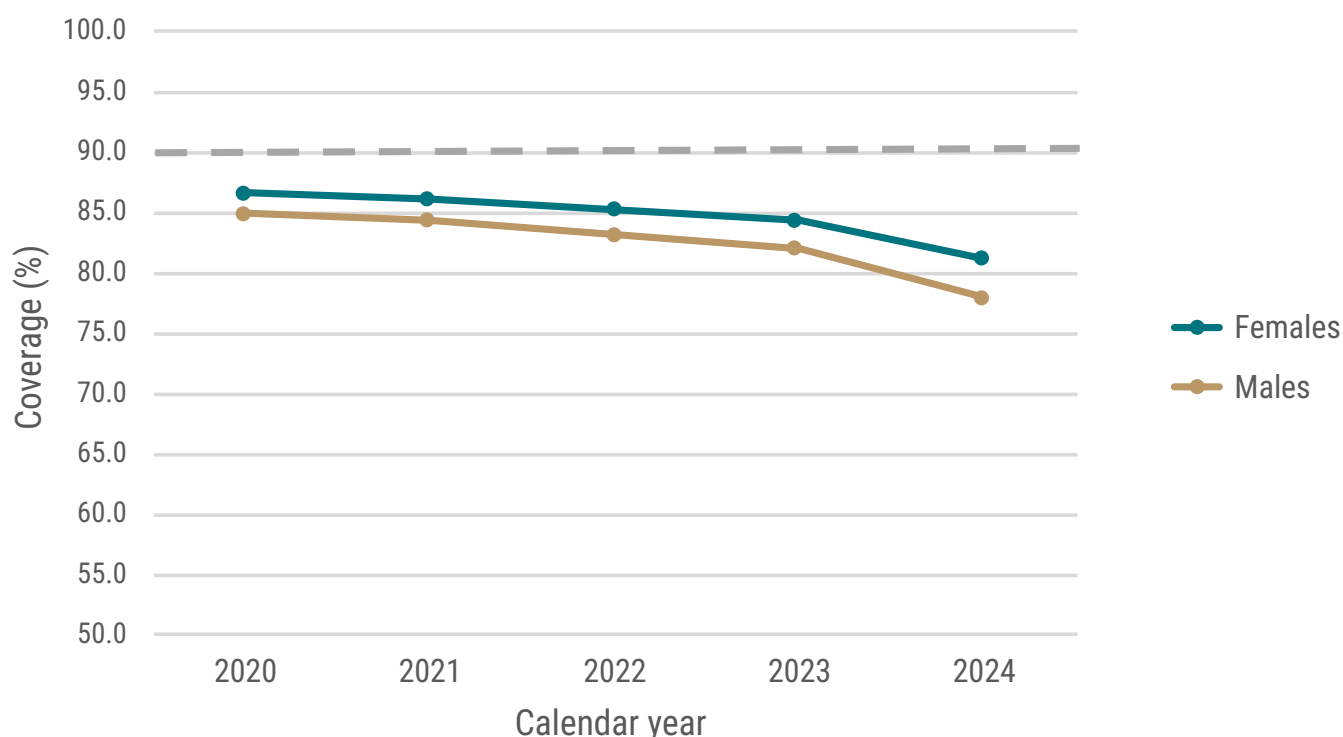
In Australia, HPV vaccination is delivered through the school-based National Immunisation Program in Year 7, typically at 12–13 years of age, as a single-dose schedule. Funded catch-up vaccination with a single dose is now available for individuals aged 25 and under. Coverage is assessed using the Australian Immunisation Register (AIR) as the proportion of individuals turning 15 each year who have received at least one dose by their 15th birthday. This standardised approach aligns with WHO recommendations, supports international comparisons, accounts for catch-up vaccinations, and enables data-driven improvements to enhance vaccine uptake.



Indicator: Single-dose HPV vaccination coverage assessed at 15 years of age

Status of indicator

Figure 1.1 National HPV vaccine coverage (%) by age 15, for females and males, 2020–2024



Source: 2025 Cervical Cancer Elimination Progress Report (Analysis of Australian Immunisation Register data, see [methodology](#)). The grey dotted line represents WHO and Australia's elimination target.

Nationally, among adolescents who turned 15 years of age in 2024, HPV vaccination coverage was 79.5%, with higher coverage in females (81.1%) than males (77.9%) (**Figure 1.1; Table 1.1**). This represents a substantial decline from the historical peak observed in the 2020 cohort, when overall coverage reached 85.7% (86.6% in females and 84.9% in males) (**Appendix Figure A1**). Notably, the 2024 cohort turned 13 years of age (when the HPV vaccine is typically given in school) in 2022, the calendar year associated with the lowest recorded school attendance³.

Coverage in 2024 varied by area-based socioeconomic status, ranging from 74.5% overall in the lowest quintile (77.1% in females, 72.0% in males) to 84.2% in the highest quintile (85.0% in females, 83.5% in males). The difference between these groups widened to 9.7

percentage points, up from 6.5 points in 2023, driven by larger declines in lower socioeconomic areas, although all quintiles experienced some decrease. The gap was more pronounced in boys (11.9 percentage points) than in girls (7.9 percentage points). By comparison, the 2020 cohort had much smaller disparities, with gaps of 2.2 points for males and 1.2 points for females (**Table 1.1**).

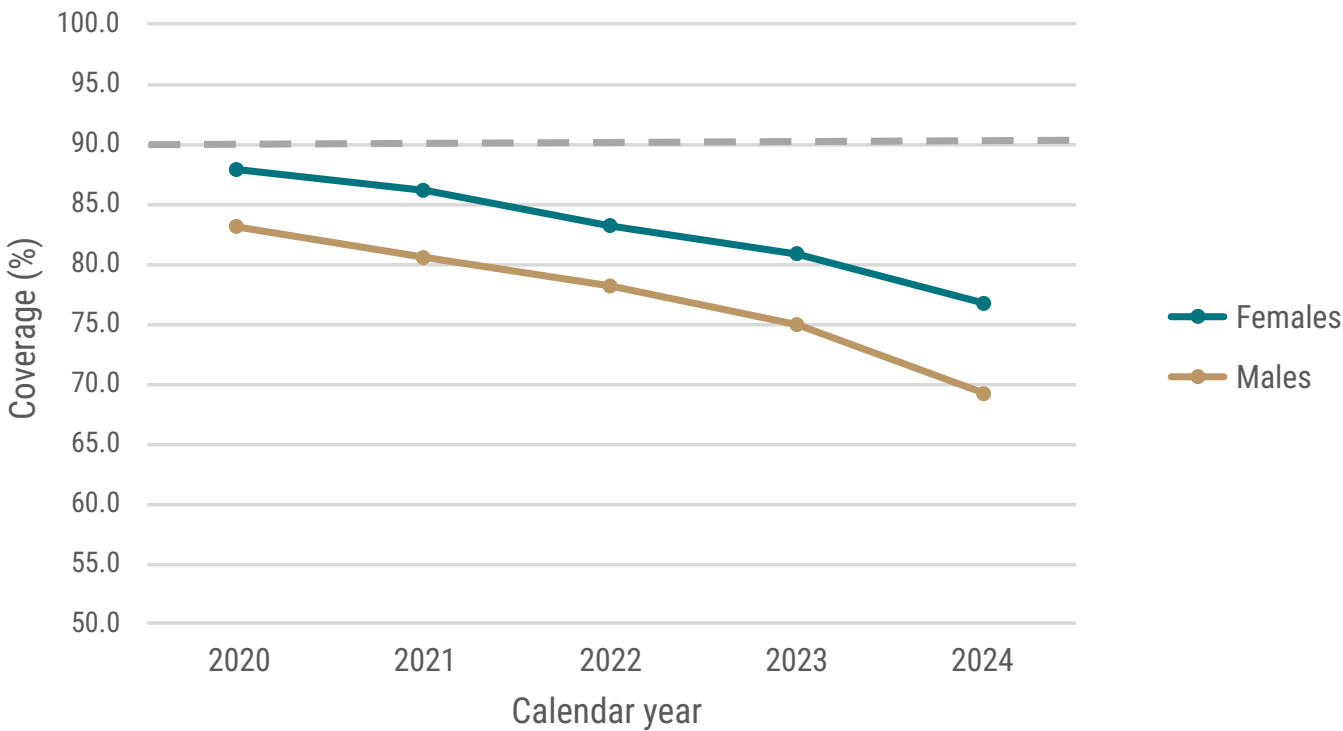
In 2024, overall HPV vaccination coverage fell below 80% across all remoteness area classifications. Coverage remained above 80% overall for NSW (80.2%), Victoria (81.3%) and the ACT (87.1%). Coverage exceeded 80% for boys only in Victoria (80.1%) and the ACT (87.0%), while coverage for girls remained above 80% in all jurisdictions except Queensland (78.2%), the NT (76.4%) and SA (79.6%) (**Table 1.1**).



Indicator: Single-dose HPV vaccination coverage for Aboriginal and Torres Strait Islander peoples assessed at 15 years of age.

Status of indicator

Figure 1.2 National HPV vaccine coverage (%) by age 15, for Aboriginal and Torres Strait Islander females and males, 2020–2024



Source: **2025 Cervical Cancer Elimination Progress Report** (Analysis of Australian Immunisation Register data, see [methodology](#)). The grey dotted line represents WHO and Australia's elimination target.

Data gaps and limitations

- Indigenous status was based on any self-reported identification recorded in the AIR.
- Available demographic variables are limited to sex, age, Indigenous status, and postcode. Postcode is used to determine jurisdiction, remoteness, and area-level socioeconomic status.
- Data on other priority populations are not available.

Data interpretation

In 2024, HPV vaccination coverage by age 15 was 79.5%, continuing a decline from a peak of 85.7% in 2020. Inequities in coverage are widening, particularly among Aboriginal and Torres Strait Islander peoples and those in remote and very remote areas, with an increasing number of subgroups now falling below 80%. Coverage also varies significantly across states and territories, likely due to differences in population demographics and differences in program delivery.

The COVID-19 pandemic had a well-documented impact on vaccine coverage globally and in Australia, particularly affecting school-based programs⁴⁻⁶. These persistent post-pandemic trends highlight ongoing challenges in re-engaging communities in the program. A contributing factor is sustained school absenteeism, as school attendance is in decline nationally³. Additional challenges include changes in program delivery, such as difficulties implementing online consent procedures, competing school priorities, staffing shortages among school personnel and immunisers, and a probable rise in vaccine hesitancy compared to pre-pandemic levels^{7,8}. Coupled with these challenges, the 2023 shift from a two-dose to a single-dose HPV vaccination schedule may also have made it more challenging to maintain multiple routine school visits, but isolating its impact on coverage is difficult given the broader post-COVID impacts⁹. Close monitoring is crucial to understanding how the single-dose schedule, which is, in theory, easier to deliver but may counterintuitively reduce in-school opportunities to do so, affects vaccine uptake within this complex context.

The growing disparities in HPV vaccination coverage across sociodemographic strata—including by remoteness, sex, jurisdiction, and among Aboriginal and Torres Strait Islander peoples—highlight the urgent need for more context-specific approaches to improve uptake. Vaccine coverage is lowest in those groups with low school attendance¹⁰. Therefore, unless attendance improves, complementary strategies to reach adolescents outside school will be necessary.

A deeper understanding of local barriers and adoption of tailored strategies that reflect the needs and preferences of diverse communities and young people will be needed. In parallel, improving awareness and accessibility of the funded HPV catch-up program to age 25 is essential to ensure those who missed vaccination opportunities, particularly due to pandemic disruptions, are not left behind.

Finally, there is a critical need to enhance the completeness and quality of HPV vaccine monitoring data to ensure the program is meeting the needs of all priority populations identified in Australia's cervical cancer elimination strategy. This includes accurate and comprehensive coverage reporting for people from culturally and linguistically diverse backgrounds, as well as those living with disabilities. Improving demographic reporting is crucial for identifying and addressing inequities in access and uptake. New opportunities through the ABS person level integrated data asset (PLIDA) and research initiatives such as INCLUDE will provide a clearer picture of coverage gaps¹¹. PLIDA links vaccination records with a wide range of national administrative health datasets and Australian Census data, enabling the identification and analysis of gaps across detailed demographic and health status characteristics. INCLUDE is a National Health and Medical Research Council-funded project that aims to improve understanding of inequalities in vaccination and screening coverage using PLIDA; however, these efforts are still underway, and detailed data are not yet available.

For additional information, refer also to the [2025 Cervical Cancer Elimination Progress Report](#) and the [NCIRS Annual Coverage Report 2024](#).

Table 1.1 HPV vaccine coverage (%) by age 15 nationally and among females and males, 2020–2024

	2020			2021			2022			2023			2024		
	Overall	Females	Males	Overall	Females	Males	Overall	Females	Males	Overall	Females	Males	Overall	Females	Males
Characteristic															
Nationally	85.7	86.6	84.9	85.3	86.2	84.4	84.2	85.3	83.1	83.0	84.2	81.8	79.5	81.1	77.9
Area-based socioeconomic status															
Group 1 (lowest)	85.4	86.1	83.1	84.1	85.7	82.5	81.9	83.5	80.3	79.7	81.8	77.6	74.5	77.1	72.0
Group 2	85.3	86.8	84.7	84.9	85.8	84.0	83.4	84.5	82.3	81.8	83.3	80.3	77.4	79.2	75.6
Group 3	85.6	86.2	84.8	85.0	86.0	84.0	83.6	84.8	82.5	82.4	83.9	81.0	78.7	80.5	77.1
Group 4	85.3	86.7	85.4	85.2	85.8	84.6	84.5	85.5	83.6	83.9	84.5	83.4	81.1	82.4	79.8
Group 5 (highest)	86.8	87.3	86.0	86.8	87.5	86.2	86.5	87.3	85.7	86.2	86.9	85.5	84.2	85.0	83.5
Remoteness area															
Major city	85.5	86.4	84.7	85.1	86.0	84.3	84.2	85.4	83.1	83.1	84.3	82.0	79.9	81.4	78.5
Inner regional	86.7	87.5	85.9	86.2	87.1	85.3	84.7	85.7	83.8	83.1	84.6	81.7	79.0	80.9	77.3
Outer regional	86.2	87.5	85.0	85.5	86.9	84.2	83.3	84.5	82.1	82.4	83.9	81.0	78.1	80.3	76.1
Remote	84.2	85.1	83.3	84.7	86.2	83.2	81.9	83.6	80.3	80.5	81.9	79.2	74.4	75.7	73.3
Very remote	84.9	87.5	82.4	82.6	84.5	80.8	80.1	80.2	80.1	76.4	78.2	74.7	73.3	77.4	69.3
State/Territory															
NSW	86.7	87.7	85.8	86.5	87.8	85.3	85.4	86.7	84.2	84.4	86.1	82.7	80.2	82.5	78.1
Vic	87.2	88.2	86.2	86.7	87.6	86.0	85.5	86.8	84.2	84.2	85.2	83.4	81.3	82.5	80.1
Qld	82.8	83.6	82.0	82.2	83.0	81.5	81.4	82.4	80.5	80.3	81.7	79.0	76.8	78.2	75.5
WA	84.6	84.9	84.3	84.2	84.3	84.2	83.8	84.5	83.1	82.2	82.4	82.0	79.6	80.4	78.9
SA	86.2	86.8	85.5	85.2	86.2	84.3	84.2	85.1	83.3	81.8	83.1	80.5	77.4	79.6	75.4
Tas	86.9	88.3	85.6	85.8	88.1	83.6	82.1	84.0	80.3	82.1	83.5	80.7	78.2	80.3	76.3
ACT	88.7	89.8	87.6	89.9	90.4	89.4	88.6	89.8	87.5	89.2	90.5	87.9	87.1	87.0	87.2
NT	85.7	87.7	83.9	83.6	86.4	80.8	79.9	81.6	78.3	75.4	78.3	72.5	71.5	76.4	67.0

Source: [2025 Cervical Cancer Elimination Progress Report](#) (Analysis of Australian Immunisation Register data, see [methodology](#)).

Table 1.2 HPV vaccine coverage (%) by age 15 for Aboriginal and Torres Strait Islander females and males, 2020–2024

	2020		2021		2022		2023		2024	
	Females	Males	Females	Males	Females	Males	Females	Males	Females	Males
Characteristic										
All Indigenous	87.8	83.0	86.1	80.6	83.0	78.1	80.9	75.0	76.7	69.2
Area-based socioeconomic status										
Group 1 (lowest)	87.6	82.0	85.4	78.3	81.7	77.1	79.9	72.7	74.7	65.4
Group 2	87.7	82.2	85.8	79.7	81.8	77.2	78.0	73.3	73.7	67.0
Group 3	88.3	84.2	87.5	82.3	83.9	79.3	83.6	77.0	78.7	72.9
Group 4	86.9	84.1	87.5	84.7	86.9	81.7	83.1	80.4	82.8	75.7
Group 5 (highest)	90.8	86.6	87.7	87.0	85.9	79.8	87.0	82.2	82.8	79.7
Remoteness area										
Major city	88.4	84.2	86.9	82.5	84.9	80.3	82.7	77.5	79.6	74.0
Inner regional	90.2	85.5	87.6	81.5	85.7	78.2	83.2	76.0	77.8	69.6
Outer regional	85.5	80.4	85.0	77.8	79.4	75.4	78.1	73.4	73.1	64.0
Remote	82.4	78.9	86.1	78.3	79.4	75.2	76.8	69.6	68.8	66.1
Very remote	88.1	80.7	83.6	78.0	78.3	77.6	75.4	69.6	73.7	63.5
State/Territory										
NSW	92.2	88.2	90.8	86.7	88.1	81.7	86.9	80.2	81.5	72.3
Vic	88.2	85.6	87.1	79.6	82.5	78.8	80.4	75.9	78.7	74.3
Qld	85.6	80.9	83.7	76.8	80.3	76.4	79.5	74.2	74.6	67.8
WA	82.4	78.7	81.0	77.6	81.4	78.6	78.5	73.4	74.8	71.9
SA	82.2	69.3	74.7	72.0	74.6	65.6	66.4	59.2	66.7	55.7
Tas	91.5	86.4	88.8	82.5	84.6	79.5	81.2	82.2	77.5	72.8
ACT	86.4	79.6	89.7	82.7	88.5	82.7	91.7	75.3	83.8	81.0
NT	89.1	82.5	87.3	79.5	78.4	75.2	72.7	65.6	72.7	59.9

Source: [2025 Cervical Cancer Elimination Progress Report](#) (Analysis of Australian Immunisation Register data, see [methodology](#)).

1.2 Monitor coverage of cervical screening

Justification for this monitoring objective

Australia’s National Cervical Screening Program (NCSP) provides an effective system for monitoring trends in HPV-related outcomes, such as HPV infections and high-grade cervical abnormalities. As more vaccine-eligible cohorts participate in screening over time, understanding cervical screening coverage among both vaccinated and unvaccinated individuals will be crucial for evaluating the impact of the HPV vaccination program on screen-detected outcomes. It is essential to monitor screening coverage (noting this measure is formally called ‘participation’ within

the NCSP) by age group and vaccination status, as well as changes in participation over time, as these variables can affect the assessment of observed trends in screen-detected endpoints.

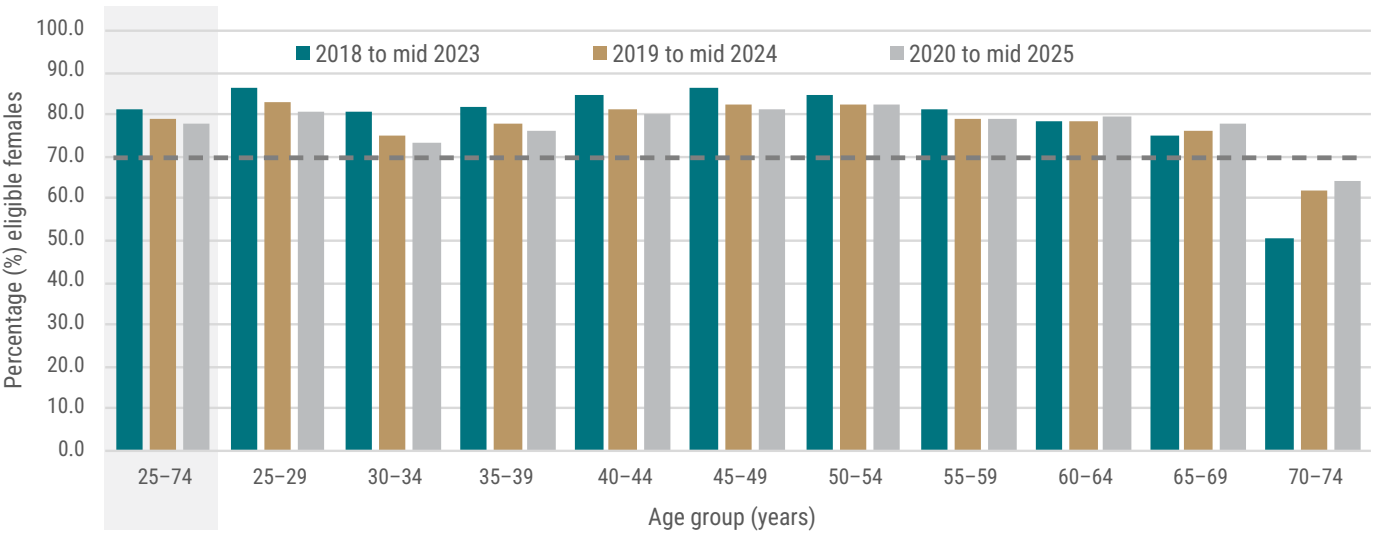
Cervical screening is also needed to expedite the elimination of cervical cancer, as HPV vaccination requires several decades to have its full effects across the population. The WHO 2030 scale-up target for all countries to be on track to achieve cervical cancer elimination is 70% of eligible people screened with a high-performance test at least twice (at ages 35 and 45)¹. Australia aims for at least 70% of eligible people (25–74 years) to participate in 5-yearly HPV screening under its NCSP².



Indicator: Number of eligible people aged 25–74 years screened in a 5-year period as a percentage of eligible people in the population

Status of indicator

Figure 1.3 Percentage (%) of the eligible population with an HPV test over three rolling 5.5-year periods* (2025), by age group at test



Source: [2025 Cervical Cancer Elimination Progress Report](#) (Analysis of National Cancer Screening Register data, see [methodology](#)). *A 5.5-year interval was used to define timely cervical screening. For the 25–74 age group, percentages were age-standardised using the Australia 2001 standard population. The grey dotted line represents Australia’s elimination target.

Nationally, over the 5.5-year period from 1 January 2020 to 30 June 2025, 78.1% of the eligible population had at least one HPV test when they were aged 25–74 years. Proportions ranged from 64.3% among those aged 70–74 years to 82.3% among those aged 50–54 years (based on age at test). The proportion with an HPV test reached 80% or more among those aged 25–29, 45–49, and 50–54 years. It exceeded 70% in all age groups except those aged 70–74 years (**Figure 1.3; Table 1.3**).

There were notable differences in the proportion with a HPV test by socioeconomic status, with the highest proportion observed in the least disadvantaged areas (87.2%) and the lowest in the most disadvantaged areas (70.2%). The proportion was highest in major cities (78.6%) and lowest in very remote areas (70.0%). Across states and territories, the proportion with a HPV test ranged from 76.9% in NSW to 80.5% in the ACT. The proportion declined slightly nationally and across most age, socioeconomic, and geographic groups during the three successive 5.5-year periods available for analysis, except among those with a test aged 55–74 years, in very remote areas, and in Tasmania and the NT (**Table 1.3**).

Table 1.3 Percentage (%) of the eligible population with an HPV test over three rolling 5.5-year periods* (2018–2025), by available demographic characteristics

	2018 to mid 2023	2019 to mid 2024	2020 to mid 2025
Characteristic			
Nationally	81.1	78.8	78.1
Age group			
25–29	86.5	83.2	80.5
30–34	80.5	75.3	73.2
35–39	82.1	77.9	76.4
40–44	84.6	81.0	79.9
45–49	86.5	82.6	81.4
50–54	84.7	82.4	82.3
55–59	81.3	78.9	79.1
60–64	78.7	78.5	79.7
65–69	74.8	76.4	78.1
70–74	50.4	62.0	64.3
Area-based socioeconomic status			
Group 1 (lowest)	73.0	70.8	70.2
Group 2	76.8	74.6	74.2
Group 3	80.4	78.3	77.6
Group 4	83.6	81.5	80.6
Group 5 (highest)	90.7	88.3	87.2
Remoteness area			
Major city	81.8	79.5	78.6
Inner regional	80.3	78.4	78.3
Outer regional	78.1	76.1	75.6
Remote	76.6	74.5	74.1
Very remote	70.3	69.7	70.0
State/Territory			
NSW	80.2	77.7	76.9
Vic	81.7	79.8	79.3
Qld	81.4	79.1	78.2
WA	80.9	78.6	78.1
SA	81.8	79.2	78.0
Tas	80.1	78.2	78.3
ACT	84.2	82.0	80.5
NT	80.4	78.4	78.5

Source: 2025 Cervical Cancer Elimination Progress Report (Analysis of National Cancer Screening Register data, see [methodology](#)). *A 5.5-year interval was used to define timely cervical screening. Percentages (except those presented by age group) were age-standardised using the Australia 2001 standard population.



Indicator: Number of eligible Aboriginal and Torres Strait Islander peoples aged 25–74 years screened in a 5-year period as a percentage of eligible people in the Aboriginal and Torres Strait Islander population

Status of indicator

Data to inform this indicator was not available.

Data gaps and limitations

- Data on Indigenous status in the National Cancer Screening Register (NCSR) are incomplete, preventing accurate reporting for Aboriginal and Torres Strait Islander peoples.
- The NCSR is not linked to the AIR, preventing the reporting of screening coverage by vaccination status.
- This measure provides a retrospective assessment over a 5.5-year period in terms of people who were screened and the age they were (and where they lived) at the time of their test. It therefore provides information about engagement with screening in the past but cannot be used directly to quantify how many people were overdue at the end of each time period, their current age, or where they now live.

Data interpretation

This measure provides a retrospective measure of engagement with HPV screening over a 5.5-year period. Based on this measure, nationally, Australia has met the national 2030 HPV screening elimination coverage target of 70% after the first five-year interval of the HPV-based program. Overall, participation remains high but has declined slightly in most age, socioeconomic, and geographic groups compared with the previous period, with notable increases only among older age groups (60–74 years) and in very remote areas. These trends reflect both longer-term patterns of screening and emerging challenges in access, highlighting where continued efforts may be needed to sustain high coverage.

Another important measure is whether women are up to date with screening at a given point in time. Trends reported in the [2025 Cervical Cancer Elimination Progress Report](#) show that, nationally, 74.2% of the eligible population aged 25–74 years were up to date at the end of 2024 (defined as having had an HPV test in the preceding 5.5 years). The proportion who were up to date was lowest among younger women (50% for those aged 25–29 and 68% for those aged 30–34). The differences between these complementary measures of the proportion who are up to date with screening and the proportion with an HPV test in a fixed 5.5-year period suggests that while many people

attend for screening at some point between the ages of 25 and 29 years, in many cases this does not appear to occur at the recommended start age of 25 (noting that an invitation to attend for screening is sent 3 months prior to people turning 25).

These patterns highlight the need for targeted outreach to younger populations to ensure timely initiation of cervical screening and thereby early detection of high-grade disease. Attendance for a first screen also provides a key opportunity to vaccinate those who remain unvaccinated (as catch-up vaccination is available up until just before an individual turns 26). Any attendance at primary care at the age of 25, therefore, provides the opportunity to offer and promote two important HPV prevention opportunities, HPV vaccination and cervical screening, supporting continued progress toward cervical cancer elimination.

Published studies, including from Australia before the switch to HPV-based screening indicate that people vaccinated against HPV are more likely to participate in cervical screening than those who are unvaccinated, although these data mostly represent cohorts offered catch-up vaccination at older ages^{6,12,13}. However, as more recent linked data is unavailable, it remains unclear whether this trend has persisted. Lower screening participation among younger populations, particularly those that remain unvaccinated, may delay detection of high-grade disease, leading to an underestimation of lesion prevalence. This delay complicates efforts to assess the actual population impact of the HPV vaccine on high-grade disease accurately. It also highlights the need for increased screening initiation among younger people to enable early detection of high-grade disease before it has time to progress to cervical cancer.

For additional information, also refer to the [2025 Cervical Cancer Elimination Progress Report](#) and the [Australian Institute of Health and Welfare \(AIHW\) National Cervical Screening Program monitoring report 2025](#).

2.

MONITORING INFECTION

2.1 Monitor the prevalence of HPV genotypes

Justification for this monitoring objective

Monitoring HPV prevalence is a critical early surveillance measure because persistent infection with certain HPV types is a necessary cause of cervical and other HPV-related cancers. The vaccine prevents infection with these specific types, so observed reductions in their circulation in vaccine-eligible populations provide a valuable indicator of vaccine impact, both through individual protection in vaccinated populations and herd effects among the unvaccinated¹⁴⁻¹⁶. In Australia, surveillance research has demonstrated greater than 90% reductions in vaccine-targeted HPV types among young vaccine-eligible people attending routine cervical screening, including comparable declines among young Aboriginal and Torres Strait Islander women^{17,18}. Reductions in HPV prevalence have also been documented in heterosexual men, likely reflecting indirect protection by the decrease in circulating HPV types^{19,20}. Following the inclusion of boys in the vaccination program, declines in anal HPV infections were observed in young men who have sex with men^{21,22}.

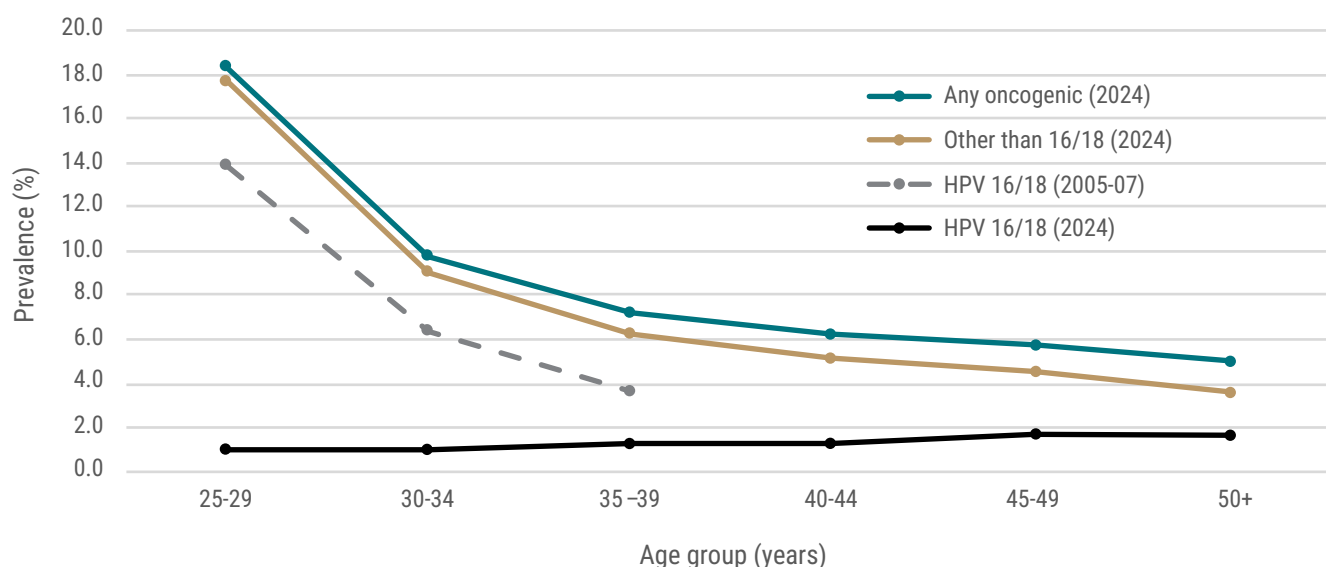
Australia's transition to primary HPV-based screening enables routine, funded monitoring of HPV prevalence in screened women, supporting long-term tracking of vaccine impact, community transmission, and indirect protection among heterosexual males. Dedicated HPV prevalence surveys using residual samples submitted for chlamydia testing have proven feasible for monitoring vaccine program effects in younger women not yet eligible for screening²³. Similarly, male rectal swabs submitted for testing for sexually transmitted infections are a validated proxy for male-to-male sexual behaviour and provide valuable data on HPV trends among gay, bisexual and other men who have sex with men (GBMSM)²⁴. Surveillance of these additional groups provides comprehensive insights into the impact of HPV vaccination. Continued monitoring will be essential to evaluate the early effects of key program changes, including the shift to the nonavalent vaccine and introduction of the single-dose schedule. Ongoing monitoring also supports detection of cross-protection and potential type-replacement, though HPV is a highly stable virus, and to date, there has been no evidence of type replacement in Australia or globally²⁵.



Indicator: Prevalence of HPV infection among National Cervical Screening Program participants

Status of indicator

Figure 2.1 Age-specific oncogenic HPV prevalence (%) among National Cervical Screening Program (NCSP) participants aged 25–74 in 2024



Source: [2025 Cervical Cancer Elimination Progress Report](#) (Analysis of National Cancer Screening Register data, see [methodology](#)) and the Women's HPV Indigenous Non-Indigenous Urban Rural Study (WHINURS) study for historical estimates of HPV types 16 and/or 18 (HPV16/18) among women attending screening in 2005–07²⁶. Estimates of prevalence in 2024: HPV16/18; prevalence of HPV types other than 16 and 18, including detection of one of the 12 other oncogenic HPV types (Other oncogenic); and any oncogenic HPV type (Any oncogenic).

2. MONITORING INFECTION

In 2024, the prevalence of any oncogenic HPV type among screening participants was 7.3%, including 1.4% with HPV16/18 and 6.2% with other oncogenic types. HPV16/18 prevalence was lowest among 25–29-year-olds (1.0%) and remained low (<2.0%) across all age groups (25–74 years). This contrasts with much higher rates observed before the introduction of the HPV vaccination program—specifically, a 2005–2007 surveillance study reported HPV16/18 prevalence of 13.9%, 6.4%, and 3.7% among women aged 25–29, 30–34, and 35–39 years, respectively, attending cervical screening²⁶ (**Figure 2.1; Table 2.1**).

The prevalence of oncogenic types other than 16/18 was highest among individuals aged 25–29 years and declined with increasing age. Patterns in 2024 were generally consistent across socioeconomic quintiles, remoteness areas, and states and territories. However, the prevalence of non-16/18 oncogenic types was higher among participants from very remote areas and the NT (**Table 2.1**).

Between 2018 and 2024, the prevalence of HPV16/18 declined from 2.1% to 1.4%, with reductions observed across all age groups, regions, and socioeconomic strata. Prevalence remained low and relatively stable, with a modest increase between 2020 and 2021, followed by a decline from 2022 onward. The overall prevalence of any oncogenic HPV type increased from 8.8% in 2018 to a peak of 11.0% in 2021, then declined to 7.0% in 2023 and increased slightly in 2024 (7.3%). This pattern was primarily driven by changes in the prevalence of oncogenic types other than HPV16/18, which remained relatively stable across most population groups over time (**Table 2.1**).

Table 2.1 Oncogenic HPV prevalence (%) among National Cervical Screening Program (NCSP) participants aged 25–74 years by available demographics, 2018–2024

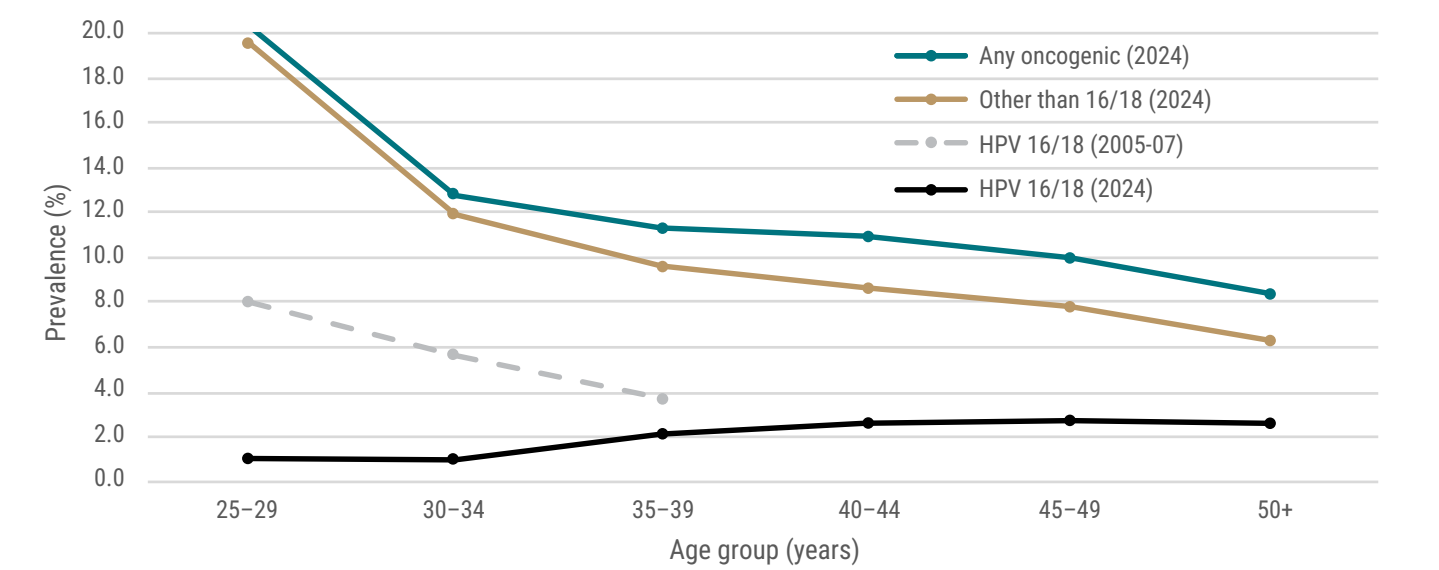
	2018			2019			2020			2021			2022			2023			2024		
Characteristic	16/18	Other	Any	16/18	Other	Any	16/18	Other	Any	16/18	Other	Any	16/18	Other	Any	16/18	Other	Any	16/18	Other	Any
Nationally	2.1	6.8	8.8	2.0	6.6	8.5	2.3	8.6	10.5	2.3	9.1	11.0	1.9	8.8	10.3	1.4	5.9	7.0	1.4	6.2	7.3
Area-based socioeconomic status																					
Group 1 (lowest)	2.2	6.8	8.9	2.1	6.7	8.7	2.4	8.4	10.4	2.4	8.7	10.7	2.0	8.6	10.3	1.6	6.2	7.5	1.7	6.5	7.9
Group 2	2.2	6.8	8.9	2.1	6.6	8.5	2.4	8.3	10.4	2.4	9.1	11.1	2.0	8.9	10.6	1.5	6.0	7.3	1.5	6.3	7.6
Group 3	2.1	6.6	8.5	1.9	6.3	8.0	2.3	8.2	10.1	2.2	8.6	10.5	1.9	8.2	9.8	1.4	5.6	6.7	1.4	6.0	7.1
Group 4	2.1	6.9	8.9	2.0	6.7	8.6	2.3	8.7	10.6	2.2	9.3	11.1	1.8	8.8	10.3	1.3	5.9	7.0	1.3	6.2	7.2
Group 5 (highest)	2.0	6.9	8.8	1.9	6.8	8.5	2.2	9.1	10.9	2.2	9.8	11.6	1.7	9.1	10.5	1.2	5.7	6.7	1.2	6.0	6.9
Remoteness area																					
Major city	2.1	7.0	8.9	1.9	6.8	8.6	2.3	8.8	10.7	2.2	9.3	11.1	1.8	8.8	10.3	1.3	5.9	7.0	1.3	6.2	7.3
Inner regional	2.2	6.1	8.1	2.1	6.0	7.9	2.4	7.8	9.9	2.4	8.5	10.5	2.0	8.1	9.8	1.5	5.5	6.7	1.6	5.8	7.1
Outer regional	2.2	6.5	8.6	2.0	6.2	8.1	2.4	7.9	10.0	2.3	8.7	10.7	2.0	8.9	10.7	1.6	5.8	7.2	1.7	6.0	7.4
Remote	2.4	7.1	9.4	2.3	6.7	8.9	2.1	8.7	10.5	2.4	9.5	11.7	2.2	9.6	11.5	1.6	7.1	8.6	1.6	7.3	8.5
Very remote	2.6	8.8	11.1	2.2	8.1	10.2	2.2	9.6	11.6	2.0	10.5	12.2	1.9	11.0	12.6	1.9	8.5	10.2	1.9	9.1	10.8
State/Territory																					
NSW	2.1	6.3	8.4	2.0	6.3	8.3	2.3	8.2	10.1	2.2	9.1	10.9	1.8	8.4	9.9	1.3	5.7	6.8	1.4	5.9	7.0
Vic	1.9	7.5	9.1	1.9	7.0	8.6	2.6	9.2	11.2	2.5	9.3	11.3	2.0	8.7	10.3	1.3	6.0	7.1	1.4	6.5	7.5
Qld	2.5	6.7	9.1	2.2	6.7	8.8	2.4	8.7	10.9	2.3	9.1	11.1	2.1	9.2	11.0	1.5	6.0	7.3	1.5	6.5	7.8
WA	1.8	7.0	8.8	1.7	6.7	8.3	1.8	8.5	10.1	1.9	9.3	10.9	1.6	9.2	10.5	1.3	5.8	7.0	1.4	6.0	7.2
SA	2.2	6.3	8.3	2.0	6.2	7.9	2.1	7.8	9.6	2.1	8.8	10.4	1.8	8.3	9.8	1.4	5.1	6.3	1.5	5.2	6.4
Tas	1.6	6.6	8.1	1.5	6.4	7.9	1.6	8.2	9.3	1.8	9.3	10.7	1.5	9.2	10.3	1.5	6.0	7.1	1.6	6.0	7.4
ACT	1.6	6.3	7.9	1.3	6.4	7.6	1.5	7.8	9.1	1.4	8.3	9.5	1.2	7.4	8.4	1.1	5.2	6.1	1.0	5.6	6.4
NT	2.7	9.3	12.0	2.6	9.1	11.5	2.7	10.4	12.9	2.4	11.0	13.1	2.2	11.2	13.2	2.0	8.5	10.4	1.7	9.0	10.4
Age group																					
25–29	2.0	17.8	19.6	1.7	17.5	18.9	1.7	18.0	19.3	1.4	18.2	19.2	1.1	17.8	18.6	1.0	17.7	18.3	1.0	17.7	18.3
30–34	2.6	9.8	12.3	2.4	9.7	11.9	2.5	10.7	12.7	2.4	11.0	13.0	1.8	10.5	11.9	1.2	9.0	9.9	1.0	9.0	9.7
35–39	2.4	6.8	9.0	2.1	6.5	8.5	2.4	7.4	9.5	2.3	7.3	9.3	2.0	7.1	8.8	1.3	5.8	6.9	1.3	6.2	7.2
40–44	2.3	5.3	7.5	2.2	5.1	7.2	2.6	6.1	8.3	2.8	6.3	8.6	2.2	6.0	7.8	1.5	4.7	5.9	1.3	5.1	6.2
45–49	2.1	4.6	6.6	2.0	4.5	6.4	2.5	5.3	7.4	2.4	5.4	7.5	2.2	5.0	6.9	1.6	4.1	5.5	1.7	4.5	5.8
50+	1.8	3.6	5.3	1.8	3.5	5.2	2.4	4.2	6.3	2.5	4.5	6.7	2.1	4.4	6.2	1.4	3.1	4.3	1.6	3.6	5.0

Source: [2025 Cervical Cancer Elimination Progress Report](#) (Analysis of National Cancer Screening Register data, see [methodology](#)). HPV types 16 and/or 18 (16/18); Types other than 16 and 18 include detection of one of the 12 other oncogenic HPV types (Other); Any oncogenic HPV types (Any).

Indicator: Prevalence of HPV infection among Aboriginal and Torres Strait Islander peoples participating in the National Cervical Screening Programs

Status of indicator

Figure 2.2 Age-specific oncogenic HPV prevalence (%) among Aboriginal and Torres Strait Islander peoples aged 25–74 years participating in the National Cervical Screening Program (NCSP) in 2024



Source: [2025 Cervical Cancer Elimination Progress Report](#) (Analysis of National Cancer Screening Register data, see [methodology](#)) and the Women’s HPV Indigenous Non-Indigenous Urban Rural Study (WHINURS) study for historical estimates of HPV types 16 and/or 18 (HPV16/18) among women attending screening in 2005–07²⁶. Estimates of prevalence in 2024: HPV16/18; prevalence of HPV types other than 16 and 18, including detection of one of the 12 other oncogenic HPV types (Other oncogenic); and any oncogenic HPV type (Any oncogenic).

In 2024, among Aboriginal and Torres Strait Islander peoples aged 25–74 years who participated in cervical screening, the prevalence of any oncogenic HPV type was 12.1%. This included 2.0% with HPV16/18 and 10.5% with other oncogenic types. The prevalence of HPV16/18 was lowest among people aged 25–29 years (1.0%), increased with age to peak at 2.7% among those aged 45–49 years, and then declined slightly to 2.6% among those aged 50 years and older (**Figure 2.2**).

Prevalence in the younger age groups is notably lower than that observed before the introduction of the HPV vaccination program, when the prevalence of HPV16/18 among Aboriginal and Torres Strait Islander screening participants was reported at 8.0%, 5.7%, and 3.7% among those aged 25–29, 30–34, and 35–39 years, respectively²⁶ (**Figure 2.2**).

Between 2018 and 2024, the prevalence of HPV16/18 among Aboriginal and Torres Strait Islander screening participants declined slightly, from 2.8% to 2.0%. During the same period, the age distribution of oncogenic HPV types other than 16 or 18 remained consistent, with the highest prevalence observed in younger age groups each year and decreasing prevalence with advancing age (**Table 2.2**).

Table 2.2 Age-specific oncogenic HPV prevalence (%) among Aboriginal and Torres Strait Islander peoples aged 25–74 years participating in the National Cervical Screening Program (NCSP), 2018–2024

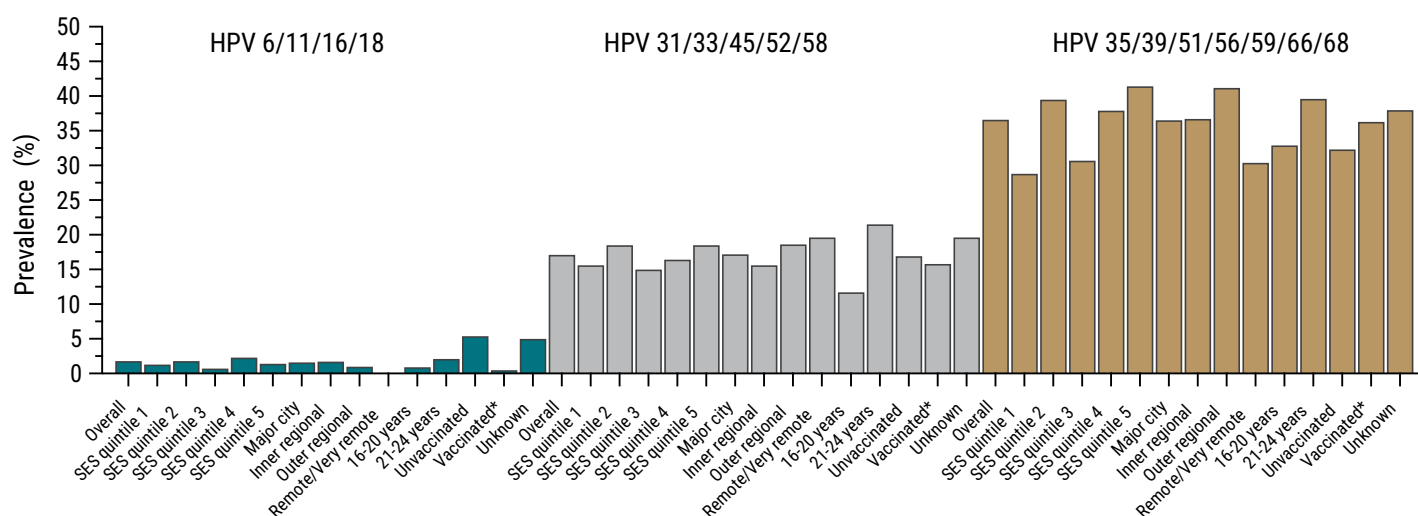
	2018			2019			2020			2021			2022			2023			2024		
	16/18	Other	Any	16/18	Other	Any	16/18	Other	Any	16/18	Other	Any	16/18	Other	Any	16/18	Other	Any	16/18	Other	Any
Nationally	2.8	9.7	12.3	2.7	10.0	12.5	2.8	11.6	14.0	2.6	11.8	14.1	2.4	12.0	14.3	2.2	10.4	12.2	2.0	10.5	12.1
Age group																					
25–29	2.2	18.1	20.1	1.5	18.5	19.9	1.7	19.3	20.6	1.4	18.2	19.4	1.2	19.1	20.0	1.0	18.9	19.7	1.0	19.5	20.3
30–34	2.9	11.8	14.5	3.1	12.7	15.6	3.0	12.8	15.3	2.8	13.7	16.1	2.1	12.7	14.7	1.6	12.5	13.7	1.0	12.0	12.8
35–39	2.8	9.6	12.2	3.0	9.3	12.1	3.9	10.7	14.0	2.9	9.5	12.0	2.9	10.6	13.0	2.1	10.5	12.2	2.2	9.6	11.3
40–44	3.4	7.3	10.6	3.7	7.6	11.1	3.6	8.8	11.7	4.4	8.5	12.4	2.9	10.2	12.9	3.0	8.7	11.2	2.6	8.7	11.0
45–49	3.2	6.8	10.0	3.0	6.8	9.5	2.8	7.5	10.1	3.3	9.2	11.8	3.9	7.7	11.0	3.4	7.3	10.1	2.7	7.8	10.0
50+	2.7	4.5	7.0	2.8	4.9	7.7	3.0	6.1	8.7	3.0	6.0	8.5	3.2	6.2	9.1	2.6	5.6	7.8	2.6	6.3	8.3

Source: 2025 Cervical Cancer Elimination Progress Report (Analysis of National Cancer Screening Register data, see [methodology](#)). HPV types 16 and/or 18 (16/18); Types other than 16 and 18 include detection of one of the 12 other oncogenic HPV types (Other); Any oncogenic HPV types (Any).



Indicator: Prevalence of HPV infection among young females (16–24 years) not yet eligible for cervical screening

Status of indicator

Figure 2.3 HPV prevalence (%) in residual chlamydia specimens from females aged 16–24 by key demographics, 2021–2022

Source: National HPV Monitoring Program (see [methodology](#)). *The prevalence of quadrivalent HPV types (6, 11, 16 or 18) among vaccinated females was less than 0.5%; absolute proportions were suppressed where the number of cases was fewer than 6, due to confidentiality requirements.

2. MONITORING INFECTION

Among nearly 1,800 residual samples from females aged 16–24 undergoing chlamydia testing, the overall prevalence of any oncogenic HPV type was 43.3%. Quadrivalent vaccine-targeted types were detected at low levels: HPV16/18 in 1.2% and HPV6/11 in 0.6%. The five additional oncogenic types included in the nonavalent vaccine (HPV 31, 33, 45, 52, 58) were detected in 17.1% of the samples. In contrast, other oncogenic types not covered by current vaccines (HPV types 35, 39, 51, 56, 59, 66, and 68) were identified in 36.6% of samples (**Figure 2.3; Table 2.3**).

The prevalence of vaccine-targeted types (HPV 6, 11, 16, 18) was low across all age groups, remoteness areas, and socioeconomic strata. In contrast, other oncogenic types were more frequently detected across all demographic categories. Variations by remoteness and socioeconomic status were modest; however, a modestly higher prevalence of any oncogenic HPV was observed in outer regional areas (45.4%) and among females in the highest socioeconomic quintile (48.7%) (**Table 2.3**).

Most participants (56.6%) had a record of receiving at least one HPV vaccine dose in the AIR, while 7.3% had no recorded doses, and 36.1% had unknown vaccination status because their records could not be verified. The prevalence of quadrivalent vaccine-preventable types was <0.5% among vaccinated females, 5.4% among unvaccinated females, and approximately 5% among those with unknown status. The prevalence of the additional oncogenic types targeted by the nonavalent vaccine was similar across vaccination groups, ranging from 15.8% to 19.6% (**Table 2.3**).

Table 2.3 HPV prevalence (%) in residual chlamydia specimens from females aged 16–24 by key demographics, 2021–2022

	Oncogenic	6/11/16/18	31/33/45/52/58	35/39/51/56/59/66/68
Characteristic				
Overall	43.3	1.8	17.1	36.6
Area-based socioeconomic status				
Group 1 (lowest)	36.4	1.3	15.6	28.8
Group 2	45.1	1.8	18.5	39.5
Group 3	37.1	0.7	15.0	30.7
Group 4	43.7	2.3	16.4	37.9
Group 5 (highest)	48.7	1.4	18.5	41.4
Remoteness area				
Major city	43.2	1.6	17.2	36.5
Inner regional	43.4	1.7	15.6	36.7
Outer regional	45.4	1.0	18.6	41.2
Remote/Very remote	39.1	0.0	19.6	30.4
Age group				
16–20 years	36.9	0.9	11.7	32.9
21–24 years	48.5	2.1	21.5	39.6
HPV vaccination status				
No doses recorded on AIR	40.8	5.4	16.9	32.3
Received at least one dose	41.9	<0.5	15.8	36.3
Unknown	46.1	~5.0	19.6	38.0

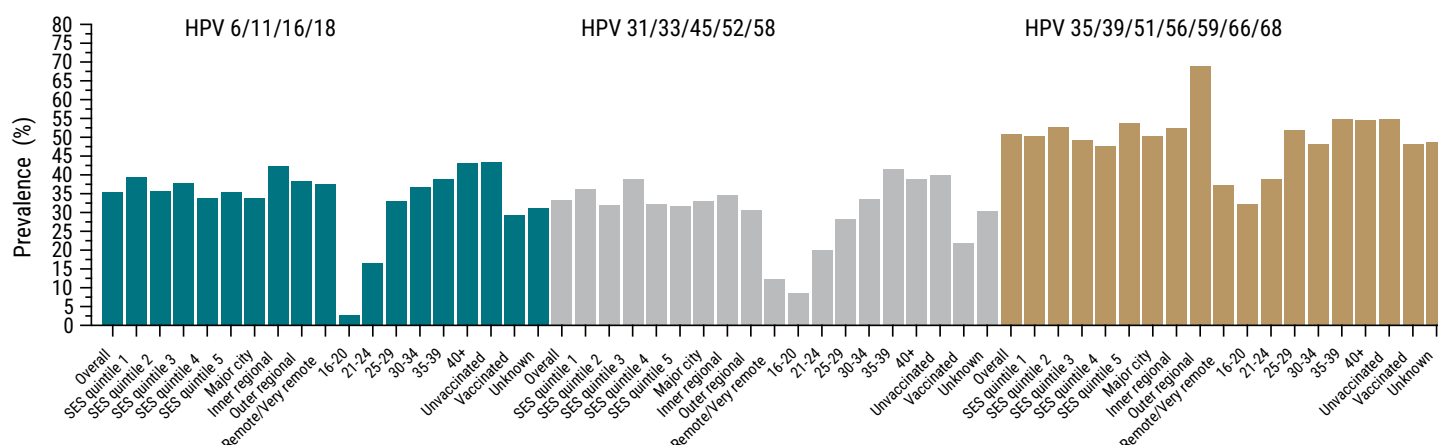
Source: IMPACT monitoring program (see [methodology](#)). *Oncogenic types (Oncogenic); Quadrivalent HPV types (6, 11, 16 or 18); Five remaining types of the nonvalent vaccine (31, 33, 45, 52 or 58); Oncogenic types, not vaccine-preventable (35, 39, 51, 56, 59, 66 or 68).



Indicator: Prevalence of HPV infection among gay, bisexual and other men who have sex with men

Status of indicator

Figure 2.4 HPV prevalence (%) in residual rectal specimens submitted for sexually transmitted infection testing, males aged ≥16 years, by key demographics, 2021–2022



Source: IMPACT monitoring program (see [methodology](#)).

Among nearly 1,500 rectal samples from males aged 16 years and older submitted for sexually transmitted infection testing in 2021–2022, the overall prevalence of any oncogenic HPV type was 66.4%. Quadrivalent vaccine-targeted types were detected in 35.5% of samples (HPV 6, 11, 16, 18). The five additional oncogenic types included in the nonavalent vaccine (HPV 31, 33, 45, 52, 58) were detected in 33.5% of the samples. In contrast, other oncogenic types not covered by current vaccines (HPV 35, 39, 51, 56, 59, 66, 68) were identified in 51.1% of samples (Figure 2.4; Table 2.4).

Across all HPV groupings, prevalence was lowest among males aged 16–20 years and increased with age, peaking in older age groups. Detection of any oncogenic HPV type ranged from 35.3% in those aged 16–20 to 77.8% in those aged 60 and above. A similar increasing pattern by age was observed for quadrivalent vaccine types (from 2.9% to 49.2%), nonavalent additional types (8.8% to 52.1%), and other oncogenic types (32.4% to 57.1%) (Figure 2.4). Geographic and socioeconomic variations were modest. Prevalence of all HPV groupings was highest among males living in outer regional areas (e.g. 80.8% for any oncogenic HPV, 69.2% for other oncogenic types). It showed some fluctuation across socioeconomic groups without a clear gradient (Table 2.4).

Most males were unvaccinated or had unknown vaccination status (93.5%). Among 95 males with HPV vaccine doses recorded in AIR (6.5%), the median age at first dose was 25 years (IQR: 14–30): 27 (28%) received their first dose before age 18; 24 (25%) between ages 18–25; and 44 (46%) at age 26 or older. The prevalence of each HPV grouping was highest among unvaccinated males, followed by those with an unknown status, and lowest among those who had received at least one vaccine dose. For example, any oncogenic HPV types were detected in 73.4% of unvaccinated males compared with 62.1% of vaccinated males; detection of the five additional nonavalent types was 40.1% and 22.1%, respectively (Table 2.4).

Table 2.4 HPV prevalence (%) in residual rectal specimens submitted for sexually transmitted infection testing, males aged ≥16 years, by key demographics, 2021–2022

	Oncogenic	6/11/16/18	31/33/45/52/58	35/39/51/56/59/66/68
Characteristic				
Overall	66.4	35.5	33.5	51.1
Area-based socioeconomic status				
Group 1 (lowest)	67.0	39.6	36.3	50.6
Group 2	67.3	35.9	32.1	52.8
Group 3	69.0	38.0	39.1	49.5
Group 4	64.6	33.8	32.5	47.8
Group 5 (highest)	66.5	35.5	32.0	53.9
Remoteness area				
Major city	65.9	33.9	33.3	50.4
Inner regional	67.6	42.3	34.8	52.6
Outer regional	80.8	38.5	30.8	69.2
Remote/Very remote*	50.0	37.5	12.5	37.5
Age group				
16–20	35.3	2.9	8.8	32.4
21–24	49.6	16.5	20.3	39.1
25–29	63.1	33.1	28.4	52.2
30–34	63.0	36.7	33.7	48.5
35–39	74.7	39.0	41.8	54.9
40+	74.5	43.1	39.1	54.7
HPV vaccination status				
No doses recorded on AIR	73.4	43.5	40.1	55.1
Received at least one dose	62.1	29.5	22.1	48.4
Unknown	62.6	31.2	30.6	48.8

Source: IMPACT monitoring program (see [methodology](#)) *Oncogenic types (Oncogenic); Quadrivalent HPV types (6, 11, 16 or 18); Five remaining types of the nonvalent vaccine (31, 33, 45, 52 or 58); Oncogenic types, not vaccine-preventable (35, 39, 51, 56, 59, 66, 68) *Proportions are based on denominators <10 and should be interpreted with caution.

Data gaps and limitations

- Aboriginal or Torres Strait Islander identification was determined by whether a person had ever self-reported their status, as recorded in the NCSR. Indigenous status data in the NCSR are incomplete. Therefore, data reflect positivity rates among participants who have ever identified as Aboriginal and/or Torres Strait Islander, rather than rates for all Aboriginal and Torres Strait Islander participants.
- The NCSR currently reports only grouped oncogenic HPV results, as many standard screening assays do not differentiate between individual HPV genotypes for non-16 or -18 oncogenic HPV types. In addition, the register does not currently capture type-specific data when available.
- Currently, there is no data linkage between the NCSR and the AIR, which prevents HPV positivity data from being reported by vaccination status within the screening program.
- For young females and males undergoing testing for sexually transmitted infections, vaccination status could not be confirmed for 32% and 41% of participants, respectively, because some pathology providers declined to release identifiers to the AIHW for linkage to AIR data.
- Data on Aboriginal and Torres Strait Islander status and other priority populations, such as those from culturally and linguistically diverse backgrounds, are not routinely reported on pathology requests. As a result, it is not possible to report on HPV prevalence in residual specimens for these groups.
- There are no ongoing routine data sources for monitoring HPV infection in females under the age of 25 years and at non-cervical sites, including anogenital sites, among GBMSM. Surveillance among these groups relies on periodic, ad hoc studies.

Data Interpretation

Australia's HPV vaccination program continues to demonstrate sustained, population-level impact. In 2024, the prevalence of HPV16/18 among screening program participants aged 25–74 was 1.4%, with the lowest rates (1.0%) observed among those aged 25–29 years. This low prevalence was consistent across socio-demographic subgroups and geographic regions. Similarly, among 16–24-year-old females attending chlamydia screening in 2021 and 2022, HPV16/18 prevalence was very low (1.2%). Fewer than 1 in 50 tested positive for any quadrivalent vaccine-preventable types, a marked

reduction from historical estimates of 1 in 5 females (22%) before program implementation²⁶. These findings clearly demonstrate the impact of the Australian HPV vaccination program.

The linkage of HPV results with vaccination records from AIR further demonstrates the vaccine's impact. Females aged 16–24 years with an AIR record of receiving at least 1 vaccine dose had a lower detection rate of quadrivalent vaccine-preventable HPV types (<0.5%) than unvaccinated females (5.4%). These findings reflect the continued impact of quadrivalent vaccination, as most vaccinated females in this group (92%) received only quadrivalent doses. In contrast, 17% had at least one of the five additional oncogenic types targeted by the nonavalent vaccine (HPV 31, 33, 45, 52, 58), and 37%. The latter group includes types classified as oncogenic but with substantially lower and less certain carcinogenic potential than vaccine-preventable types, including types that are only considered probable or not definitively carcinogenic.

Following the 2018 shift to the nonavalent vaccine, only females aged ≤17 years in 2022 would have received nonavalent doses through the school-based program. Among the small proportion who received at least one nonavalent dose, 6.5% had one of the five additional nonavalent vaccine types detected, compared with 16% among those who received only quadrivalent doses. This may indicate the early impact of the nonavalent HPV vaccine. Cross-protection, particularly against HPV31, 33, and 45, may contribute to the observed reductions in overall prevalence for this mostly quadrivalent-vaccinated cohort, as documented by previous surveillance studies^{27,28}.

Among screening program participants, reductions in older age groups may also reflect the program's impact: by 2024, those eligible for catch-up vaccination in 2007 (aged under 26 at the time) had reached age 43, while those vaccinated through the school-based program had reached age 34 (See Implementation timeline). Overall, oncogenic HPV prevalence declined from 8.8% in 2018 to 7.3% in 2024, after rising transiently in 2021 and 2022, consistent with many participants attending their second screening after an HPV-negative test five years prior. The slight increase in HPV positivity in 2024 compared with 2023, driven mainly by non-16/18 types, likely reflects the high uptake of cervical screening self-collection samples rather than a true rise in infection. Vaginal self-collected samples generally have a higher HPV detection rate than cervical samples²⁹. However, this may also be influenced by the characteristics of women who choose self-sampling, particularly those who were previously under-screened²⁹.

Oncogenic HPV prevalence was higher among Aboriginal and Torres Strait Islander participants (12.1%). This may partially reflect age-related differences, with a younger median age among Aboriginal and Torres Strait Islander women compared to the overall population (24 years and 38 years in 2021, respectively)³⁰, and lower screening participation rates³¹. However, HPV16/18 positivity was very low for Aboriginal and Torres Strait Islander cervical screening participants in birth cohorts offered HPV vaccination, similar to the national population, highlighting the success of Australia's school-based HPV vaccination program in achieving comparable impacts¹⁸. Despite this, disparities in cervical screening participation and disease outcomes for Aboriginal and Torres Strait Islander peoples (and those living in remote and very remote areas) persist, emphasising the ongoing need to address inequities.

For additional information on HPV prevalence among cervical screening program participants, also refer to the [2025 Cervical Cancer Elimination Progress Report](#) and the [AIHW National Cervical Screening Program monitoring report 2025](#).

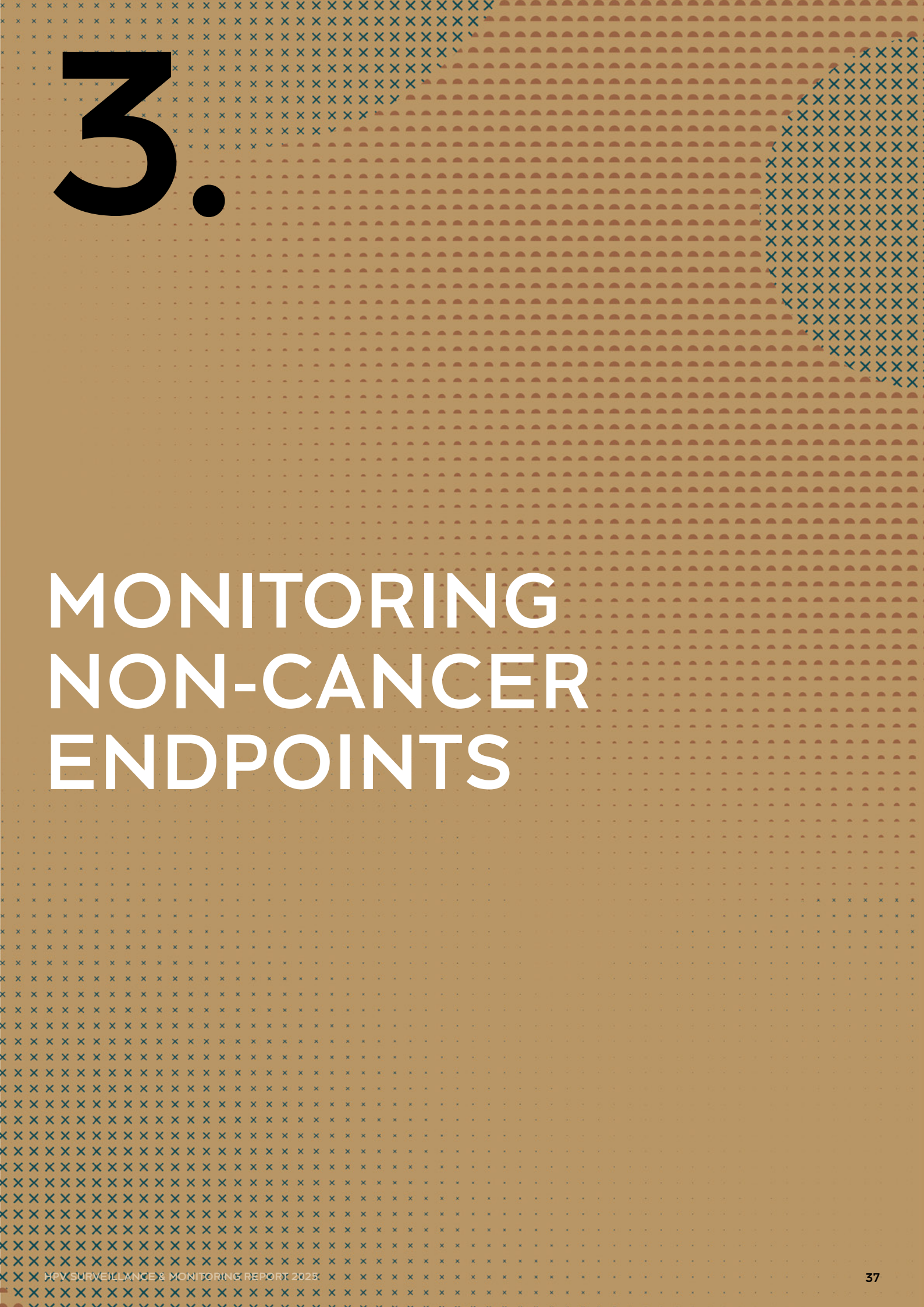
Among males, HPV prevalence in rectal samples remained high, reflecting ongoing transmission and the historically more limited vaccine coverage, given the more recent introduction of HPV vaccination for boys.

Among those aged ≥ 16 years submitting rectal samples for testing in 2021 and 2022, 66% had anal oncogenic HPV, and more than 20% had HPV16/18. Consistent with previously published estimates, one-third (34%) had at least one of the five additional types targeted by the nonavalent vaccine^{21,32,33}. Prevalence was lowest among males aged 16–20 years, increasing gradually with age. This pattern contrasts with females, where prevalence decreases with increasing age, and likely reflects high rates of newly acquired HPV infection from ongoing sexual exposure³³.

Most males in this sample were unvaccinated or had unknown vaccination status (93.5%). Among those with an AIR record of at least one dose, the median age at vaccination was 25 years (IQR 14–30). This reflects the limited eligibility for males: by 2020, only those aged ≤ 22 years had access to school-based vaccination, with community catch-up available up to age 19. Early evidence suggests that universal vaccination has already reduced HPV prevalence among young GBMSM, with declines similar to those observed in females²². In this sample, the small proportion of vaccinated males (6.5%) had a lower prevalence of quadrivalent HPV types (29.5%) compared with unvaccinated males (43.5%). However, all vaccinated

males with quadrivalent vaccine-preventable types had received their first dose after adolescence (≥ 23 years). Australian Technical Advisory Group on Immunisation (ATAGI) recommends HPV vaccination for all MSM at any age because of their markedly elevated anal cancer risk; however, the vaccine is publicly funded only up to and including age 25 years. The prevalence of quadrivalent vaccine-preventable types and the five additional types in the nonavalent vaccine was lowest among 16–20-year-olds (2.9% and 8.8%, respectively) and 21–24-year-olds (16.5% and 20.3%). By contrast, oncogenic HPV types not targeted by vaccination remained common across all age groups ($>30\%$), a pattern consistent with the early impacts of universal vaccination on vaccine-preventable HPV types.

Surveillance studies in Australia have provided important baseline prevalence estimates for populations eligible for three-dose quadrivalent vaccination, as well as for GBMSM before universal vaccination. Since the program's introduction in 2007, these surveys have tracked reductions in the prevalence of vaccine-preventable types (particularly among young females), demonstrating the population-wide effectiveness of the quadrivalent vaccine program, achieved through sustained high coverage. Subsequent surveys among nonavalent vaccine-eligible populations will enable evaluation of nonavalent program impacts.



3.

MONITORING NON-CANCER ENDPOINTS

3. MONITORING NON-CANCER ENDPOINTS

3.1 Monitor the incidence of genital warts

Justification for this monitoring objective

Genital warts, also known as anogenital warts or condylomata acuminata, are benign lesions caused by infection with low-risk HPV types 6 or 11 in over 90% of cases^{34,35}. While often asymptomatic, they can cause bleeding, itching and pain as well as psychological and psychosexual distress. HPV vaccines used in Australia's national HPV vaccination program protect against HPV types 6 and 11.

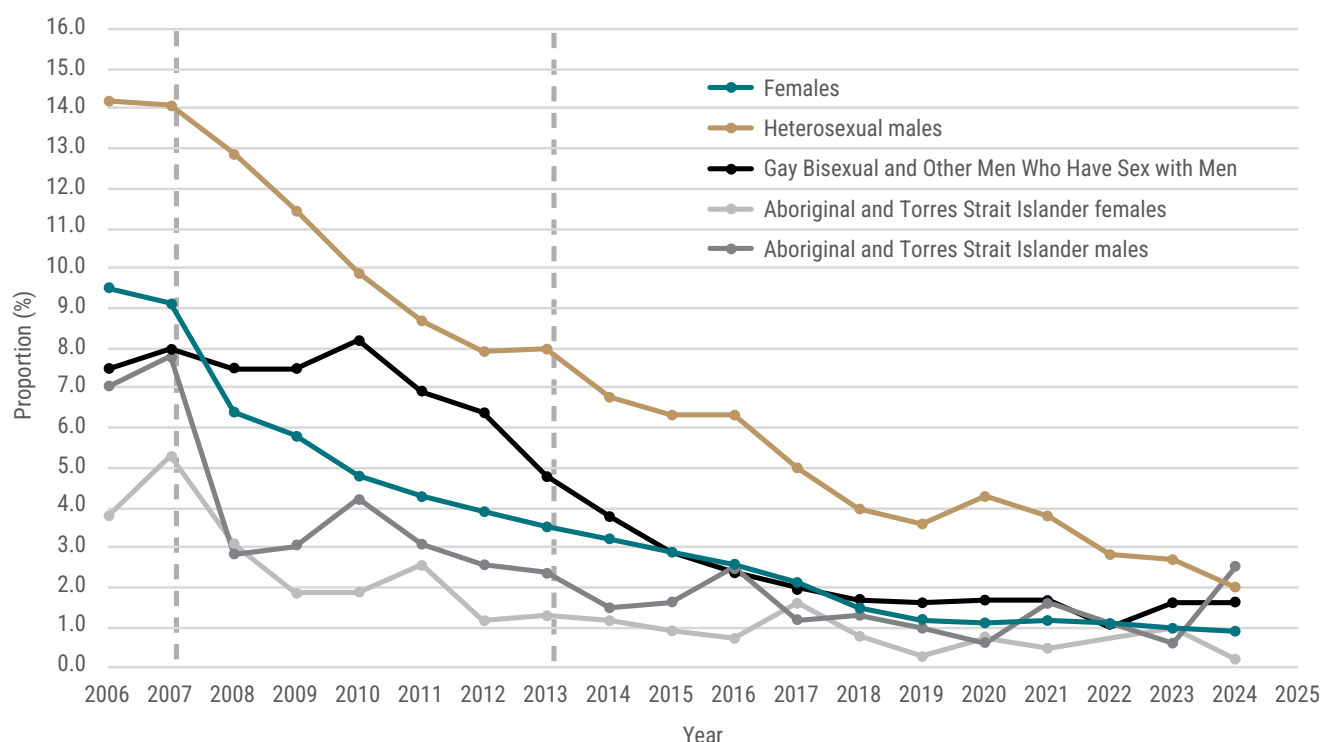
Genital warts are a practical, early, and measurable outcome for monitoring vaccine impact, as they develop soon after infection and are easily diagnosed. Prior to the HPV vaccination program, they were one of the most common reasons for presentation to sexual health services³⁶. Genital warts are also the only routinely available early disease outcome for monitoring vaccine impact in boys and people without a cervix, until longer-term cancer outcomes can be assessed.



Indicator: Genital wart incidence in sexual health clinic populations

Status of indicator

Figure 3.1 Proportion (%) of sexual health clinic attendees diagnosed with genital warts at first visit, 2006–2024



Source: The Australian Collaboration for Coordinated Enhanced Sentinel Surveillance of Sexually Transmissible Infections and Blood-Borne Viruses (see [methodology](#)) Crude/unadjusted rates are included in this figure. The first dotted line represents the start of the national HPV vaccination program for girls in 2007. The second line represents the program expansion to include boys in 2013.

As of 2024, diagnoses of genital warts at first sexual health clinic visits remain low across all population groups, continuing a sustained decline since 2006 (**Figure 3.1**). This decline has been especially pronounced in age cohorts eligible for HPV vaccination.

Among females, genital wart diagnoses at first visit dropped substantially, particularly in younger age groups. In 2024, only 0.7% of females aged 21–24 years were newly diagnosed, compared to 13.8% in 2006. Before the HPV vaccination program, diagnoses peaked at ages 21–24; in recent years, the highest proportions have shifted to females aged 35–39 and ≥40 years. Geographic disparities observed in 2006 have narrowed significantly. At that time, diagnosis rates ranged from 5.2% in inner regional areas to 11.4% in remote/very remote areas. In 2024, this range had declined to 0–1.1%, with a 0% diagnosis rate in remote/very remote areas in three of the last six years. Socioeconomic differences have also diminished. In 2024, the difference between the highest- and lowest-SES quintiles in residential areas was only 0.4%, down from 4.3% in 2006 (**Table 3.1**).

Among males, the incidence of genital warts has also decreased steadily. For heterosexual males aged 21–24 years, diagnoses dropped from 17.0% in 2006 to 1.2% in 2024 (**Table 3.2**). Among GBMSM, the rate in the same age group declined from 10.0% to 1.1%. In 2006, geographic variation among heterosexual males ranged from 9.1% in remote/very remote areas to 16.1% in major cities; among GBMSM, it ranged from 4.4% in outer regional areas to 18.2% in remote/very remote areas (**Table 3.2**). By 2024, diagnoses were ≤2.2% across all regions for both heterosexual and GBMSM males, with 0% in remote/very remote areas (**Table 3.2; Table 3.3**).

Among Aboriginal and Torres Strait Islander peoples, genital wart diagnoses at first visit have also declined. In 2024, 0.2% of females and 2.5% of males were diagnosed, compared to 3.8% and 7.0%, respectively, in 2006. Among those aged 21–24 years, the diagnosis rate was 0.0% for both genders in 2024, down from 7.3% for females and 9.2% for males in 2006 (**Table 3.4; Table 3.5**).

Table 3.1 HPV Proportion (%) of female sexual health clinic attendees diagnosed with genital warts at first visit, 2006–2024

	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Characteristic																			
Nationally	9.5	9.1	6.4	5.8	4.8	4.3	3.9	3.5	3.2	2.9	2.6	2.1	1.5	1.2	1.1	1.2	1.1	1.0	0.9
Area-based socioeconomic status																			
Group 1 (lowest)	9.6	9.0	5.3	5.1	4.3	4.2	3.5	2.9	3.1	2.7	2.9	1.4	0.9	1.0	1.0	1.8	1.5	1.2	1.1
Group 2	7.5	8.3	5.6	5.0	3.9	3.8	3.2	3.0	2.8	2.6	2.6	2.1	1.6	1.0	1.1	1.0	1.3	1.2	1.0
Group 3	9.7	8.6	5.7	6.2	4.7	4.2	4.0	3.4	3.0	3.1	2.8	2.0	1.7	1.5	1.4	1.5	0.9	1.1	0.7
Group 4	11.1	10.8	8.8	7.1	5.4	5.0	4.5	4.0	3.3	3.3	3.0	2.8	1.9	1.4	0.8	0.9	1.5	1.1	1.0
Group 5 (highest)	11.8	11.7	8.0	7.0	6.6	5.3	5.2	4.5	4.3	3.7	3.1	2.6	1.6	1.3	1.3	1.2	0.6	0.7	0.8
Remoteness area																			
Major city	11.6	11.1	7.7	7.0	5.7	5.4	4.8	4.2	3.9	3.7	3.4	2.8	2.0	1.6	1.3	1.3	1.2	1.2	1.1
Inner regional	5.2	5.5	4.3	4.1	3.1	2.8	3.2	2.6	2.2	1.9	2.0	1.2	1.2	0.8	0.9	0.9	0.6	0.5	0.2
Outer regional	5.9	6.5	4.3	2.8	2.4	1.6	1.4	1.3	1.1	0.5	0.7	0.8	0.4	0.3	0.3	0.5	0.6	0.5	0.3
Remote/Very remote	11.4	7.9	4.0	8.5	3.1	1.1	2.4	0.6	0.7	1.5	1.0	1.3	1.7	0.0	0.9	1.5	1.6	0.0	0.0
Age group																			
15–20	11.8	12.0	6.6	4.5	2.4	1.7	2.2	1.5	1.5	1.6	1.4	0.8	0.4	0.5	0.2	0.3	0.5	0.1	0.3
21–24	13.8	13.5	8.5	7.1	5.3	5.0	3.5	3.7	2.7	2.8	2.3	1.9	1.5	1.0	0.9	0.6	0.3	0.3	0.7
25–29	9.4	7.9	7.5	6.0	5.9	5.1	5.3	4.4	4.0	3.2	3.2	2.6	1.8	1.4	1.1	1.0	1.0	1.1	0.9
30–34	8.0	7.3	6.2	7.2	7.4	5.6	5.4	4.3	4.5	3.7	3.4	2.6	2.0	1.6	1.4	1.7	1.3	1.3	1.3
35–39	6.3	6.5	4.7	5.9	5.7	6.3	4.6	6.3	5.0	4.9	4.1	3.0	1.5	1.4	1.2	1.9	2.0	2.0	1.6
≥40	3.7	3.6	3.3	5.8	4.5	5.0	5.1	3.9	4.1	3.9	4.0	3.2	2.8	2.1	1.9	3.3	3.3	3.0	1.3

Source: The Australian Collaboration for Coordinated Enhanced Sentinel Surveillance of Sexually Transmissible Infections and Blood-Borne Viruses (see [methodology](#)).

3. MONITORING NON-CANCER ENDPOINTS

Table 3.2 Proportion (%) of heterosexual male sexual health clinic attendees diagnosed with genital warts at first visit, 2006–2024

	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Characteristic																			
Nationally	14.2	14.1	12.9	11.5	9.9	8.7	7.9	8.0	6.8	6.3	6.3	5.0	4.0	3.6	4.3	3.8	2.8	2.7	2.0
Area-based socioeconomic status																			
Group 1 (lowest)	11.7	12.8	10.1	9.8	9.0	9.6	7.1	8.9	6.4	5.9	6.6	4.1	3.1	3.3	3.2	4.5	3.0	2.5	2.2
Group 2	12.2	12.1	11.7	10.2	9.6	7.5	7.4	6.8	6.1	5.9	6.1	6.1	4.4	4.0	3.8	4.1	2.2	2.2	2.3
Group 3	15.3	14.4	12.5	11.8	9.3	8.2	7.8	8.5	5.9	5.4	6.3	5.5	3.6	3.1	5.2	3.2	2.9	2.4	1.3
Group 4	16.2	16.7	16.3	13.8	11.7	9.6	8.7	8.6	7.4	7.7	7.2	5.8	4.8	3.8	5.4	4.7	3.1	3.0	2.3
Group 5 (highest)	16.1	15.7	15.4	14.4	11.9	10.8	10.3	9.3	8.7	7.3	7.1	5.5	4.5	3.1	4.0	3.3	2.1	2.5	2.1
Remoteness area																			
Major city	16.1	15.8	14.9	13.3	11.5	10.2	9.0	9.0	7.6	7.3	7.5	5.9	4.5	3.8	4.6	4.1	2.7	2.6	2.2
Inner regional	10.0	10.1	9.8	9.5	7.7	5.5	7.6	6.1	5.8	3.6	3.5	5.4	3.3	1.7	4.2	2.3	1.3	1.7	1.3
Outer regional	10.3	9.3	7.8	7.7	6.6	5.6	5.4	5.2	3.9	3.8	2.7	2.0	3.0	2.3	3.0	3.7	3.2	2.5	1.4
Remote/Very remote	9.1	7.0	7.2	5.3	2.7	3.3	3.6	4.1	4.3	1.0	4.2	4.8	4.1	1.0	2.0	2.1	0.0	0.0	0.0
Age group																			
15–20	10.9	10.8	8.0	5.0	2.9	2.9	1.8	1.7	1.9	1.3	0.9	1.3	0.6	0.6	0.3	0.4	0.5	0.4	0.0
21–24	17.0	18.1	16.0	12.5	10.4	7.6	7.4	7.0	5.8	5.3	5.2	4.0	3.4	2.4	3.1	2.6	0.9	1.2	1.2
25–29	17.4	16.9	15.5	14.7	12.9	10.5	9.3	9.1	7.9	8.0	7.7	5.8	4.6	4.3	4.7	3.7	2.9	3.1	2.3
30–34	15.8	16.1	15.9	14.2	12.1	11.8	9.6	9.8	9.3	7.6	8.1	6.4	5.4	4.8	5.8	5.7	4.2	3.3	2.2
35–39	13.4	12.7	12.9	14.3	12.3	13.4	10.9	9.9	9.9	9.2	7.9	7.2	4.6	6.1	5.5	5.4	4.5	3.3	4.1
≥40	8.8	8.1	7.0	7.4	7.9	7.8	8.3	9.6	6.5	6.6	7.3	5.7	5.2	4.6	5.9	5.2	4.6	5.0	2.6

Source: The Australian Collaboration for Coordinated Enhanced Sentinel Surveillance of Sexually Transmissible Infections and Blood-Borne Viruses (see [methodology](#)).

Table 3.3 Proportion (%) of gay, bisexual and other men who have sex with men (GBMSM) sexual health clinic attendees diagnosed with genital warts at first visit, 2006–2024

	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Characteristic																			
Nationally	7.5	8.0	7.5	7.5	8.2	6.9	6.4	4.8	3.8	2.9	2.4	2.0	1.7	1.6	1.7	1.7	1.0	1.6	1.6
Area-based socioeconomic status																			
Group 1 (lowest)	6.8	8.4	6.9	9.2	7.3	9.0	8.3	5.0	4.0	3.0	2.8	1.5	1.1	2.2	1.7	1.5	0.8	1.5	1.8
Group 2	6.3	6.5	6.3	6.1	6.6	4.7	4.7	3.4	3.2	3.7	2.3	2.5	2.0	1.5	1.4	2.2	1.2	1.6	2.1
Group 3	7.3	8.7	7.0	9.1	7.7	7.7	7.3	5.4	3.5	2.7	2.5	2.1	1.6	1.7	2.1	1.9	0.7	1.8	1.6
Group 4	7.7	8.8	7.5	7.2	8.1	7.3	5.5	4.9	3.6	2.6	2.8	2.0	1.7	1.8	1.7	1.3	1.4	1.3	0.9
Group 5 (highest)	8.9	8.4	9.2	7.6	10.5	7.4	7.4	5.7	4.6	3.0	2.1	2.0	1.8	1.2	2.1	1.6	0.9	1.7	1.5
Remoteness area																			
Major city	8.1	8.7	7.9	7.8	8.7	7.4	6.5	5.2	4.0	2.9	2.4	2.2	1.8	1.7	1.8	1.8	1.0	1.6	1.6
Inner regional	4.5	6.2	4.6	7.1	8.7	4.6	5.6	2.5	2.5	4.3	2.5	1.2	1.1	0.9	2.0	0.3	0.9	1.6	1.1
Outer regional	4.4	4.5	6.7	4.1	2.8	3.4	5.1	1.7	2.4	2.0	4.2	1.9	1.4	1.5	1.7	1.4	0.7	1.0	0.6
Remote/Very remote	18.2	13.3	0.0	0.0	0.0	6.3	10.0	0.0	6.3	4.8	7.7	0.0	0.0	5.0	0.0	0.0	0.0	0.0	0.0
Age group																			
15–20	6.7	6.9	6.7	6.3	7.8	7.5	4.9	3.4	3.2	3.1	1.5	0.9	0.6	1.0	0.4	0.0	0.2	1.3	1.2
21–24	10.0	13.5	10.2	8.9	9.6	7.2	6.7	5.7	3.9	3.3	2.9	2.4	1.7	1.4	1.4	1.6	1.0	0.9	1.1
25–29	9.3	8.4	8.5	9.0	7.9	6.6	6.9	4.7	4.5	4.1	2.9	2.6	2.3	1.9	2.1	1.5	1.2	1.7	1.7
30–34	8.4	9.8	6.8	8.5	9.1	8.2	6.7	5.3	3.9	1.4	2.2	1.7	1.9	1.6	1.2	2.4	1.0	1.5	1.9
35–39	6.4	5.8	7.5	7.7	6.8	6.7	6.2	4.7	3.3	2.7	1.4	2.3	1.8	1.6	1.5	2.5	0.6	1.7	2.2
≥40	4.9	4.4	5.3	4.7	7.4	5.6	5.9	4.5	3.1	2.5	2.2	1.5	1.2	1.7	3.0	1.8	1.0	2.2	1.2

Source: The Australian Collaboration for Coordinated Enhanced Sentinel Surveillance of Sexually Transmissible Infections and Blood-Borne Viruses (see [methodology](#)).**Table 3.4 Proportion (%) of Aboriginal and Torres Strait Islander female sexual health clinic attendees diagnosed with genital warts at first visit, 2006–2024**

	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Characteristic																			
All	3.8	5.3	3.1	1.9	1.9	2.6	1.2	1.3	1.2	0.9	0.7	1.6	0.8	0.3	0.7	0.5	0.7	1.0	0.2
Age group																			
15–20	4.2	7.0	2.9	1.0	1.0	0.2	0.4	0.2	0.5	0.5	0.3	0.0	0.5	0.0	0.0	0.0	0.0	0.0	0.0
21–24	7.3	5.0	3.7	5.4	4.2	6.1	0.6	0.7	0.0	0.0	0.7	3.0	0.6	0.5	1.5	0.0	0.8	0.8	0.0
25–29	4.3	6.3	5.3	3.7	1.9	4.3	1.9	2.0	1.7	0.0	0.0	0.0	0.0	0.6	1.6	1.0	0.0	0.0	0.9
30–34	0.0	3.7	4.3	0.0	1.9	4.8	2.8	2.5	3.4	2.9	0.0	2.0	3.3	1.0	1.1	0.0	4.3	0.0	0.0
35–39	4.1	0.0	5.8	1.9	1.7	1.9	7.0	4.1	4.6	2.6	5.1	6.7	2.8	0.0	0.0	2.4	3.4	5.7	0.0
≥40	1.8	5.6	0.0	3.2	3.9	6.5	1.9	2.6	1.6	2.1	0.7	2.9	0.6	0.0	0.7	0.9	0.0	3.4	0.0

Source: The Australian Collaboration for Coordinated Enhanced Sentinel Surveillance of Sexually Transmissible Infections and Blood-Borne Viruses (see [methodology](#)).

3. MONITORING NON-CANCER ENDPOINTS

Table 3.5 Proportion (%) of Aboriginal and Torres Strait Islander male sexual health clinic attendees diagnosed with genital warts at first visit, 2006–2024

	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Characteristic																			
All	7.0	7.8	2.8	3.0	4.2	3.1	2.6	2.4	1.5	1.6	2.5	1.2	1.3	1.0	0.6	1.6	1.1	0.6	2.5
Age group																			
15–20	5.1	5.0	2.5	0.3	2.1	2.4	0.8	1.2	0.0	0.4	0.7	0.5	0.3	0.3	0.5	0.5	0.0	0.0	0.0
21–24	9.2	13.2	2.2	5.5	9.2	6.2	2.4	4.3	2.5	2.6	5.1	0.6	2.6	0.6	0.7	1.1	0.0	0.0	0.0
25–29	9.9	13.0	4.0	7.0	2.8	4.5	7.8	5.2	6.3	5.6	4.0	1.1	0.5	3.2	0.7	4.4	2.6	0.8	1.7
30–34	9.9	5.7	9.3	3.4	4.8	4.1	4.1	1.4	0.0	1.3	4.3	2.4	4.2	2.2	1.2	2.7	1.7	0.0	4.8
35–39	7.5	8.9	3.7	6.5	10.2	0.0	5.5	6.2	0.0	3.1	2.9	5.3	0.0	0.0	0.0	0.0	0.0	0.0	7.1
≥40	6.5	5.8	0.0	6.3	5.4	1.7	5.6	1.8	2.0	0.6	2.4	1.0	1.7	1.0	0.5	1.3	2.2	2.9	4.4

Source: The Australian Collaboration for Coordinated Enhanced Sentinel Surveillance of Sexually Transmissible Infections and Blood-Borne Viruses (see [methodology](#)).

Data gaps and limitations

- Data are drawn from 49 sexual health clinics participating in The Australian Collaboration for Coordinated Enhanced Sentinel Surveillance of Sexually Transmissible Infections and Blood-Borne Viruses (ACCESS). Rates will therefore reflect those in higher-risk populations typically seen at sexual health clinics and may not be generalisable to the broader population.
- Among GBMSM, changes in behaviour and the characteristics of those attending services over time likely contributed to the decline in genital wart diagnoses observed prior to the introduction of male vaccination.
- Information on HPV vaccine doses administered within the clinics is available in ACCESS; however, this is likely to be a substantial underestimate, as most individuals would have received their vaccinations elsewhere.
- Aboriginal and Torres Strait Islander identification is based on self-reported status in sexual health clinic databases. While data quality has improved, some variability remains: approximately 14% of first visits each year lack this information or include “prefer not to say” responses. Reported genital wart rates, therefore, reflect only those with recorded Indigenous status.
- Small numbers in some age groups for Aboriginal and Torres Strait Islander males and females and remote/very remote areas (<100 individuals in some years) warrant cautious interpretation.
- Males were classified as heterosexual based on self-reported partner gender in the past 12 months. Some misclassification may occur due to recall or reporting biases.
- Males were classified as GBMSM based on self-reported sexual orientation or ever having had an anorectal swab taken for chlamydia or gonorrhoea²⁴. Some misclassification may occur due to recall or reporting biases.
- The dataset does not distinguish between gay, bisexual, or other men who have sex with men, which may mask subgroup trends.

Data interpretation

Sustained high HPV vaccination coverage has led to substantial declines in genital wart diagnoses in populations attending sexual health clinics. Reductions were observed overall and across all sub-groups, with the most significant declines among younger cohorts eligible for school-based vaccination. By 2024, females aged ≤21 and 21–24 years had the lowest incidence, in contrast to patterns in 2006, when these groups had the highest rates. A similar trend was seen among heterosexual males. These findings are consistent with previous reports from national sentinel surveillance in sexual health clinics^{37,38}, hospitalisation data for severe disease³⁹, and general practice records^{40,41}.

Among GBMSM, genital wart diagnoses declined modestly after the introduction of female HPV vaccination in 2007 and continued to fall after the inclusion of boys in 2013, stabilising at around 1–2% by 2017–18. This decline likely exceeds what would be expected from herd protection alone and may reflect changes in the risk profile of men attending sexual health clinics over time³⁸.

Socioeconomic and geographic inequities in genital wart diagnoses have also narrowed. By 2024, declines were seen across all socioeconomic groups with minimal variability. Disparities by remoteness have also largely resolved, suggesting equitable vaccine impacts across metropolitan, regional, and remote areas.

Genital wart diagnoses have steadily declined among Aboriginal and Torres Strait Islander peoples attending sexual health clinics since 2006, especially in younger females eligible for vaccination. Diagnoses also declined among males, although rates among those aged 30 years and older have fluctuated in recent years, remaining below pre-vaccination levels.

Maintaining high vaccination coverage remains essential for sustaining low incidence. Improving the completeness of Aboriginal and Torres Strait Islander identification in sexual health clinic data and linking data with vaccination records would further support the evaluation of vaccine impact.

For additional information, also refer to the [HIV, viral hepatitis and sexually transmissible infections in Australia: Annual surveillance report 2025](#).

3.2 Monitor the incidence of recurrent respiratory papillomatosis

Justification for this monitoring objective

Recurrent respiratory papillomatosis is a rare, incurable condition caused by infection with HPV, primarily HPV6 or HPV11⁴²⁻⁴⁴. In the juvenile-onset form of the disease (JoRRP), HPV infection is acquired before or during birth, with the presence of maternal genital warts a significant risk factor, and causes the repeated growth of wart-like lesions predominantly in the upper airways, characteristically in the larynx⁴⁵. HPV exposure during birth is common, however, and it is an immunological deficit, leading to a failure to control HPV in the airways, which results in the disease⁴⁶. Classically, the disease has been noted to occur more frequently among vaginally delivered, first-born children of young mothers⁴⁷. Affected individuals often require multiple rounds of debulking surgery to maintain airway and voice function, resulting in high morbidity from the disease and risk of a fatal outcome due to complications of the disease or its treatment. Lesions may also undergo malignant transformation⁴⁸.

JoRRP is usually diagnosed in early childhood between the ages of 3 and 4 years⁴⁸, with adult-onset disease (AoRRP), which is likely a result of HPV infection acquired through sexual transmission, typically presenting between the ages of 20 and 40 years and is more common among males than females⁴⁹.

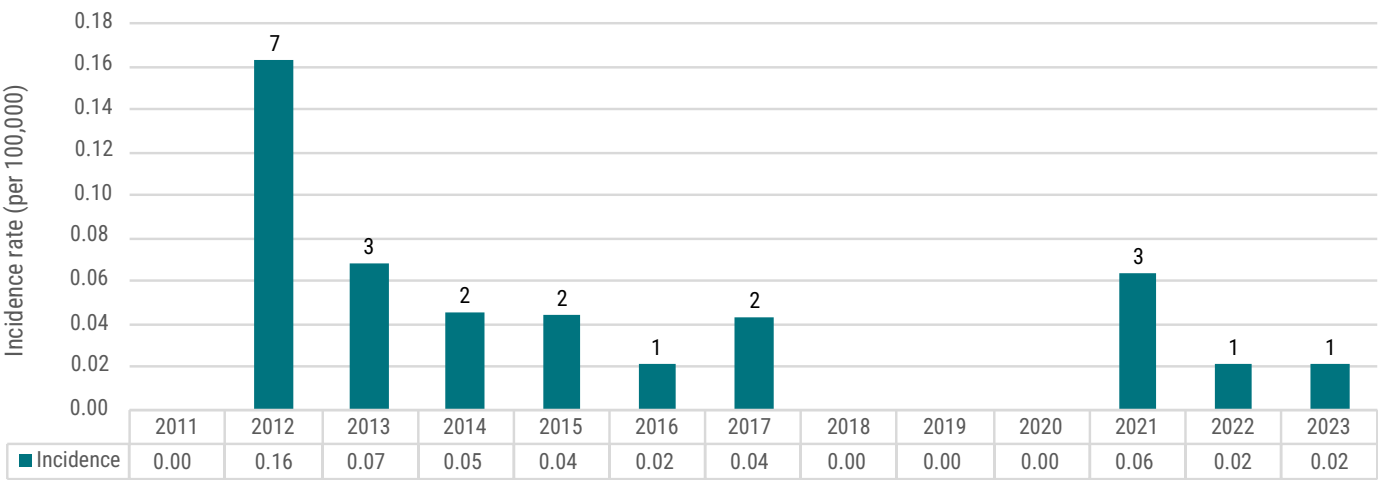
National surveillance of JoRRP through the Australian Paediatric Surveillance Unit (APSU) commenced in 2011, aimed at capturing all new cases of JoRRP for children aged under 15 years at diagnosis, presenting to paediatric ear, nose and throat specialists participating in the surveillance program⁴⁵.

Australia’s HPV vaccination program protects against HPV6 and 11, meaning the disease can be prevented through interruption of vertical transmission from mother to child and horizontal transmission of HPV 6 and 11 in adults, and even potentially eliminated in the future. It is therefore essential to monitor trends in incidence and morbidity (hospitalisation) data.

Indicator: Incidence of juvenile-onset recurrent respiratory papillomatosis

Status of indicator

Figure 3.2 Incidence rate of newly notified cases of juvenile-onset recurrent respiratory papillomatosis (JoRRP) per 100,000 children aged 0–14 years, 2011–2023



Source: Australian Paediatric Surveillance Unit Annual Surveillance Reports (see [methodology](#)). Absolute counts shown above bars.

Over the 13 years, a total of 22 new cases of JoRRP were reported in children aged 0–14 years, including 7 cases in males and 15 in females. Of these, 17 cases (77.3%) occurred before 2018. From 2011 to 2023, the incidence among Australian children aged 0–14 years has remained low, at 0.2 cases or fewer per 100,000 children, with some fluctuation likely due to the small number of cases (Figure 3.2).



Indicator: Incidence of juvenile-onset recurrent respiratory papillomatosis in Aboriginal and Torres Strait Islander children

Status of indicator

The incidence of JoRRP has remained very low from 2011 to 2023 among Aboriginal and Torres Strait Islander children aged 0–14 years. Over the 13 years, two cases of JoRRP were reported (1 male and 1 female)⁵⁰.



Indicator: HPV type in cases of juvenile-onset recurrent respiratory papillomatosis

Status of indicator

HPV genotyping information was known for 11 out of the 22 cases of JoRRP notified between 2011 and 2023. Of these, eight tested positive for HPV6, and three tested positive for HPV11, with HPV genotype results unknown for the remaining cases⁵⁰.



Indicator: HPV types in cases of juvenile-onset recurrent respiratory papillomatosis in Aboriginal and Torres Strait Islander children

Status of indicator

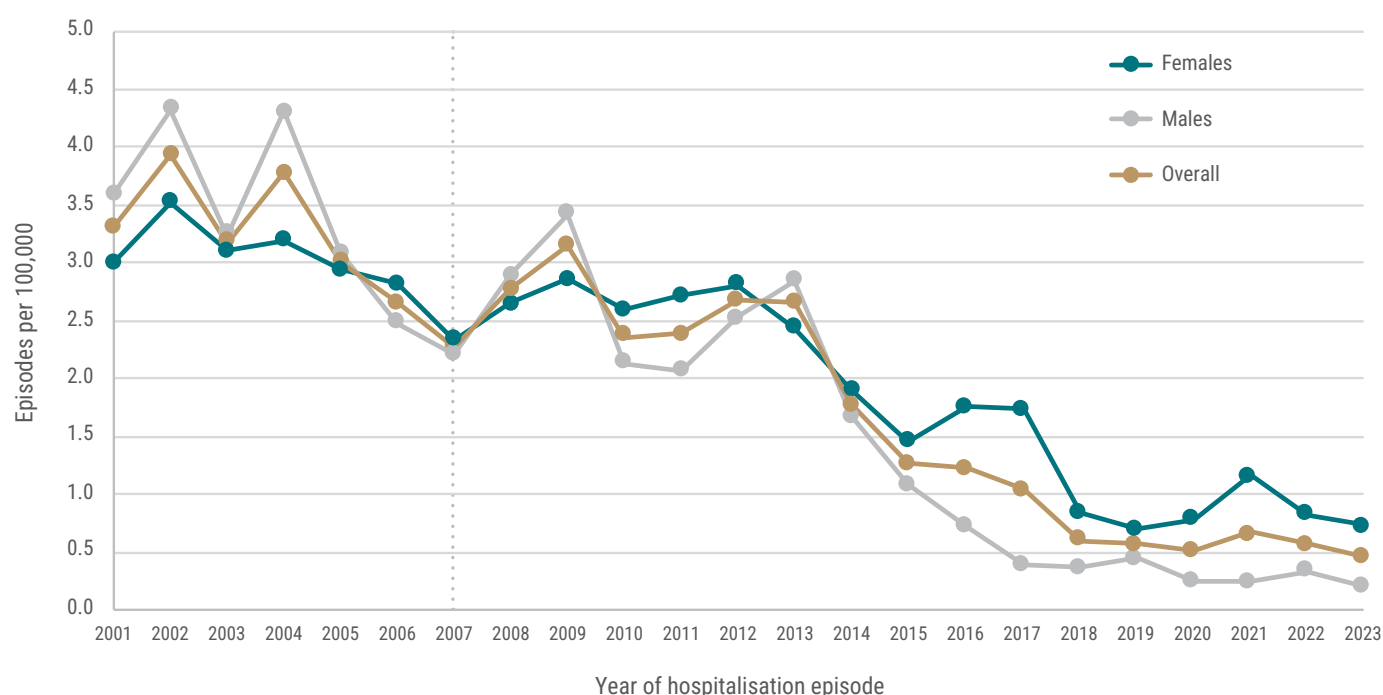
Data for this indicator are not available, as HPV genotyping data were not collected by Indigenous status.



Indicator: Hospitalisations for benign neoplasm of the larynx (coded using ICD code D14.1) by sex over time

Status of indicator

Figure 3.3 Hospitalisations for benign neoplasm of the larynx per 100,000 persons, in children aged 0–14 years by sex, 2001–2023



Source: National Hospital Morbidity Database (see [methodology](#)). The dotted line indicates the commencement of the National HPV Vaccination Program.

Between 2001 and 2023, a total of 1,957 hospitalisation episodes for benign neoplasms of the larynx were recorded among Australian children aged 0–14 years (1,004 episodes in females and 953 in males). The average annual number of hospitalisation episodes declined substantially over time, from 132 episodes per year between 2001 and 2006 to 69 episodes per year between 2007 and 2023, and further to an average of 26 episodes per year over the most recent five years.

Consistent with this reduction in hospitalisation burden, hospitalisation episode rates per 100,000 children aged 0–14 years declined. Between 2001 and 2023, annual hospitalisation episode rates ranged from 0.2 to 4.3 per 100,000 children in males and from 0.7 to 3.5 per 100,000 children in females. Overall hospitalisation episode rates (females and males combined) decreased from a peak of 3.9 per 100,000 children in 2002 to 0.5 per 100,000 children in 2023 (Figure 3.3, Table 3.6).



Indicator: Hospitalisations for benign neoplasm of the larynx (coded using ICD code D14.1) by Aboriginal and Torres Strait Islander status and sex over time

Status of indicator

Data on hospitalisations for benign neoplasms of the larynx (ICD-10-AM code D14.1) among Aboriginal and Torres Strait Islander children aged 0–14 years were available for the period 2011 to 2021. Over these 11 years, hospitalisation episode rates were low in both males and females, with some year-to-year fluctuation due to small numbers.

A total of 25 hospitalisation episodes were recorded among male children and 17 among female children. Annual hospitalisation episode rates ranged from 0.0 to 5.1 per 100,000 children per year in males and from 0.0 to 3.5 per 100,000 children per year in females (**Table 3.6**).

Table 3.6 Hospitalisation rates for benign neoplasm of the larynx per 100,000 persons, in children aged 0–14 years, 2001–2023 (Nationally) and 2011–2023 (Aboriginal and Torres Strait Islander Children)

	Nationally		Aboriginal and Torres Strait Islander Children	
	Females	Males	Females	Males
Nationally				
2001	3.0 (58)	3.6 (73)	-	-
2002	3.5 (68)	4.3 (88)	-	-
2003	3.1 (60)	3.2 (66)	-	-
2004	3.2 (62)	4.3 (88)	-	-
2005	2.9 (57)	3.1 (63)	-	-
2006	2.8 (55)	2.5 (51)	-	-
2007	2.3 (46)	2.2 (46)	-	-
2008	2.7 (53)	2.9 (61)	-	-
2009	2.9 (58)	3.4 (73)	-	-
2010	2.6 (53)	2.1 (46)	-	-
2011	2.7 (56)	2.1 (45)	0.0 (0)	3.5 (5)
2012	2.8 (59)	2.5 (56)	4.6 (7)	1.4 (1–4)**
2013	2.4 (52)	2.8 (64)	5.1 (8)	2.0 (1–4)
2014	1.9 (41)	1.7 (38)	0.6 (1–4)	1.3 (1–4)
2015	1.5 (32)	1.1 (25)	0.0 (0)	1.3 (1–4)
2016	1.8 (39)	0.7 (17)	0.6 (1–4)	0.0 (0)
2017	1.7 (39)	0.4 (9)	0.0 (0)	0.0 (0)
2018	0.8 (19)	0.4 (9)	1.2 (1–4)	0.0 (0)
2019	0.7 (16)	0.5 (11)	2.4 (1–4)	0.0 (0)
2020	0.8 (18)	0.2 (6)	1.2 (1–4)	0.6 (1–4)
2021	1.2 (27)	0.2 (5)	0.0 (0)	1.3 (1–4)
2022	0.8 (19)	0.3 (8)	Not available	Not available
2023	0.7 (17)	0.2 (5)	Not available	Not available

Source: National Hospital Morbidity Database (2) (see [methodology](#)). Counts fewer than 5 (excluding 0) are shown as "1–4". Crude rates are shown. Hospitalisation rates are expressed as the number of hospitalisation episodes per 100,000 children aged 0–14 years; individual children may contribute more than one episode.

Data gaps and limitations

- Surveillance of JoRRP has been in place since late 2011, meaning we do not have pre-vaccination data on the incidence of JoRRP (other than that estimated using hospitalisation data)⁴⁸.
- Lack of availability of HPV genotyping data.
- Hospitalisation episodes do not refer to individual children but to hospitalisation events, which may occur multiple times per year per patient with recurrent respiratory papillomatosis. An audit of Australian cases in 2000–2009 found that the median hospitalisation rate per year for cases was 3.69⁴⁸. Hospitalisation rates need to be interpreted with care to avoid overestimating the number of children impacted.
- Changes in the effectiveness of treatments over time, and the patterns of care impacting whether hospitalisation is required to deliver treatment, will also impact the rate of hospitalisations with the disease over time.
- There is no specific International Classification of Diseases (ICD) code for either JoRRP or AoRRP. The ICD code 'D14.1 Benign neoplasm of the larynx' has been validated for use in children in Australia (medical record review found it was predominantly used to code recurrent respiratory papillomatosis with a positive predictive value of 98.1% in the period 2000–2009)⁴⁸. Validation of the code in adults (who have a broader range of benign laryngeal diseases) has not yet been published. Disease coding practices and their consistency in application across geographical regions and over time may also vary.
- There is currently no method of surveillance of AoRRP in Australia.

Data interpretation

Both APSU notification data and hospitalisation data for the ICD code D14.1 'Benign neoplasm of the larynx' suggest that JoRRP has significantly declined in Australia since the introduction of HPV vaccination in 2007. Following the catch-up vaccination program (2007–2009), in which more than half of all females aged 12–26 years were vaccinated, HPV infection rates and diagnoses of genital warts fell rapidly among young females, driven by both direct protection and substantial herd protection. This will have reduced the risk of vertical transmission of HPV 6 and 11 to infants born to these young females, presenting as a reduction in incident cases in young children in the subsequent years. Whilst Australia was the first country

to document such a reduction in incidence⁴⁵, a similar reduction in JoRRP following HPV vaccination has since been demonstrated in the USA⁵¹.

Not all countries use HPV vaccines that protect against HPV 6 and 11, and it is notable that recent cases detected by JoRRP surveillance in Australia are in children born overseas (in 2022 and 2023) or to unvaccinated overseas-born mothers (2 cases in 2021)^{52–54}.

Whilst surgical debulking remains the mainstay of recurrent respiratory papillomatosis treatment, recent developments include the use of Bevacizumab, a recombinant monoclonal antibody treatment that blocks angiogenesis (new vascular growth), resulting in tumour shrinkage⁵⁵. Additionally, HPV vaccination is increasingly given as an adjuvant treatment. Meta-analyses of case series and cohort studies suggest a possible reduction in recurrence rates after HPV vaccination, but there is no randomised trial evidence of its effectiveness (due in part to the difficulty of conducting a trial for such a rare disease)^{56,57}. It is unclear if either of these treatment developments is reducing the rates of severe disease and hospitalisation amongst patients.

There is, as yet, no evidence that HPV vaccination prevents AoRRP. This is likely because it is still too early to observe any effect, given the average age at diagnosis (36 years in a recent UK study⁵⁸). If medical record auditing indicates that ICD code D14.1 has a reasonable positive predictive value for the disease, as it does in JoRRP, it could be used to monitor hospitalisation rates over time. Alternatively, a recurrent respiratory papillomatosis register, such as has been recently established in the UK⁵⁹, or a surveillance system based upon notification of incident cases by a network of treating clinicians as is done for JoRRP, could be established.

For additional information, also refer to the [Australian Paediatric Surveillance Unit Annual Reports](#)

3.3 Monitor the prevalence of screen-detected cervical abnormalities

Justification for this monitoring objective

High-grade cervical abnormalities (HGAs; also known as high-grade squamous intraepithelial lesions, cervical intraepithelial neoplasia grades 2 and 3 [CIN2/CIN3] and inclusive of adenocarcinoma in situ [AIS]) are due to persistent HPV infection, with the most severe lesions (CIN3/AIS) well-established precursors to cervical cancer. HGAs were the primary endpoint in the efficacy trials for HPV vaccine licensure⁶⁰. Tracking changes in the prevalence of HGAs diagnosed on histopathology serves as a crucial indicator for assessing the impact of HPV vaccination. The majority are caused by infection with oncogenic HPV types 16 and 18, which can be prevented

through vaccination⁶¹. Since high-grade abnormalities can be detected in the years following infection in most cases, they allow for earlier assessment of vaccine effects compared to cervical cancer, which, on average, takes at least a decade to develop⁶². A decline in the rates of high-grade abnormalities at the population level suggests reduced HPV transmission, increased herd protection, and lower healthcare costs from fewer diagnostic and treatment procedures¹⁶.

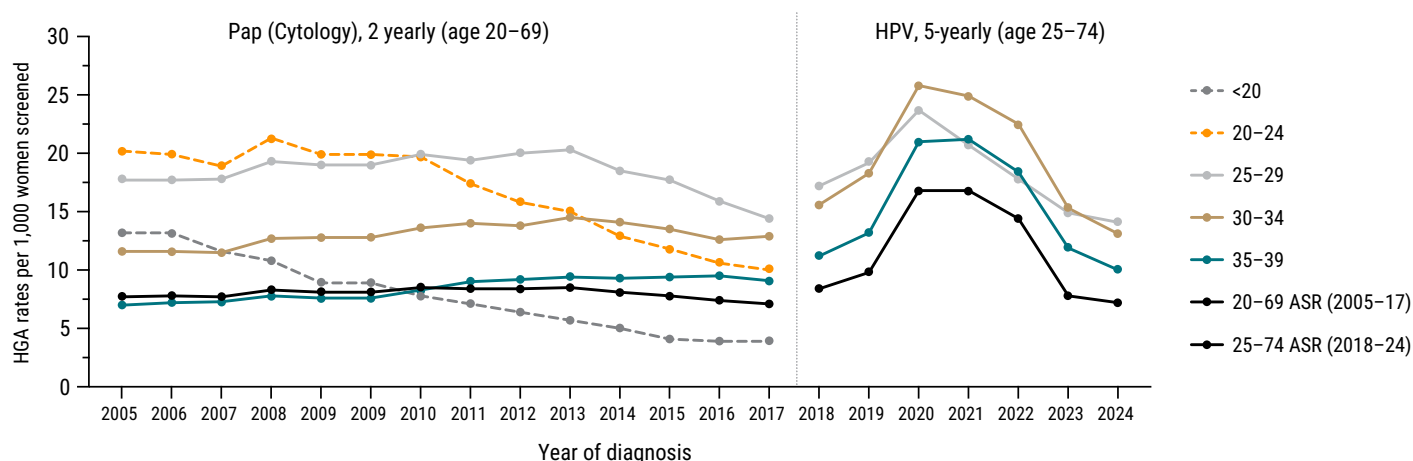
In addition to monitoring the impact of HPV vaccination, tracking rates of screen-detected HGAs over time also provides insight into the effects of transitioning from cytology to HPV-based screening, highlighting how changes in the test method, age of initiation, and screening interval influence the detection of this vaccine-preventable disease.



Indicator: Number of participants aged 25–74 years with a high-grade cervical abnormality detected on histology in a calendar year per 1,000 participants screened

Status of indicator

Figure 3.4 Histologically confirmed high-grade cervical abnormality (HGA) detection rates per 1,000 participants screened nationally, by age: historical (2005–2017) and current (2018–2024)



Source: [2025 Cervical Cancer Elimination Progress Report](#) (Analysis of National Cancer Screening Register data, see [methodology](#)). ASR: Age-standardised rate. *Note: There was likely under-notification of these data to the NCSR in 2018 and 2019.

3. MONITORING NON-CANCER ENDPOINTS

In 2024, the national histologically confirmed HGA detection rate was 7.2 per 1,000. The highest detection rates were observed in participants aged 25–29 and 30–34 years, at 14.1 and 13.1 per 1,000, respectively. Detection rates declined with increasing age, from 10.0 per 1,000 in the 35–39 age group to 1.8 per 1,000 in the 70–74 age group. These 2024 rates follow a period of elevated HGA detection in 2020 and 2021, when rates peaked across all age groups (**Figure 3.4; Table 3.7**).

Historically, between 2005 and 2017, during the 2-yearly cytology-based cervical screening program, HGA detection rates showed clear age-specific trends. Among those under 20 years, detection rates dropped significantly over the period, reflecting early effects of the HPV vaccination program. At the start of this period, participants aged 20–24 years had the highest detection rates. After

the 2007–2009 HPV vaccination catch-up campaign, rates in this group decreased, and from 2010 onwards, participants aged 25–29 years had the highest detection rates per 1,000 participants screened. Although rates among the 25–29 age group remained the highest, they declined slightly over time. Conversely, detection rates for those aged 30–34 and 35–39 years stayed relatively stable throughout. Overall, the national age-standardised detection rate held steady at about 7–9 cases per 1,000 (**Figure 3.4; Table A3.1**).

The transition to HPV-based screening in 2017 has led to a shift in peak detection rates from participants aged 25–29 years to those aged 30–34 years, likely reflecting cohort effects from widespread HPV vaccination, as vaccinated cohorts became age-eligible for screening (**Figure 3.4; Table 3.7**).

Table 3.7 Histologically confirmed high-grade cervical abnormality (HGA) detection rates, per 1,000 participants aged 25–74 screened nationally, 2018–2024

	2018	2019	2020	2021	2022	2023	2024
Nationally (Crude rate)	8.4	9.8	16.8	16.8	14.4	7.8	7.2
Age group							
25–29	17.2	19.2	23.7	20.7	17.8	14.9	14.1
30–34	15.6	18.3	25.8	24.9	22.5	15.3	13.1
35–39	11.2	13.2	21.0	21.2	18.4	11.9	10.0
40–44	8.7	10.0	17.1	18.9	15.8	9.0	8.1
45–49	5.9	6.9	11.9	12.7	11.9	6.3	6.2
50–54	3.3	4.3	8.6	8.9	7.8	4.0	4.2
55–59	2.4	3.0	6.8	8.1	7.2	2.8	3.1
60–64	2.0	2.8	6.8	8.0	6.2	2.3	2.6
65–69	1.4	2.2	5.9	6.8	5.1	1.9	1.9
70–74	1.9	2.1	5.0	4.8	4.8	1.7	1.8

Source: 2025 Cervical Cancer Elimination Progress Report (Analysis of National Cancer Screening Register data, see [methodology](#)). *Note: There was likely under-notification of these data to the NCSR in 2018 and 2019.



Indicator: Number of Aboriginal and Torres Strait Islander participants aged 25–74 years with a high-grade cervical abnormality detected on histology in a calendar year per 1,000 Aboriginal and Torres Strait Islander participants screened

Status of indicator

Table 3.8 Histologically confirmed high-grade cervical abnormality (HGA) detection rates, per 1,000 Aboriginal and Torres Strait Islander participants screened aged 25–74, 2018–2024

	2018	2019	2020	2021	2022	2023	2024
Overall (Crude rate)	13.2	14.6	22.7	24.4	20.2	13.5	13.4
Age group							
25–29	21.7	22.8	29.1	26.5	22.4	16.9	18.4
30–34	21.0	23.2	30.8	34.9	32.7	22.9	19.2
35–39	11.7	16.2	30.3	34.0	24.7	16.5	16.2
40–44	13.6	12.4	18.5	28.0	19.9	15.1	15.3
45–49	6.5	8.4	15.2	16.9	12.7	11.4	10.8
50–54	5.0	6.6	8.5	11.3	9.3	6.9	8.1
55–59	6.1	6.2	11.0	11.2	7.9	4.3	5.4
60–64	5.2	4.5	9.9	7.2	11.5	4.3	4.0

Source: 2025 Cervical Cancer Elimination Progress Report (Analysis of National Cancer Screening Register data, see [methodology](#)). *Note: There was likely under-notification of these data to the NCSR in 2018 and 2019. Case numbers were too small to publish for 65–69 and 70–74 age groups.

In 2024, the histologically confirmed HGA detection rate among participants identified by the NCSR as Aboriginal and/or Torres Strait Islander was 13.4 per 1,000 participants screened aged 25–74 years. The highest detection rate was observed in participants aged 30–34 years, at 19.2 per 1,000, while the lowest rate was in participants aged 60–64 years, at 4.0 per 1,000 participants screened. Overall detection rates among Aboriginal and Torres Strait Islander participants followed trends similar to national patterns, with peaks in 2020 and 2021 across most age groups, followed by declines through 2023 and 2024 ([Table 3.8](#)).

Data gaps and limitations

- Aboriginal or Torres Strait Islander identification was determined by whether a person had ever self-reported their status, as recorded in the NCSR. Indigenous status data in the NCSR are incomplete. Therefore, data reflect rates among participants who have ever identified as Aboriginal and/or Torres Strait Islander, rather than rates for all Aboriginal and Torres Strait Islander participants.
- The NCSR is not linked to the AIR, preventing the reporting of the prevalence of screen-detected cervical abnormalities by vaccination status.
- Histopathology data are known to be under-reported to the NCSR for 2018 and 2019, resulting in likely underestimation of rates.

- HGA rates from 2018 onwards are based on HPV screening and cannot be directly compared with the earlier Pap-based program, which differed in test method, interval, and target age group.

Data interpretation

Rates of HGA detection through screening are dependent upon many factors, including the risk profile of those attending screening (i.e. true underlying disease rate), the sensitivity of the test (how likely is it to find disease that is present), and the diagnostic accuracy of determining disease status in those who are screen positive (i.e. colposcopy and biopsy). These factors were relatively stable in the previous cervical cytology-based program, which offered screening every two years to those aged 20–69 years. Following the introduction of the quadrivalent HPV vaccine in 2007, a decline in the rate of high-grade abnormality was detected relatively rapidly among young cervical screening program participants and was confirmed to be due to vaccination by vaccine effectiveness estimates from linked vaccination and screening data^{63,64}. By 2017, the final year of the cytology-based screening program, rates of high-grade abnormality had declined by 70% among participants under 20, 50% among those aged 20–24, and 19% among those aged 25–29. Notably, the age group with the highest rate of high-grade abnormality had shifted from 20–24-year-olds

to 25–29-year-olds, as women vaccinated at younger ages (when vaccination is most effective) began to participate in screening⁶¹.

The transition to HPV-based screening introduced a discontinuity in the ability to use epidemiological data to monitor the impact of the HPV vaccine. This is due to several factors: the greater sensitivity of HPV testing, which detects more high-grade abnormalities than cytology; the shift in the screening age range, with people under 25 no longer routinely screened; and changes in the risk profile of screening participants resulting from the transition from 2-yearly to 5-yearly intervals. In the first two years of the new program (2018–2019), most participants were regular screeners (with a likely compounding effect of under-notification of histopathology to the new register in this period)⁷⁶. However, from 2020 to 2022, the majority consisted of overdue or never-screened women, except for those entering at age 25, resulting in a higher-risk screened population. This shift is reflected in both increased HPV prevalence (see Monitoring infection section) and elevated high-grade abnormality detection rates. It will likely take time for participant risk levels to stabilise across calendar years.

Under-identification of Aboriginal and Torres Strait Islander status in the NCSP remains a key challenge. Completion depends on respectful inquiry, willingness to disclose, accurate recording in Medicare or pathology forms, and proper data transmission – any breakdown in this process can affect the completeness and accuracy of the data. As a result, reported rates of high-grade abnormality detection reflect only those who identified as Aboriginal and/or Torres Strait Islander, and may not represent the broader Indigenous population if non-identifiers differ demographically or by other risk factors.

Available data indicate higher rates of disease detection among Aboriginal and Torres Strait Islander participants compared to national estimates. However, these comparisons should be interpreted cautiously, as key risk factors such as smoking, a known co-factor in cervical carcinogenesis⁶⁵, which is more prevalent in some Aboriginal and Torres Strait Islander communities due to complex social and historical determinants and lasting impacts of colonisation⁶⁶, have not been accounted for. Also, age-stratified rates are provided where possible; however, they may not fully account for the strong age-related influence on infection and disease rates. The Aboriginal and Torres Strait Islander population has a notably younger age structure (median age of 24.0 years in 2021) than that of the overall population (median age of 38.4 years in 2021)⁶⁷. Other contributing factors include

potential differences in participation rates in cervical screening. These findings align with both previous sub-national analyses and the higher cervical cancer burden among Aboriginal and Torres Strait Islander women. They highlight the potential benefit of both HPV vaccination and cervical screening in preventing disease, and the need to achieve high coverage of both measures to reduce disparities. Expanding vaccine eligibility to 25 years of age may further improve access for Indigenous communities that may have missed earlier doses. The availability of cervical screening self collection also offers a more accessible screening option for many Aboriginal and Torres Strait Islander participants. However, culturally safe and accessible care must be ensured across the entire pathway, including timely and respectful follow-up, particularly through public and private colposcopy services. Complete and accurate reporting of Indigenous status is also required.

For additional information, also refer to the [2025 Cervical Cancer Elimination Progress Report](#) and the [AIHW National Cervical Screening Program monitoring report 2025](#).

3.4 Monitor the distribution of HPV genotypes detected in high-grade cervical lesions

Justification for this monitoring objective

Monitoring the distribution of HPV genotypes in high-grade cervical lesions is essential for evaluating and refining cervical cancer prevention strategies. Genotype-specific data help assess the impact of current HPV vaccines

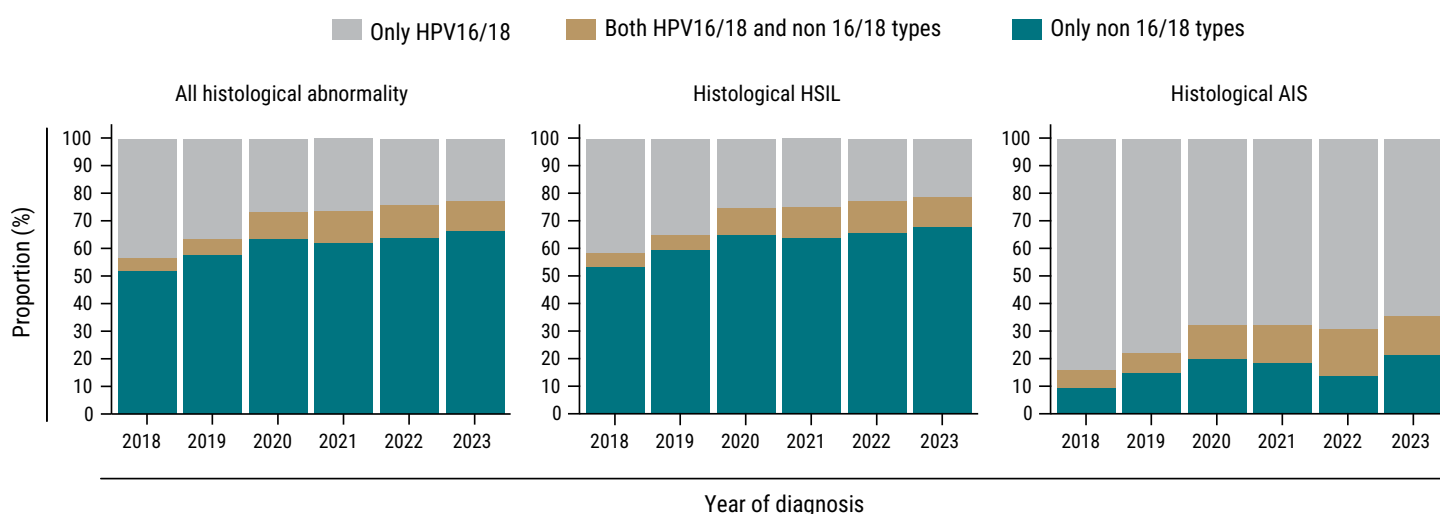
on disease burden—particularly for vaccine-covered types such as HPV16 and HPV18—and reveal the types responsible for the remaining burden of disease. With the introduction of the nonavalent HPV vaccine, this monitoring also enables evaluation of how vaccination and screening interact as vaccinated cohorts enter the screening program. Tracking changes in genotype prevalence over time can inform updates to screening algorithms, ensuring they remain effective for both vaccine and non-vaccine types.



Indicator: Prevalence of HPV by type in participants where a high-grade abnormality is detected on histology

Status of indicator

Figure 3.5 Prevalence (%) of HPV by type in participants of all ages with a high-grade histological abnormality, overall and by histology type, 2018–2023



Source: National Cancer Screening Register (see [methodology](#)). HSIL: high-grade squamous intraepithelial lesions; AIS: adenocarcinoma in situ.

From 2018 to 2023, the proportion of people with high-grade histological abnormalities who had a routine clinical HPV test result recorded over the five years preceding the high-grade histology result increased from 87.9% to 98.7%, with unknown status dropping from 12.1% to 1.3% (**Appendix Table A3.2**). Among those tested, the HPV detection rate ranged from 97.5% to 98.2% year by year. The proportion with only HPV16/18 declined steadily from 43.4% to 22.6%, while mixed infections with HPV16/18 and other oncogenic types more than doubled from 5.1% to 11.4%. Other oncogenic types alone rose steadily from 51.5% to 66.0% (**Figure 3.5; Appendix Table A3.2**).

For HSIL (high-grade squamous intraepithelial lesions), the detection rate for HPV16/18 alone decreased from 41.7% to 21.0%. In comparison, the detection of only other oncogenic types rose from 53.3% to 67.6%, and mixed infections increased from 5.0% to 11.3%. In AIS, the proportion with only HPV16/18 dominated over time.

However, the proportion with only 16 or 18 detected fell from 83.9% to 64.4%, while other oncogenic types and mixed infections increased from 9.3% to 21.2% and 6.8% to 14.4%, respectively (**Figure 3.5; Appendix Table A3.2**). Similar trends were observed for screening age-eligible individuals aged 25–74 (**Appendix Table A3.2**).

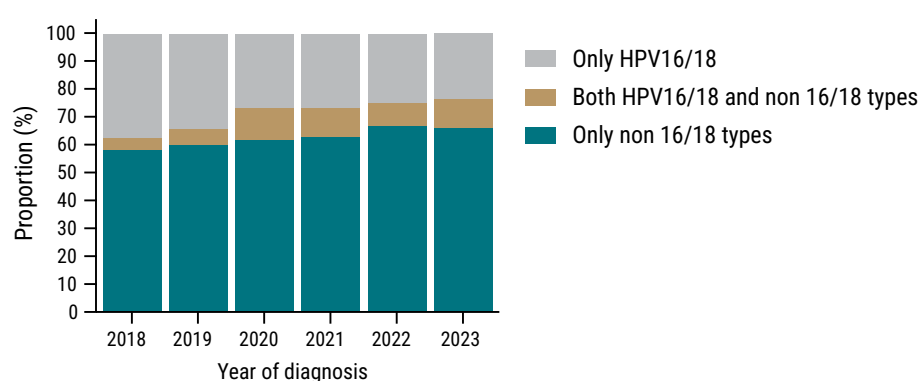
By age group, the most notable decline over time in detection of HPV16/18-only infections occurred in people under 40 years: from 28.3% to 10.2% in those aged 25–29 years, 48.4% to 18.1% in 30–34 years, and 54.0% to 26.3% in 35–39 years. Other oncogenic types increased in all age groups, from high levels in younger people (i.e. 67.5% to 81.6% in those aged 25–39 years) to moderate increases in the oldest group (38.7% to 47.0%). Mixed infections increased across most age groups, rising most notably in those aged 50–64 years and showing the smallest changes in those under 35 years (**Appendix Table A3.3**).



Indicator: Prevalence of HPV by type among Aboriginal and Torres Strait Islander participants where a high-grade abnormality is detected on histology

Status of indicator

Figure 3.6 Prevalence (%) of HPV by type among Aboriginal and Torres Strait Islander peoples of all ages with a high-grade histological abnormality, 2018–2023



Source: National Cancer Screening Register (see [methodology](#)).

From 2018 to 2023, the proportion of Aboriginal and Torres Strait Islander peoples of all ages with high-grade histological abnormalities who had a routine clinical HPV test result recorded over the 5 years preceding the high-grade histology result increased from 86.9% to 99.6%, while the unknown status dropped from 13.1% to 0.4% (**Appendix Table A3.4**). Among those with known status, HPV detection rates ranged from 96.7% to 98.9% year by year. The proportion with only HPV16/18 decreased steadily from 37.5% to 23.6%, while those with only other oncogenic types rose from 58.3% to 65.9%. Mixed infections with both HPV16/18 and other oncogenic types increased from 4.2% in 2018 to a peak of 11.5% in 2020, and remained elevated at 10.5% in 2023 (**Figure 3.6; Appendix Table A3.4**). Similar trends were observed for all ages, as well as for those aged 25–74 (**Appendix Table A3.4**).

By age group, younger people (25–34 years) experienced a marked decline in HPV16/18-only infections between 2018 and 2023, from 31.0% to 12.4%, accompanied by an increase in infections with only other oncogenic types, from 64.8% to 79.2%. Middle-aged groups (35–44 and 45–54 years) showed generally decreasing HPV16/18-only proportions over this time period, with rising or stable prevalence of other types and mixed infections. Older age groups (55–64 and 65–74 years) exhibited more variable patterns; for instance, the proportion of the 55–64 age group with HPV16/18-only infections declined from 57.6% in 2018 to 27.6% in 2021, then rose again, reaching 48.0% in 2023, while other oncogenic types declined. Mixed infections increased notably (**Appendix Table A3.4**).

Data gaps and limitations

- Small case numbers in some age groups make results more variable and less reliable for trend analysis.
- Possible under-ascertainment of non-16 and 18 types in participants who also have HPV16/18 detected due to clinical reporting rules, which could bias the observed prevalence of mixed versus single-type infections with HPV16/18 (but would not affect results for only non-16 and 18 types).
- Vaccination status is not linked to screening records, limiting the ability to stratify distribution by vaccination history.
- Routine program clinical HPV data cannot attribute high-grade lesions to specific HPV types. Standard tests report the presence of HPV in the collected sample (cervical or vaginal, self- or clinician-collected), which may contain multiple types. Still, the causal type for a particular lesion cannot be determined without specialised methods such as lesion tissue genotyping.

Data interpretation

Declines in HPV16/18 over time were most pronounced in younger age groups, with similar shifts now evident in vaccinated cohorts aged 40–44 years as they transition into older screening bands. These changes reflect the gradual replacement of unvaccinated cohorts who were offered vaccination, albeit in some age groups, which represent those vaccinated at older ages and with lower coverage in the early years of the vaccination program. Pre-vaccine data provide helpful context for interpreting these patterns. In a 2012 study, 66% of HPV positive HSIL in women aged 26–35 years were attributed to HPV16/18⁶⁸. In contrast, current 2023 data show HPV16/18-only infections in just 10% of HSIL cases among people aged 25–29 years and 18% among those aged 30–34 years. Including both HPV16/18-only and mixed infection groups, the prevalence increases modestly to 18% and 27%, respectively. While the estimates are not directly comparable due to differences in study design, testing methods, and data granularity, the magnitude of this reduction is notable and broadly consistent with a substantial vaccine effect.

Beyond its role in protecting against HPV through vaccination, age also affects the distribution of HPV types: before vaccination, younger populations were more likely to have HPV16-associated HSIL, while older women more commonly had lesions linked with other oncogenic types⁶⁸. Since vaccination, these natural age-related differences have become less marked.

Other factors may also contribute to observed patterns. Some year-to-year variations also reflect program changes and the timing of lesion detection, as clinical management guidelines differ for HPV16/18 vs. non-16 and 18 types (i.e., if initial cytology is negative, it will take longer to diagnose non-16 and 18 lesions). The lower HPV detection rate in 2018 may be related to the transition to the NCSP and to the higher-grade detection arising from follow-up of cytology-detected abnormalities from the pre-renewal program, consistent with the lower proportion of available HPV results that year. The timing of lesion detection differs for HPV16/18 (as individuals with these test results are referred after the first test in which this is detected, starting in 2018), and for non-16 and 18 (as they may be referred either at their initial test or a follow-up test, depending on cytology results).

3. MONITORING NON-CANCER ENDPOINTS

An analysis of histological outcomes from the first two years of the HPV screening program in Australia found that approximately one-third of high-grade lesions detected due to non-16 and 18 types (only) are in those referred during follow-up, having been classified as intermediate risk at their baseline screening test⁶⁹.

This effect for non-16 and 18 types further differs by age: women aged 70–74 years are referred after their first test, where non-16 and 18 types are detected (because they are referred for colposcopy if HPV is detected, regardless of type). Those aged 50–69 years with only non-16 and 18 HPV types may be referred at either the initial test or the 12-month follow-up, depending on their initial cytology result. This pattern also applied to people younger than 50 years in 2018 and 2019. NCSP Guidelines were updated in late 2020 to recommend another 12-month follow-up in the absence of high-grade or glandular cytological abnormalities, thereby shifting detection of some non-16 and 18 lesions in this age group from around 12 months after their initial screen to around 24 months after, temporarily lowering the proportion of “only other oncogenic” HPV types⁷⁰.

When interpreting the relative proportion of type grouping causing disease over time, it is essential to note that rising proportions of non-16 and 18 disease do not necessarily indicate an increase in the number of people with these lesions. Absolute numbers of high-grade lesions diagnosed over time (see Indicator 3.3) will reflect many factors, including the sensitivity of the screening test, characteristics of people attending screening over time, the effectiveness and timeliness of follow-up and referral mechanisms, the biopsy rate, and the completeness of histopathology reporting to the register. As 16/18 disease burden declines as a relative cause of high-grade disease due to vaccination, the proportion of remaining disease caused by non-16/18 types will rise. It will be important for future monitoring of this indicator that further differentiation of the non-16 and 18 group into component HPV types is available, as cohorts vaccinated using the nonavalent HPV vaccine, which will reduce some of the non-16 and 18 burden (types 31, 33, 45, 52, 58), but not all, reach screening age (from 2030).

4.

MONITORING CANCER ENDPOINTS

4. MONITORING CANCER ENDPOINTS

4.1 Monitor cervical cancer incidence and mortality

Justification for this monitoring objective

HPV vaccines were developed primarily to prevent cervical cancer by targeting the HPV types most commonly responsible for the disease. In Australia, HPV types 16 and 18 cause approximately 75% of cervical cancers, while the additional 5 oncogenic types covered by the nonavalent HPV vaccine (types 31, 33, 45, 52, 58) bring vaccine-related protection up to about 90% of cases. Substantial reductions in circulating HPV infection and cervical lesions have already been observed (See report sections 2 and 3). However, significant declines in cervical cancer incidence and mortality will take longer to achieve due to the long

natural history of the disease, typically more than a decade from infection to invasive cancer, and the lag between vaccination at ages 12–13 and peak cancer diagnosis at ages 30–49^{71,72}.

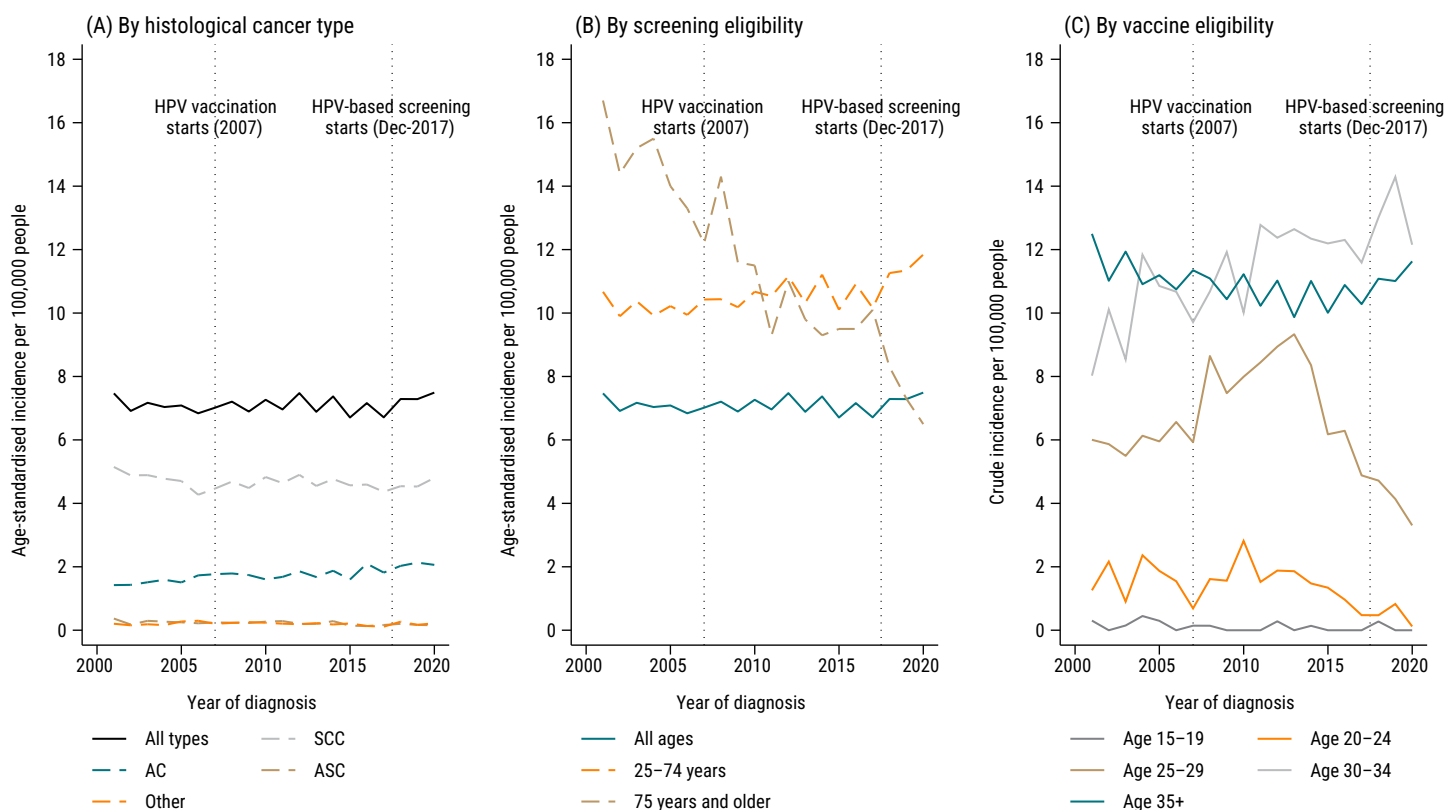
Therefore, ongoing monitoring of cervical cancer incidence and mortality is essential to accurately assess vaccine impact, track progress toward elimination goals, and guide future prevention strategies. Note that incidence and mortality rates in this report are age-standardised to the 2001 Australian population. This is consistent with AIHW cancer incidence monitoring reports, but differs from the method used in the annual cervical cancer elimination report, which standardises to the world population to facilitate international comparisons.



Indicator: Number of new cases of cervical cancer by type of cancer and vaccination status in females aged 25–74 years per 100,000 estimated resident population in a calendar year

Status of indicator

Figure 4.1 Incidence rates of cervical cancer in females nationally, by a) histologic type; b) broad age group (screen-eligibility) and c) broad age group (vaccine-eligibility), per 100,000 estimated resident population, 2001–2020



Source: Australian Cancer Database (see [methodology](#)). Panels A and B: Standardised to the 2001 Australian population. Data were not available by vaccination status. SCC: Squamous cell carcinoma; AC: Adenocarcinoma; ASC: Adenosquamous cell carcinoma; Other: All other cervical cancers.

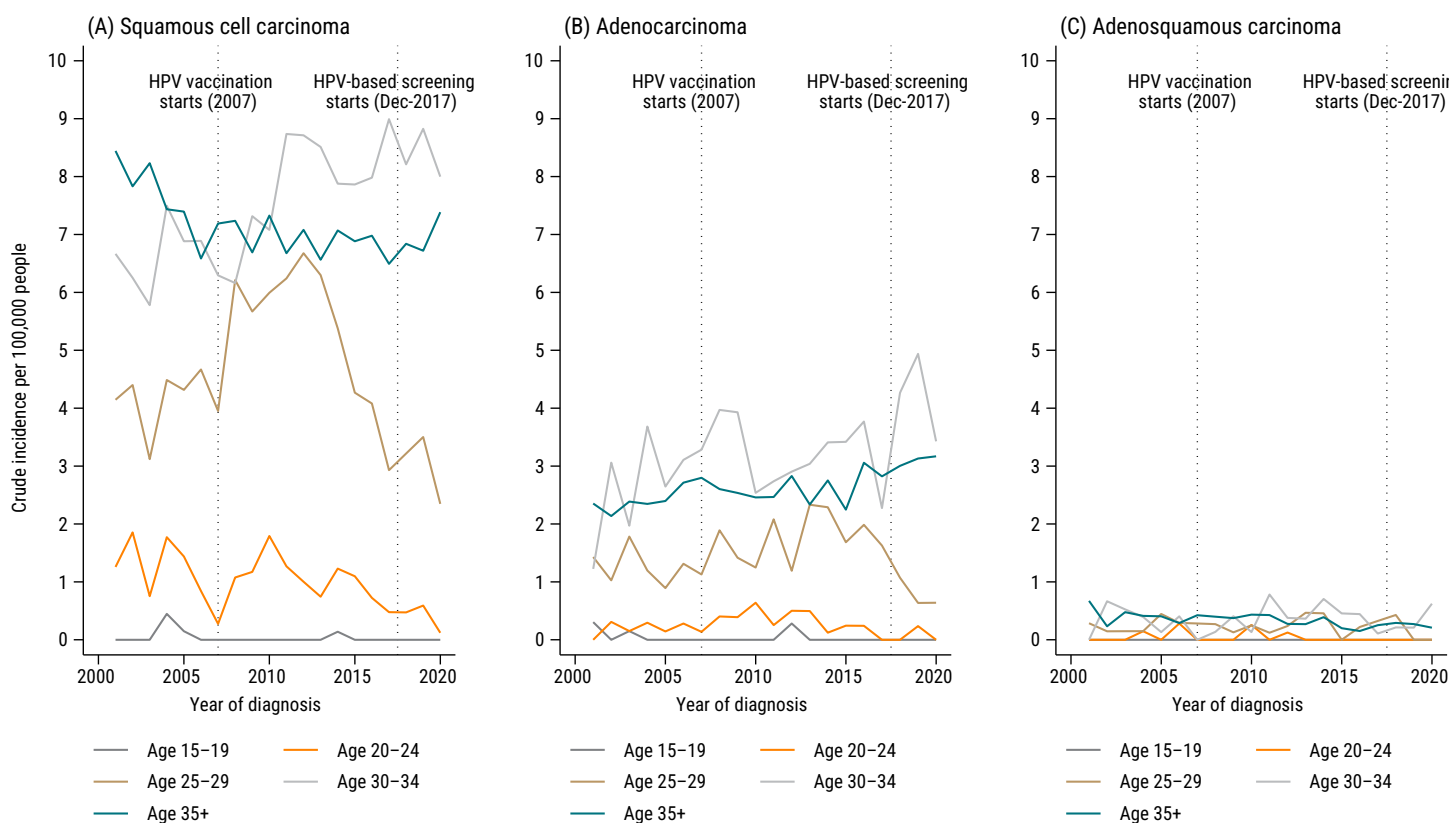
Cervical cancer incidence rates remained relatively stable across 2001–2020 (**Figure 4.1; Table A4.1**). Age-standardised rates per 100,000 females were 7.5 in 2001, 7.2 in 2016—the last full year before HPV-based screening—and 7.5 in 2020. Among females aged 25–74 years, incidence ranged from 9.9 to 11.8 per 100,000. A slight increase was observed following the introduction of HPV screening, particularly in 2018–2020, reflecting the higher sensitivity of the test; this increase was most pronounced in the 25–74-year age range. In contrast, rates steadily declined among those aged ≥ 75 years, from 16.7 to 6.5 per 100,000 (**Figure 4.1B; Table A4.1**).

Age-specific trends were more variable. Prior to HPV-based screening, cervical cancer incidence declined among females aged ≥ 35 years, before modestly increasing from 2018. In younger females, rates rose slightly among those aged 30–34 years, while those aged 25–29 years showed a temporary plateau between 2008 and 2013, followed by a sharp decrease thereafter. Among females aged ≥ 35 years, rates increased modestly from 2018 to 2020. Overall, rates among females aged 20–29 years fell to the lowest levels observed, and rates among those under 20 years remained consistently very low (**Figure 4.1C; Table A4.1**).

Trends by histologic type differed. SCC decreased slightly from 5.1 per 100,000 in 2001 to 4.8 in 2020, adenocarcinoma increased from 1.4 to 2.1 per 100,000, and adenosquamous carcinoma remained very low at 0.2–0.3 per 100,000. Among females aged 25–74 years, SCC ranged from 6.3 to 7.6 per 100,000 and adenocarcinoma from 2.1 to 3.4 per 100,000, indicating that most cases occurred within this age range (**Figure 4.2A; Figure 4.2B; Table A4.1**). Adenosquamous carcinoma remained rare across all age groups (**Figure 4.2C; Table A4.1**).

Age-specific adenocarcinoma trends showed that in females aged 25–34 years, incidence increased gradually until 2016, then declined in the 25–29-year group, while rates in the 30–34-year group rose from 1.2 per 100,000 in 2001 to 3.4 in 2020. Across the 25–74-year age group, adenocarcinoma rates increased from 2.1 per 100,000 in 2001 to 3.3 in 2020. Incidence in females under 20 years was very low, with zero cases in most years (**Figure 4.2B; Table A4.1**).

Figure 4.2 Age-specific incidence rates of cervical cancer in females nationally by histologic type, per 100,000 estimated resident population, 2001–2020



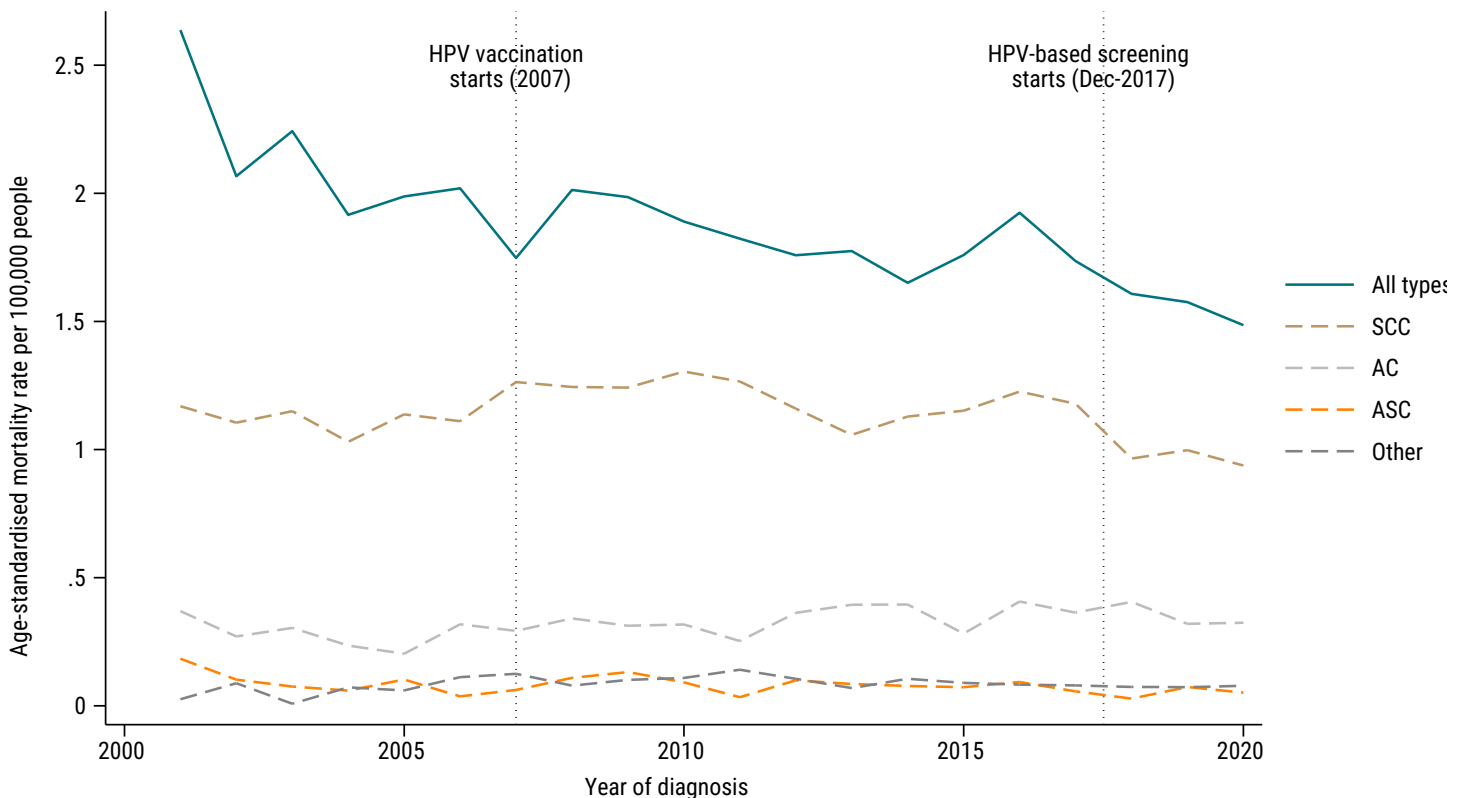
Source: Australian Cancer Database (see [methodology](#)). Data were not available by vaccination status.



Indicator: Number of deaths from cervical cancer by type of cancer and vaccination status in females aged 25–74 years per 100,000 estimated resident population in a calendar year

Status of indicator

Figure 4.3 Age-standardised mortality rates from cervical cancer in females nationally, overall and by histologic type, per 100,000 estimated resident population, 2001–2020



Source: Australian Cancer Database (see [methodology](#)). Standardised to the 2001 Australian population; Data were not available by vaccination status; SCC: Squamous cell carcinoma; AC: Adenocarcinoma; ASC: Adenosquamous cell carcinoma; Other: All other cervical cancers.

4. MONITORING CANCER ENDPOINTS

Cervical cancer mortality rates declined from 2.6 per 100,000 females in 2001 to 1.5 per 100,000 females in 2020 (**Figure 4.3; Table A4.2**). Among females aged 25–74 years, mortality rates declined from 3.2 per 100,000 in 2001 to 2.0 per 100,000 in 2020, reflecting that most deaths occur in this age range (**Table A4.2**).

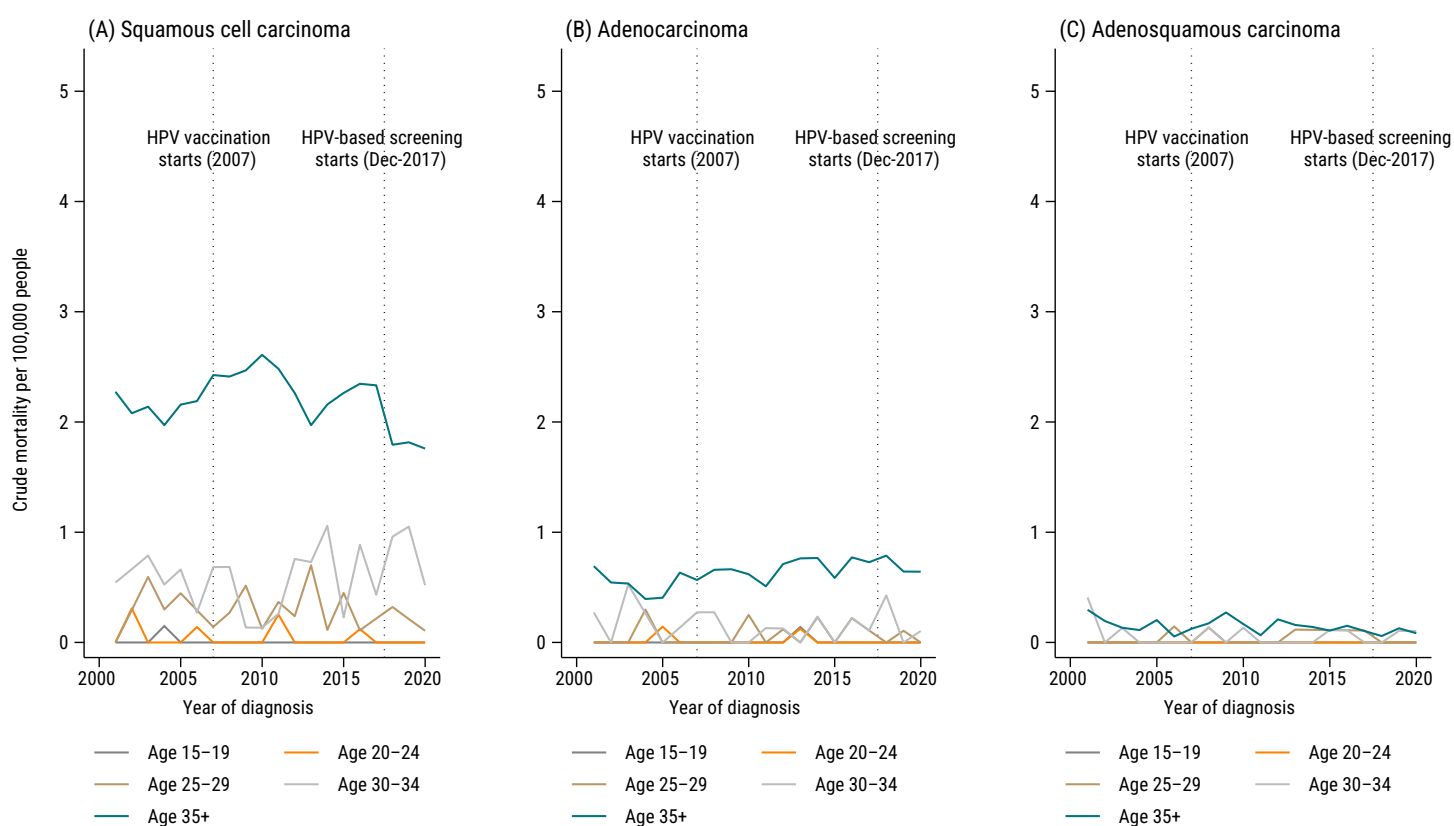
There have been declines in age-standardised cervical cancer mortality per 100,000 females due to SCC (from 1.2 in 2001 to 0.9 in 2020) and adenosquamous carcinoma (from 0.2 to 0.1), but relatively less change in mortality due to adenocarcinoma (fluctuated within the range 0.2 to 0.4 per 100,000 throughout) (**Figure 4.3; Table A4.2**).

In contrast to incidence, which increased somewhat after the introduction of HPV screening, there was no corresponding increase in cervical cancer mortality.

The overall decline in cervical cancer mortality was primarily driven by decreases in SCC among females aged 35 and above (from 2.3 to 1.8 per 100,000), with rates being considerably lower in younger age groups, and subject to year-to-year variation (**Figure 4.4A; Table A4.2**). Overall, deaths due to adenocarcinoma and adenosquamous carcinoma remained low across the period (**Figure 4.4B; Figure 4.4C; Table A4.2**).

Deaths due to cervical cancer were extremely rare in females aged < 25, with 39 cases in total over the period 2001 to 2020. There were 220 cervical cancer deaths among females aged 25–29 years, but 163 were cancers other than SCC and adenocarcinoma (data not shown); these other histological types are less likely to be prevented or detected early through screening.

Figure 4.4 Age-specific mortality rates from cervical cancer in females nationally by histologic type, per 100,000 estimated resident population, 2001–2020



Source: Australian Cancer Database (see [methodology](#)). Data were not available by vaccination status.



Indicator: Number of new cases of cervical cancer by type of cancer and vaccination status in Aboriginal and Torres Strait Islander females aged 25–74 years per 100,000 estimated resident Indigenous population in a calendar year

Status of indicator

Table 4.1 Cervical cancer incidence rates in Aboriginal and Torres Strait Islander females per 100,000 estimated resident Indigenous population, 5-year pooled for 2016–2020

	All cervical cancers	Squamous cell carcinoma	Adenocarcinoma	Adenosquamous carcinoma	Other and unspecified types
All ages	14.0	10.0	2.7	0.1	0.4
<20	0.1	0.0	0.0	0.0	0.1
20–24	0.0	0.0	0.0	0.0	0.0
25–29	5.5	4.3	1.2	0.0	0.0
30–34	23.9	21.7	2.2	0.0	0.0
35–39	15.6	13.1	0.8	0.8	0.0
40–44	21.7	16.5	4.3	0.9	0.0
45–49	25.0	18.3	5.0	0.0	0.0
50–54	30.1	18.5	9.7	0.0	0.0
55–59	18.1	14.7	0.0	0.0	1.1
60–64	18.2	15.2	3.0	0.0	0.0
65+	27.7	14.8	7.4	0.0	2.8

Source: Australian Cancer Database (see [methodology](#)). All ages category standardised to the 2001 Australian population. Data were not available by vaccination status.

Pooled 5-year (2016–2020) cervical cancer incidence rates among Aboriginal and Torres Strait Islander females were 14.0 per 100,000. Rates per 100,000 population for SCC, adenocarcinomas and adenosquamous carcinomas were 10.0, 2.7 and 0.1, respectively. Adenocarcinomas tended to occur at a somewhat older age than SCC (**Table 4.1**).



Indicator: Number of deaths from cervical cancer by type of cancer and vaccination status in Aboriginal and Torres Strait Islander females aged 25–74 years per 100,000 estimated resident Indigenous population in a calendar year

Status of indicator

Table 4.2 Cervical cancer mortality rates in Aboriginal and Torres Strait Islander females per 100,000 estimated resident Indigenous population, 5-year pooled for 2016–2020

	All cervical cancers	Squamous cell carcinoma	Adenocarcinomas	Adenosquamous carcinoma	Other and unspecified types
All ages	5.1	4.2	0.8	0.1	0.1
<20	0.0	0.0	0.0	0.0	0.0
20–24	0.0	0.0	0.0	0.0	0.0
25–29	1.2	0.6	0.6	0.0	0.0
30–34	4.3	4.3	0.0	0.0	0.0
35–39	4.9	4.1	0.8	0.0	0.0
40–44	7.8	6.1	0.9	0.9	0.0
45–49	6.7	6.7	0.0	0.0	0.0
50–54	10.7	9.7	1.0	0.0	0.0
55–59	7.9	6.8	1.1	0.0	0.0
60–64	12.2	9.1	3.0	0.0	0.0
65+	12.9	9.2	2.8	0.0	0.9

Source: Australian Cancer Database (see [methodology](#)). All ages category standardised to the 2001 Australian population. Data were not available by vaccination status.

The pooled 5-year (2016–2020) cervical cancer mortality rate among Aboriginal and Torres Strait Islander females was 5.1 per 100,000 population. Mortality rates per 100,000 population for SCC, adenocarcinomas and adenosquamous carcinomas were 4.2, 0.8 and 0.1, respectively. Mortality tended to increase with increasing age (Table 4.2).

Data gaps and limitations

- Individual-level vaccine status is not yet routinely linked to cancer incidence, mortality and screening data, making it difficult to identify the impact of vaccination. Even if it were, comparison of cancer rates between vaccinated and unvaccinated individuals in the same birth cohort would likely underestimate the effectiveness of vaccination, as strong herd effects against HPV infections, genital warts and high-grade cervical abnormalities have been seen in unvaccinated individuals who were age-eligible for routine vaccination^{17,73,74}.
- Trends in incidence and mortality could not be examined for Aboriginal and Torres Strait Islander females, due to uncertainties about the consistency of the population estimates over time and the need to pool cases over multiple years due to small numbers.
- Aboriginal and Torres Strait Islander rates were restricted to jurisdictions with adequate Indigenous identification in cancer registry data. Consequently, estimates should not be interpreted as representing complete coverage of Australia.
- Mortality data were sourced from the Australian Cancer Database rather than the National Mortality Database (NMD), as the NMD does not allow for stratification of deaths by histology type. Consequently, data could only be reported up to 2020, rather than 2022, the most recent year available in the NMD.
- Interpretation of cervical cancer trends may be affected by the misclassification of tumours that involve multiple sites at diagnosis (including cervix) but originate in other sites, such as the endometrium or ovary⁷⁵. These tumours can present with overlapping symptoms and share similar histological features, but are not related to HPV.

Data interpretation

Cervical cancer incidence in Australia remained generally stable from 2001 to 2020, with a modest rise following the introduction of HPV-based screening, particularly among screening-eligible age groups aged 35 years and older. Reductions among females aged 20–29 years were evident from 2014, which may be attributed to the introduction of the vaccine. A steady decline was observed among females aged 75 and older, likely due to cohorts of females who have been routinely screened during their lifetimes reaching this age group. Incidence remained very low in females under 20 throughout the period.

Data from the [2025 Cervical Cancer Elimination Progress Report](#) report show that in 2021, no cervical cancer cases were diagnosed in Australia in those aged <25 years, down from one case in 2020. While rates in this age group are unstable due to very small numbers, the trend is consistent with the expected early impact of HPV vaccination. Among those aged 30–34 years, a transient increase observed in 2018–2019 (13.1 and 14.4 per 100,000, respectively) declined to 9.7 per 100,000 in 2021, consistent with short-term effects of the transition to HPV-based screening and the maturing impact of vaccination. Rates in females aged ≥35 years have remained stable throughout the HPV screening program period⁷⁶.

In addition to HPV vaccination, cervical cancer incidence and mortality are also strongly related to cervical screening, including program design and participation. Trends since the introduction of vaccination need to be interpreted in light of previous patterns and changes to the screening program that occurred at a similar time to when cohorts vaccinated at the recommended age (12–13 years) began to enter screening. Prior to the introduction of HPV vaccination, there had already been a strong decline in cervical cancer incidence, which began soon after the introduction of an organised NCSP in 1991, but rates plateaued after about the first decade⁷⁷. This decrease was most strongly seen in SCC and adenosquamous carcinomas. The impact of cytology-based screening on the incidence of adenocarcinomas was limited, with incidence rates appearing to increase from around the mid-2000s⁷⁷. In contrast, HPV vaccination is expected to effectively prevent adenocarcinoma, with over 80% of HPV positive cases caused by HPV types 16 and 18^{78,79}, and HPV screening is more effective at preventing and detecting (thus downstaging) underlying adenocarcinoma than cytology-based screening⁸⁰.

In December 2017, the NCSP transitioned from Pap smear test to primary HPV screening, including an increase in the recommended start age, from 20 to 25 years, and in the screening interval, from 2 to 5 years, due to high sensitivity and effectiveness in identifying females who are not at risk and can safely be screened less frequently^{80,81}. These major changes in screening program design were anticipated to have some transient effects on cervical cancer incidence. As the screening test was more sensitive, a transient increase in cervical cancer incidence (but not mortality) was anticipated after the program transitioned, due to improved detection of prevalent cancers⁸². This effect is becoming apparent among those now eligible for screening. The change in start age would potentially shift the detection of some cancers to occur soon after age 25, rather than between the ages of 20–24 years, as seen elsewhere⁸³. Finally, extending the screening interval led to larger numbers of people attending for routine screening in the first 2 to 3 years of the 5-year screening round than in the later years, as anticipated⁸⁴, potentially impacting the timing of diagnosis of screen-detected cancers. Consequently, interpreting trends in cervical cancer incidence and mortality has become more challenging, affecting rates from 2018 onwards. This poses a challenge in monitoring the impact of HPV vaccination on cervical cancers, which will require a longer period of follow-up and datasets linking vaccination, screening, and cancer.

For additional information, also refer to the [2025 Cervical Cancer Elimination Progress Report](#) and the [AIHW National Cervical Screening Program monitoring report 2025](#).

4.2 Monitor anogenital and oropharyngeal cancer incidence and mortality

Justification for this monitoring objective

A significant proportion of anogenital cancers are caused by persistent infection with HPV, including approximately 88% of anal, 78% of vaginal, 25% of vulval, and 50% of penile cancers⁸⁵. HPV16 is the cause of the vast majority (90% of those that are HPV positive) of these cancers, followed by HPV18, with HPV31, 33, 45, 52 or 58 contributing smaller proportions⁸⁵. SCC is the most common type of cancer in the anus⁸⁶, vulva⁸⁷, vagina⁸⁸, and penis⁸⁹.

Whilst HPV-related anogenital cancers other than cervical are rare cancers in the general population, their burden is disproportionately high in certain groups. Because persistent HPV infection is a necessary cause, people with compromised immune function are at greatest risk, as they are less able to clear HPV infection. For anal cancer, there are clearly defined risk groups⁹⁰. People living with HIV (PLHIV) are at a greatly elevated risk due to their immunocompromised state, even in the era of effective antiretroviral therapy (ART). Among this group, men who have sex with men living with HIV are most affected, with over 100-fold higher risk than the general population⁹⁰. Other groups at increased risk include women with a previous history of cervical, vulval or vaginal cancer or pre-cancer, men who have sex with men without HIV, and solid organ transplant recipients⁹⁰. Recent developments to address this burden include the release of anal cancer screening guidelines for PLHIV by the Australasian Society for HIV, Viral Hepatitis and Sexual Health Medicine⁹¹, and the submission of an application to the Medical Services Advisory Committee (MSAC) for Medicare-funded screening and treatment for precancerous anal lesions for all high-risk groups.

For HPV-related vulval, vaginal, and penile cancers, risk factors are less clearly defined. These cancers tend to occur at older ages, with HIV infection and other immunocompromising conditions further elevating risk⁹².

Head and neck cancers, predominantly SCC, can occur at various anatomical sites in the upper aerodigestive tract, including the nasopharynx, oral cavity, oropharynx, hypopharynx, and larynx⁹³. Oropharyngeal SCC (comprising cancers of the tonsils, base of the tongue, soft palate and uvula⁹⁴) are most strongly associated with HPV infection, with around 30% of cases globally caused by HPV infection⁹⁵, and as many as 80% of cases in the USA⁹⁶. Over 80% of oropharyngeal SCC are caused by HPV16.

For HPV-related anogenital and cervical cancers, smoking is a well-established cofactor in disease development. In contrast, oropharyngeal cancer arises through two distinct pathways: one driven by smoking and alcohol, which can act synergistically—and the other driven by HPV infection, most commonly HPV16, which can be acquired through oral sex⁹⁷. In the latter pathway, smoking contributes as a cofactor but is not the main driver of pathogenesis.

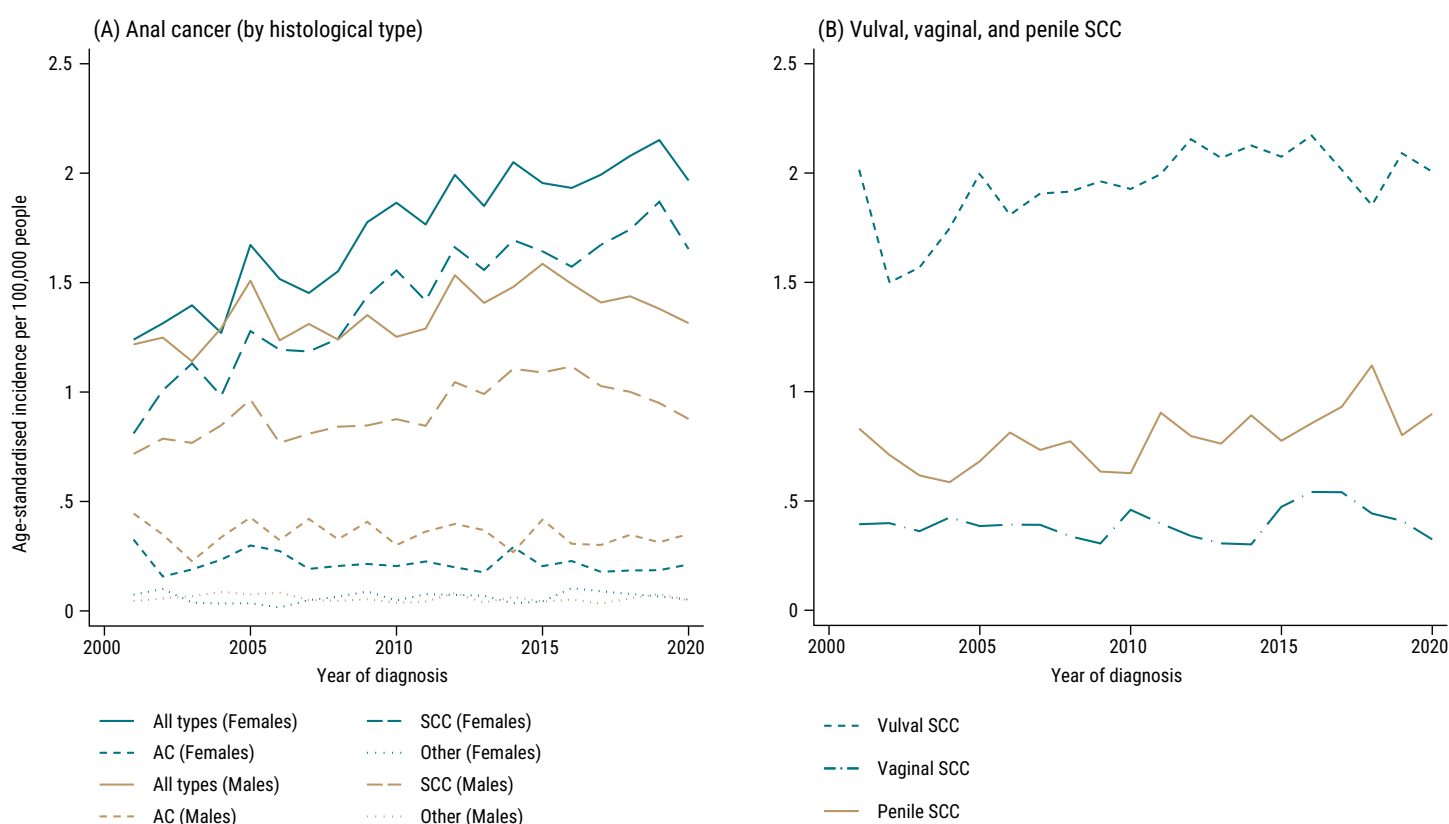
The Australian HPV vaccination program, which targets oncogenic HPV types 16, 18, 31, 33, 45, 52, and 58, was primarily designed to prevent cervical cancer. However, the vaccine also protects against a substantial proportion of other HPV-related cancers, including anogenital and oropharyngeal cancers. It is offered to both females and males to reduce the overall burden of HPV-associated disease, including cancers affecting men. Monitoring the program's impact on these outcomes is critical to a comprehensive assessment of its population-level benefits.



Indicator: Number of new cases of anogenital and oropharyngeal cancer, by age, type, vaccination status and sex per 100,000 estimated resident population in a calendar year

Status of indicator

Figure 4.5 Age-standardised incidence rates of anal cancer by histologic type (A) and vulval, vaginal and penile squamous cell carcinoma (SCC) (B) in females and males, per 100,000 estimated resident population, 2001–2020



Source: Australian Cancer Database (see [methodology](#)). Standardised to the 2001 Australian population; Data were not available by vaccination status; SCC: Squamous cell carcinoma; AC: Adenocarcinoma; Other: All other cancers.

Incidence of anal SCC in females has increased gradually and consistently from 0.8 to 1.7 cases per 100,000 females between 2001 and 2020. Incidence of anal adenocarcinomas has remained low and unchanged at approximately 0.2 cases per 100,000 females (**Figure 4.5A**). The largest increase was in the older age groups, particularly the 60–64-year age group, which rose from 1.7 to 7.7 cases per 100,000 females during this period. Incidence remained similarly high among females aged 65 years and older (**Table A4.3**).

Incidence of anal SCC in males has increased only modestly from 0.7 to 0.9 cases per 100,000 males between 2001 and 2020. Incidence of anal adenocarcinomas has remained low and unchanged at 0.4 cases per 100,000

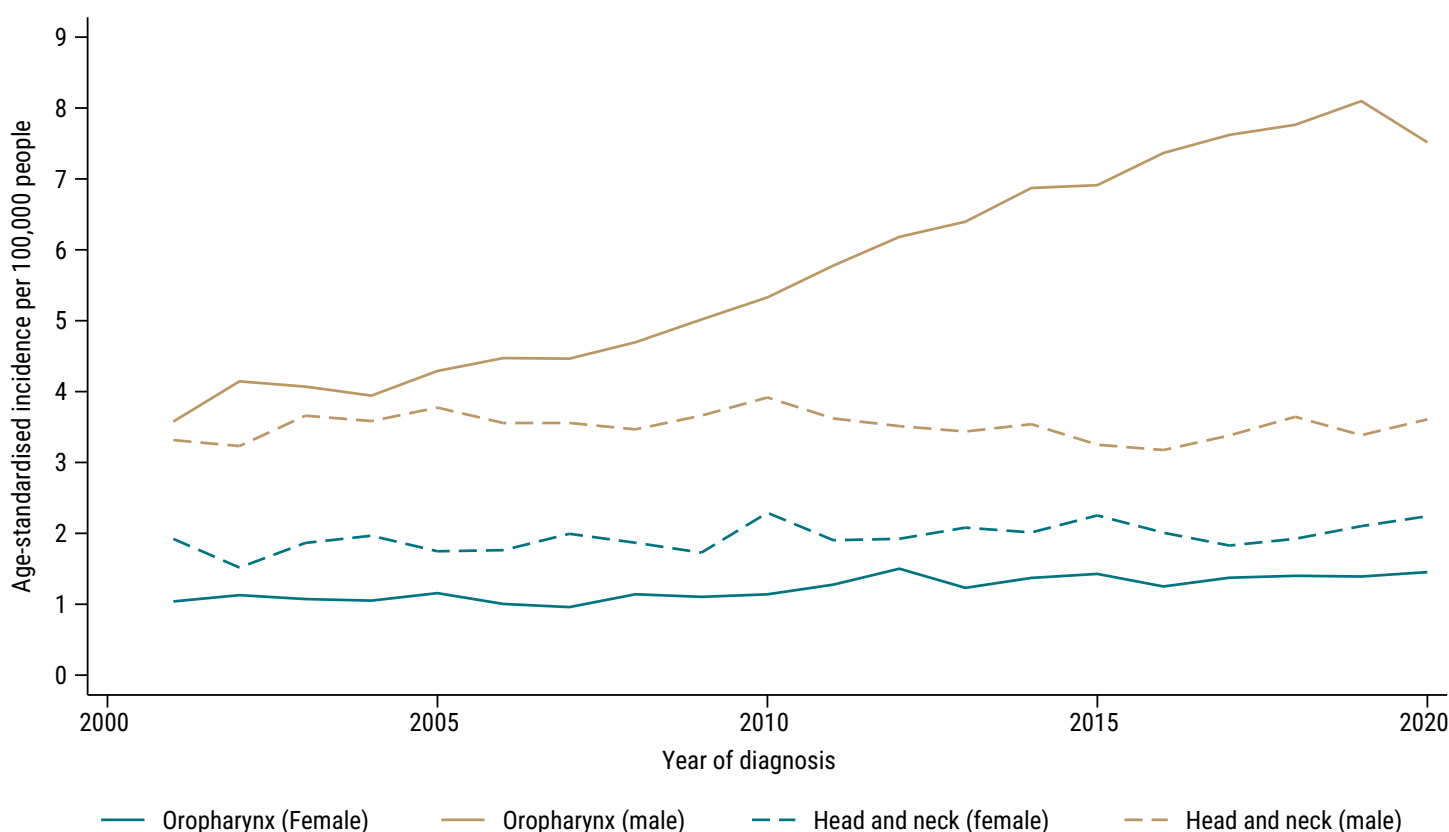
males in 2001 and 2020 (**Figure 4.5A**). Even in older age groups, the incidence has not increased substantially (**Table A4.4**).

The incidence of vulval SCC remained relatively stable between 2001 and 2020. In 2020, the incidence was 2.0 cases per 100,000 (**Figure 4.5B**; **Table A4.5**).

The incidence of vaginal SCC has remained stable over time and in 2020 was 0.3 cases per 100,000 population (**Figure 4.5B**; **Table A4.5**).

The incidence of penile SCC has remained stable, with 0.9 cases per 100,000 population in 2020 (**Figure 4.5B**; **Table A4.5**).

Figure 4.6 Age-standardised incidence rates of oropharyngeal cancers (frequently associated with HPV)* and other head and neck cancers (not usually associated with HPV**), in females and males, per 100,000 population, 2001–2020



Source: Australian Cancer Database (see [methodology](#)). Standardised to the 2001 Australian population; Data were not available by vaccination status. *Oropharynx: denotes cancers frequently associated with HPV: base of tongue, lingual tonsil, soft palate, uvula, tonsil, oropharynx, Waldeyer ring. **Head and neck: denotes cancers not usually associated with HPV: upper lip (inner aspect), lip (unspecified, inner aspect), dorsal surface of tongue, anterior two-thirds of tongue (part unspecified), gum, floor of mouth, hard palate, other and unspecified parts of mouth.

Since oropharyngeal cancer can develop through both HPV-dependent and HPV-independent pathways, detecting the impact of HPV vaccination on incidence is challenging. Comparing incidence trends in HPV-related oropharyngeal cancers with those in head and neck cancers not usually linked to HPV—associated with tobacco and alcohol use—may help disentangle the effects of vaccination from changes in exposure to these carcinogens⁹⁸.

Oropharyngeal cancers frequently associated with HPV infection: These cancers include SCC of the base of the tongue, the lingual tonsil, the soft palate, uvula, tonsil, oropharynx, and the Waldeyer ring⁹⁸.

For females, there was a very small increase in incidence, from 1.0 to 1.5 cases per 100,000, between 2001 and 2020. Incidence in males was notably higher and increased steadily and consistently, rising from 3.6 to 7.5 cases per

100,000 between 2001 and 2020 (**Figure 4.6; Table A4.7**). This increase was particularly notable in males aged 50 and over, where rates have almost doubled between 2001 and 2020 (**Table A4.7**).

Head and neck cancers not usually associated with HPV infection: These cancers include SCC of the upper lip (inner aspect), the lip (unspecified, inner aspect), the dorsal surface of the tongue, the anterior two-thirds of the tongue (part unspecified), the gum, floor of the mouth, hard palate, and other and unspecified parts of the mouth⁹⁸.

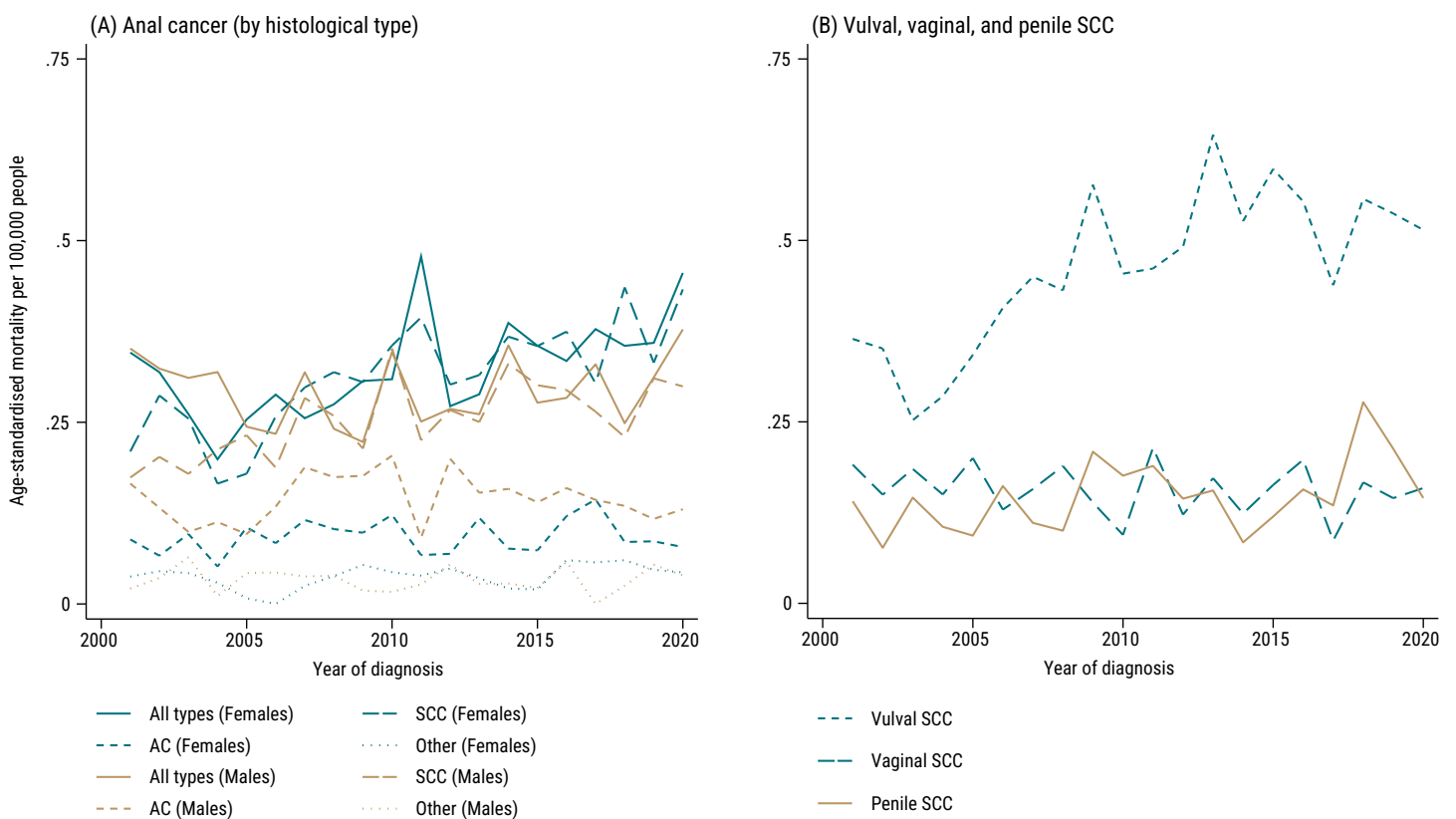
Incidence remained stable for males over the time period and in 2020 was 3.6 cases per 100,000. For females, incidence was also stable but lower than for males at 2.2 cases per 100,000 in 2020 (**Figure 4.6; Table A4.8**).



Indicator: The number of deaths caused by anogenital and oropharyngeal cancer, by age, type, vaccination status and sex per 100,000 estimated resident population in a calendar year

Status of indicator

Figure 4.7 Age-standardised mortality rates of anal cancer by histologic type (A) and vulval, vaginal and penile squamous cell carcinoma (SCC) (B) in females and males, per 100,000 estimated resident population, 2001–2020



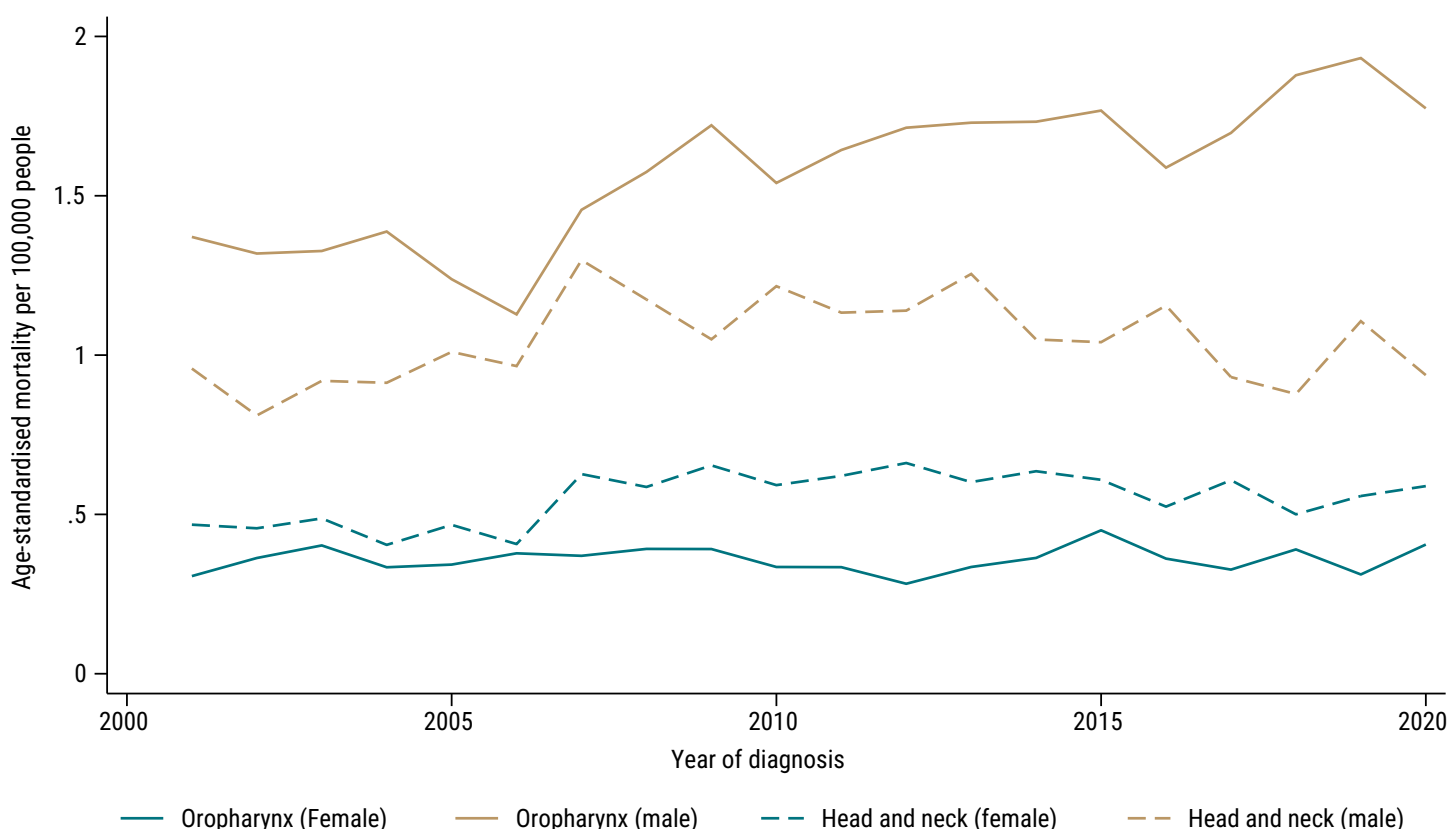
Source: Australian Cancer Database (see [methodology](#)). Standardised to the 2001 Australian population; Data were not available by vaccination status; SCC: Squamous cell carcinoma; AC: Adenocarcinoma; Other: All other cancers.

Mortality from anal SCC in females was low but has risen gradually from 0.2 to 0.4 deaths per 100,000 population between 2001 and 2020. The highest mortality rate was in the ≥ 75 years group, which increased from 1.5 to 3.1 deaths per 100,000 population during this period. Deaths from anal adenocarcinoma were also very rare and stable over time. Anal cancer mortality in males followed a pattern similar to that of females (Figure 4.7A; Table A4.9; Table A4.10).

Mortality from vulval and vaginal SCC was rare and stable over time and in 2020 was 0.5 deaths and 0.2 deaths per 100,000 population, respectively (Figure 4.7B; Table A4.11). The highest mortality rate was in the ≥ 75 years group for both types of cancer (Table A4.11).

Mortality from penile SCC has remained stable over time and in 2020 was 0.1 deaths per 100,000 population (Figure 4.7B; Table A4.12).

Figure 4.8 Age-standardised mortality rates from oropharyngeal cancers (frequently associated with HPV*) and other head and neck cancers (not usually associated with HPV**), in females and males, per 100,000 population, 2001–2020



Source: Australian Cancer Database (see [methodology](#)). Standardised to the 2001 Australian population; Data were not available by vaccination status; *Oropharynx: denotes cancers frequently associated with HPV: base of tongue, lingual tonsil, soft palate, uvula, tonsil, oropharynx, Waldeyer ring; **Head and neck: denotes cancers not usually associated with HPV: upper lip (inner aspect), lip (unspecified, inner aspect), dorsal surface of tongue, anterior two-thirds of tongue (part unspecified), gum, floor of mouth, hard palate, other and unspecified parts of mouth.

Oropharyngeal cancers frequently associated with HPV infection: For females, mortality rates have remained stable at between 0.3 and 0.4 deaths per 100,000 population over the period. In males, mortality rates appear to have increased modestly from 1.4 to 1.8 deaths per 100,000 population between 2001 and 2020. For both females and males, the highest mortality rates were in those aged 60 years and older. Mortality rates were 3–4 times higher in males than in females (**Figure 4.8; Table A4.13**).

Head and neck cancers not usually associated with HPV infection: For females, mortality rates were stable at between 0.5 and 0.6 deaths per 100,000 over the period. For both females and males, the highest mortality rates were in those aged 55 years and older. Mortality rates were only slightly higher in males than in females (**Figure 4.8; Table A4.14**).



Indicator: The number of new cases of anogenital and oropharyngeal cancer among Aboriginal and Torres Strait Islander peoples, by age, type, vaccination status and sex per 100,000 estimated resident Aboriginal and Torres Strait Islander population in a calendar year

Status of indicator

Table 4.3 Anogenital squamous cell carcinoma (SCC) incidence rates in Aboriginal and Torres Strait Islander peoples per 100,000 estimated resident Indigenous population, 5-year pooled for 2016–2020

	Anal		Vulval		Vaginal		Penile	
	Females	Males	Females	Females	Females	Males	Females	Males
All ages	1.9	1.8	3.7	1.5	1.5			
0–14	0.0	0.0	0.0	0.0	0.0	0.0		
15–19	0.0	0.0	0.0	0.0	0.0	0.0		
20–24	0.0	0.0	0.0	0.0	0.0	0.0		
25–29	0.6	0.0	0.6	0.0	0.0	0.0		
30–34	0.0	1.4	3.6	0.0	0.0	0.0		
35–39	1.6	0.0	4.1	0.0	0.0	0.0		
40–44	4.3	0.9	1.7	0.0	0.0	0.9		
45–49	0.8	3.5	5.8	0.0	0.0	0.0		
50–54	1.9	2.1	8.7	4.9	0.0	0.0		
55–59	4.5	6.1	6.8	1.1	3.7			
60–64	6.1	1.6	7.6	1.5	6.5			
65+	5.5	6.6	10.2	8.3	7.6			

Source: Australian Cancer Database (see [methodology](#)). All ages category standardised to the 2001 Australian population. Data were not available by vaccination status.

The 5-year pooled incidence (2016–2020) of anal SCC was comparable between females and males at 1.9 and 1.8 per 100,000 resident Aboriginal and Torres Strait Islander peoples, respectively. The incidence increases with age for both sexes (**Table 4.3**). Across 2016–2020, fewer than 5 cases of anal adenocarcinoma were reported in females and in males combined.

The 5-year pooled incidence (2016–2020) of vulval SCC was 3.7 per 100,000, more than double that of anal cancer. The incidence of vaginal SCC was lower at 1.5 per 100,000. Incidence increased with increasing age for both vulval and vaginal cancers, with an incidence of 10.2 and 8.3 per 100,000, respectively, in females aged 65+ (**Table 4.3**).

The 5-year pooled incidence (2016–2020) of penile SCC was low at 1.5 cases per 100,000 resident Aboriginal and Torres Strait Islander males, with incidence increasing with increasing age (**Table 4.3**).

Table 4.4 Incidence rates of oropharyngeal cancers (frequently associated with HPV*) and other head and neck cancers (not usually associated with HPV) in Aboriginal and Torres Strait Islander peoples per 100,000 estimated resident Indigenous population, 5-year pooled for 2016–2020**

	Oropharyngeal cancer (frequently associated with HPV*)		Head and neck cancer (not usually associated with HPV**)	
	Females	Males	Females	Males
All ages	3.8	13.7	2.0	5.4
0–14	0.0	0.0	0.0	0.0
15–19	0.0	0.0	0.0	0.0
20–24	0.0	0.0	0.0	0.0
25–29	0.0	0.0	0.0	0.0
30–34	0.0	0.7	0.0	0.7
35–39	1.6	5.1	0.8	0.8
40–44	0.0	10.0	2.6	2.7
45–49	3.3	15.0	0.0	4.4
50–54	6.8	22.1	4.9	10.5
55–59	18.1	41.8	4.5	14.8
60–64	7.6	60.6	9.1	32.7
65+	13.8	41.5	6.5	15.3

Source: Australian Cancer Database (see [methodology](#)). All ages category standardised to the 2001 Australian population. Data were not available by vaccination status; *Oropharynx: denotes cancers frequently associated with HPV: base of tongue, lingual tonsil, soft palate, uvula, tonsil, oropharynx, Waldeyer ring⁹⁸; **Head and neck: denotes cancers not usually associated with HPV: upper lip (inner aspect), lip (unspecified, inner aspect), dorsal surface of tongue, anterior two-thirds of tongue (part unspecified), gum, floor of mouth, hard palate, other and unspecified parts of mouth⁹⁸.

Oropharyngeal cancers frequently associated with HPV infection: The 5-year pooled incidence (2016–2020) was more than three times higher in males than in females (13.7 versus 3.8 per 100,000, respectively). In both sexes, incidence increased with increasing age. However, the difference between the sexes was even more pronounced in older age groups, with a rate of 60.6 per 100,000 in males aged 60–64, compared with 7.6 per 100,000 in females of the same age group (Table 4.4).

Head and neck cancers not usually associated with HPV infection: The 5-year pooled incidence (2016–2020) in males was more than 2.5 times that of females (5.4 versus 2.0 per 100,000, respectively). The differences between the sexes were even more pronounced in older age groups, with a rate of 32.7 per 100,000 in males aged 60–64 compared with 9.1 per 100,000 in females of the same age group (Table 4.4).



Indicator: The number of deaths caused by anogenital and oropharyngeal cancer among Aboriginal and Torres Strait Islander peoples, by age, type, vaccination status and sex per 100,000 estimated resident Aboriginal and Torres Strait Islander population in a calendar year

Status of indicator

Table 4.5 Anogenital squamous cell carcinoma (SCC) mortality rates in Aboriginal and Torres Strait Islander peoples per 100,000 estimated resident Indigenous population, 5-year pooled for 2016–2020

	Anal		Vulval		Vaginal		Penile	
	Females	Males	Females	Females	Females	Males	Females	Males
All ages	0.5	0.3	0.7	0.8	0.3			
0–14	0.0	0.0	0.0	0.0	0.0			
15–19	0.0	0.0	0.0	0.0	0.0			
20–24	0.0	0.0	0.0	0.0	0.0			
25–29	0.0	0.0	0.0	0.0	0.0			
30–34	0.0	0.0	0.0	0.0	0.0			
35–39	0.8	0.0	0.0	0.0	0.0			
40–44	0.0	0.0	0.0	0.0	0.0			
45–49	0.8	0.9	1.7	0.0	0.0			
50–54	1.9	1.1	0.0	0.0	0.0			
55–59	1.1	0.0	0.0	1.1	0.0			
60–64	1.5	3.3	0.0	0.0	1.6			
65+	0.9	0.0	4.6	5.5	2.2			

Source: Australian Cancer Database (see [methodology](#)). All ages category standardised to the 2001 Australian population. Data were not available by vaccination status.

The 5-year pooled mortality rate (2016–2020) of anal SCC was low for females and males (0.5 and 0.3 per 100,000, respectively) (Table 4.5). There were fewer than 5 deaths from adenocarcinoma in the time period (data not shown).

The 5-year pooled mortality rate (2016–2020) of vulval and vaginal SCC was low and comparable at 0.7 and 0.8 per 100,000, respectively. However, age-specific mortality rates increased with increasing age, to 4.6 and 5.5 per 100,000 Aboriginal and Torres Strait Islander peoples aged 65 years and older (Table 4.5).

The 5-year pooled mortality rate (2016–2020) of penile SCC was low at 0.3 per 100,000. The rate remained at 0.0 until the 60–64 age group, when it increased to 1.6 per 100,000 and then to 2.2 per 100,000 for those aged 65 years and older (Table 4.5).

Table 4.6 Mortality rates from oropharyngeal cancers frequently associated with HPV* and other head and neck cancers not usually associated with HPV in Aboriginal and Torres Strait Islander peoples per 100,000 estimated resident Indigenous population, 5-year pooled for 2016–2020**

	Oropharyngeal cancer (frequently associated with HPV*)		Head and neck cancer (not usually associated with HPV**)	
	Females	Males	Females	Males
All ages	1.6	6.0	1.2	1.8
0–14	0.0	0.0	0.0	0.0
15–19	0.0	0.0	0.0	0.0
20–24	0.0	0.0	0.0	0.0
25–29	0.0	0.0	0.0	0.0
30–34	0.0	0.0	0.0	0.0
35–39	0.0	1.7	0.0	0.0
40–44	0.9	3.6	1.7	0.0
45–49	1.7	8.0	0.0	1.8
50–54	6.8	8.4	1.9	6.3
55–59	5.7	8.6	2.3	3.7
60–64	4.6	31.1	3.0	4.9
65+	3.7	21.8	5.5	6.6

Source: Australian Cancer Database (see [methodology](#)). All ages category standardised to the 2001 Australian population. Data were not available by vaccination status. *Frequently HPV-associated: Base of tongue, lingual tonsil, soft palate, uvula, tonsil, oropharynx, Waldeyer ring. **Not usually HPV-associated: upper lip (inner aspect), lip (unspecified, inner aspect), dorsal surface of tongue, anterior two-thirds of tongue (part unspecified), gum, floor of mouth, hard palate, other and unspecified parts of mouth.

Oropharyngeal cancers frequently associated with HPV infection: The 5-year pooled mortality rate (2016–2020) of oropharyngeal SCC (frequently associated with HPV infection) was almost four times higher in males than females (6.0 versus 1.6 per 100,000, respectively). This difference was especially pronounced in older age groups, with an age-specific mortality rate of 31.1 per 100,000 males compared with 4.6 per 100,000 females in those aged 60–64 (Table 4.6).

Head and neck cancers not usually associated with HPV infection: The 5-year pooled mortality rate (2016–2020) of head and neck SCC (not usually associated with HPV) was slightly higher in males than females (1.8 versus 1.2 per 100,000, respectively). The age-specific mortality rate increased with increasing age for both sexes (Table 4.6).

Data gaps and limitations

- Individual-level vaccine status is not routinely linked to cancer incidence and mortality data. However, any effects are unlikely to be evident yet, as vaccine-eligible cohorts have not yet reached the typical age of onset.
- The collection of national cancer incidence data in the Australian Cancer Database is delayed by four years, limiting timely assessment of trends.
- Mortality data were sourced from the Australian Cancer Database rather than the National Mortality Database (NMD), as the NMD does not allow for stratification of deaths by histology type. Consequently, data could only be reported up to 2020, rather than 2022, the most recent year available in the NMD.
- Incidence and mortality data by HIV status are not available. Given the elevated risk of HPV-associated cancers in immunocompromised individuals—particularly people with HIV, solid organ transplant recipients—such stratification would support trend monitoring in high-risk groups, especially for anal cancer.
- There are both HPV-associated and HPV-independent causes of head and neck cancers. Without linking p16 test results (a marker of HPV positivity), it is not possible to determine HPV attribution and therefore the potential preventability through vaccination. Although oropharyngeal sites are grouped based on HPV relevance, other risk factors—like smoking—may outweigh HPV impact in some populations, complicating interpretation.
- Histology codes 8085 (HPV-positive SCC) and 8086 (HPV-negative SCC) have been available since 2006 to distinguish cancers by HPV status. However, their inconsistent use and reporting across pathology laboratories limit their reliability for estimating incidence and mortality rates of HPV-associated cancers.
- Age-standardisation was carried out using the 2001 Australian population structure for both the national Australian population and Aboriginal and Torres Strait Islander peoples. Whilst this was done largely to enable direct comparison between datasets, it was also due to a lack of a robust age-standardised population for the Indigenous population in Australia.
- Aboriginal and Torres Strait Islander rates were restricted to jurisdictions with adequate Indigenous identification in cancer registry data. Consequently, estimates should not be interpreted as representing complete coverage of Australia.

- Further to the above, trends in incidence and mortality could not be examined for Aboriginal and Torres Strait Islander peoples, due to uncertainties about the consistency of the population estimates over time and the need to pool cases over multiple years due to small numbers.

Data interpretation

Anogenital (excluding cervical) and head and neck cancers tend to occur in older age groups, with a higher median age of diagnosis of these cancers (above 60 years) than cervical cancer (Australian median age 46 in 2020)⁹⁹. Therefore, it is too soon to see any potential impact of the Australian HPV vaccination program on incidence or mortality rates of these malignancies. It will likely be several more decades before the impact is seen⁹⁰.

Incidence rates of anal SCC in females have continued to rise gradually, consistent with global trends¹⁰⁰. Changes in sexual behaviour, such as an increased number of sexual partners and anal sexual practices, as well as the anatomical proximity of the vagina and anus, are hypothesised to be responsible for this increase in incidence over time¹⁰⁰. However, incidence rates of vulval, vaginal, and penile cancers have remained largely unchanged over the last 20 years.

Mortality rates of anal SCC have increased slightly in both males and females, remaining higher in females. This increase is attributed to the disease often being diagnosed at an advanced stage, due to a lack of screening or treatment guidelines during the data reporting period^{101,102}. However, mortality rates for vulval, vaginal, and penile SCC have remained low and largely unchanged over the last 20 years, with only a slight increase in mortality rates for vulval SCC.

Incidence rates of oropharyngeal SCC, which is frequently associated with HPV infection, have doubled over the last 20 years in males, consistent with global trends⁹⁸, and trends previously observed in Queensland, Australia¹⁰³. Australian studies have also shown an increase in the proportion of oropharyngeal cancers with HPV detected over time, from 20.2% in 1987–1995 to 63.5% in 2006–2010¹⁰⁴. This is attributed to changes in patterns of sexual behaviour, such as increased numbers of sexual partners, and increased rate and earlier commencement age of oral sex¹⁰³. By contrast, incidence rates and mortality rates of head and neck cancer not usually associated with HPV infection have remained consistent and low over the last 20 years, with only a slight increase in the mortality rate in males. It is somewhat surprising that a decrease was

4. MONITORING CANCER ENDPOINTS

not observed in the incidence of head and neck cancer not usually associated with HPV, given the reduction in tobacco use in Australia over the past 20 years, and the strong correlation of tobacco use with head and neck cancer^{105,106}. However, given that tobacco smoking is also an overlapping risk factor for many of the oropharyngeal cancers linked to HPV infection, these data are difficult to interpret in the absence of direct information on HPV.

Incidence and mortality associated with oropharyngeal SCC, which is frequently associated with HPV infection, were substantially higher in Aboriginal and Torres Strait Islander males than females and increased with increasing age. While these trends are consistent with the broader national population, both incidence and mortality rates were higher among Aboriginal and Torres Strait Islander peoples. In 2016–2020, age-standardised incidence was 2.7 times higher in females and 1.8 times higher in males, and mortality 4.0 times higher in females and 3.3 times higher in males among Aboriginal and Torres Strait Islander peoples compared to non-Indigenous rates. Similar disparities were found in previous analyses of cancer data, including the period back to 1998, with around twice the age-standardised incidence^{107,108}, and over 3.5 times the mortality of head and neck cancers among Aboriginal and Torres Strait Islander peoples compared with non-Indigenous rates¹⁰⁸, indicating no improvements over time.

There are likely multiple contributors to higher incidence and mortality among Aboriginal and Torres Strait Islander peoples, in particular males. Tobacco use, alcohol consumption and HPV infection contribute to the development of oropharyngeal cancer. Government policies and broader social determinants have contributed to the higher rates of tobacco use and addiction among Aboriginal and Torres Strait Islander peoples¹⁰⁹. Aboriginal and Torres Strait Islander men have higher rates of daily smoking and risky alcohol consumption compared to women, contributing to elevated risks for malignancy, including oropharyngeal cancer. There have been considerable declines in tobacco smoking among Aboriginal and Torres Strait Islander peoples, with a 9.8% reduction in daily smoking for those aged over 18 years between 2004–05 and 2018–19¹¹⁰. However, the long latency period for the development of oropharyngeal cancers means any corresponding decrease in incidence and mortality may not become evident for some time. Aboriginal and Torres Strait Islander peoples are also more likely to abstain from drinking alcohol than non-Indigenous peoples. However, those who do drink are more likely to consume alcohol in ways that put their health at risk¹¹⁰.

Although risky alcohol consumption has declined over time, it is too soon to observe any impact on oropharyngeal cancer incidence and mortality¹¹¹.

In the national population, where overall tobacco use is relatively low, the proportion of cases attributable to HPV infection is likely higher, because there are fewer cases due to tobacco in the denominator. Conversely, when tobacco use is higher, as is the case in Aboriginal and Torres Strait Islander men compared with the national population, the proportion of cases attributable to HPV infection is lower, since there will be a greater number attributed to tobacco. Since non-HPV-associated oropharyngeal cancers usually have a poorer prognosis than HPV-associated, it would be expected that mortality rates in Aboriginal and Torres Strait Islander men would be higher due to the higher proportion of non-HPV-associated oropharyngeal cancers. This higher mortality rate is also consistent with published reports showing Aboriginal and Torres Strait Islander peoples often present at a younger age, with worse survival and poorer prognosis for oral cavity and oropharyngeal carcinomas^{112,113}. Higher rates of co-morbidities in Aboriginal and Torres Strait Islander peoples likely also contribute to increased mortality¹¹⁴.

Systemic racism and a lack of culturally safe healthcare environments create barriers for Aboriginal and Torres Strait Islander peoples, impacting timely access to cancer diagnosis and treatment. These systemic issues contribute to higher cancer incidence, later detection, lower survival rates, and increased mortality. Although specific research on oropharyngeal cancer is limited, broader cancer disparities are closely linked to structural inequities, including socioeconomic disadvantage, reduced life expectancy, and geographic isolation. These challenges are further compounded by systemic barriers, cultural perceptions of cancer, and a lack of culturally appropriate care, all of which contribute to poorer outcomes³¹.

4.3 Monitor the distribution of HPV genotypes detected in cervical cancer

Justification for this monitoring objective

With the introduction of the nonavalent HPV vaccine and primary HPV-based screening, monitoring the HPV types that cause cervical cancer is critical for evaluating the effectiveness of both programs over time. As vaccinated cohorts move through the screening program, ongoing

surveillance will be essential to track reductions in cancers caused by vaccine-targeted types, clarify the role of non-vaccine types in the remaining burden, and document HPV-negative cancers. Such data are central to assessing vaccine impact, explaining residual disease, informing future prevention strategies, and ensuring Australia stays on track to achieve—and sustain—cervical cancer elimination.



Indicator: Prevalence of HPV, by type, age and vaccination status, in cervical cancer specimens.

Status of indicator

Figure 4.9 Prevalence (%) of HPV by type among people of all ages with a first cervical cancer histology result, 2018–2023



Source: National Cancer Screening Register (see [methodology](#)).

Between 2018 and 2023, the proportion of people with cervical cancer who had a routine clinical HPV test result recorded over the 5 years preceding the result increased steadily, from 76.2% to 89.0% across all ages, with a corresponding decline in unknown status. Among cases with known HPV status, HPV detection remained high, ranging from 88.8% to 92.3%. Across all ages, HPV16/18-only cases decreased from 78.1% to 64.2%, non-16/18-only cases increased from 18.2% to 26.5%, and cases with both HPV16/18 and other types rose from 3.7% to 9.3%. Trends among people aged 25–74 were similar (**Appendix Table A15**).

By age group, the largest changes were observed in younger people: under 30, HPV16/18-only fell from 82.9% to 36.0%, non-16 and 18-only rose from 17.1% to 52.0%, and mixed types increased from 0% to 12.0%; in ages 30–39, HPV16/18-only decreased from 83.2% to 59.2%, non-16 and 18-only increased from 12.0% to 28.8%, and mixed types rose from 4.9% to 12.0%. Among people 40–49 and 50–59, HPV16/18-only declined modestly; non-16/18-only and mixed types showed small increases. In those aged 60–74 and 75+, HPV16/18-only, non-16/18-only, and mixed types fluctuated over the years, with no clear trend (**Figure 4.9; Appendix Table A15**).



Indicator: Prevalence of HPV, by type, age and vaccination status, in cervical cancer specimens from Aboriginal and Torres Strait Islander females

Status of indicator

Table 4.7 Prevalence (%) of HPV by type among Aboriginal and Torres Strait Islander people of all ages with a first cervical cancer histology result, 2018–2023

	2018	2019	2020	2021	2022	2023
Known HPV status	83.3	87.5	87.2	96.7	90.3	91.4
Unknown HPV status	16.7	12.5	12.8	3.3	9.7	8.6
HPV detected	93.3	90.5	88.2	93.1	92.9	87.5
HPV never detected (a)	6.7	9.5	11.8	6.9	7.1	12.5
Only HPV16/18 ^a	71.4	84.2	76.7	70.4	80.0	75.0
Only non-HPV16/18 types (a) ^a	25.0	10.5	10.0	25.9	20.0	21.4
Both HPV16/18 and other (a) ^a	3.6	5.3	13.3	3.7	0.0	3.6

Source: National Cancer Screening Register (see [methodology](#)). (a) Some rates are based on numerators < 10 or denominators < 20 and should be interpreted with caution.

^a Among those with a known HPV result over the 5 years preceding the cancer histology result.

Between 2018 and 2023, the proportion of Aboriginal and Torres Strait Islander people with cervical cancer who had a routine clinical HPV test result recorded over the 5 years preceding the result increased from 83.3% to 91.4% across all ages, with a corresponding decline in unknown status. Among cases with known HPV status, HPV detection remained high but fluctuated slightly, ranging from 88.2% to 93.3%, with HPV never detected in 6.7% to 12.5% of cases (**Table 4.7**).

Among people of all ages with known HPV status, the proportion of HPV16/18-only cases ranged from 70.4% to 84.2%, non-16/18-only cases ranged from 10.0% to 25.9%, and cases with both HPV16/18 and other types ranged from 0.0% to 13.3%. There were no incident cervical cancers for Aboriginal and Torres Strait Islander people outside the 25–74 year age group, therefore proportions in the 25–74 year and all ages groups were identical (**Table 4.7**). There were insufficient cases to assess proportions by age reliably.

Data gaps and limitations

- These data reflect only cancers reported to the NCSR and not all cervical cancers notified to the cancer registries; therefore, the observed patterns may not fully represent all cervical cancers diagnosed in a given year.
- Age-specific trends could not be reported for Aboriginal and Torres Strait Islander peoples due to small numbers (typically fewer than 50 cases per year)¹¹⁵. A small number of cases also results in wider variation in the proportions within each HPV grouping by calendar year than seen in the overall Australian cohort.
- Vaccination status is not linked to screening records, limiting the ability to stratify distribution by vaccination history.
- Possible under-ascertainment of non-16 and 18 types in people who also have HPV16/18 detected due to clinical reporting rules, which could bias the observed prevalence of mixed versus single-type infections with HPV16/18 (but would not affect results for only non-16 and 18 types).
- Typing results from standard clinical HPV tests are reported for the sample (which may have been collected from the cervix or vagina, depending on whether it was self-collected or clinician-collected), not specifically at the lesion level. Multiple HPV types may be present in the cervical/ vaginal sample, without the causal type of the cancer being identifiable.
- While completeness of HPV information improved over time, around 1 in 10 cervical cancer cases still lack recorded HPV test results in the preceding 5 years, potentially biasing prevalence estimates.
- Due to the limited number of cases, stratification of the analysis by histopathological type of cervical cancer has not been undertaken. Variation in HPV type distribution over time will naturally reflect the proportions of cancers diagnosed as squamous cell, adenocarcinoma, or other types.

Data interpretation

Before HPV vaccination was introduced, cervical cancers in Australia were overwhelmingly associated with HPV16/18. In a study of 847 cancers diagnosed between 2005 and 2015, HPV was detected in 92.9% of cases, and more than three-quarters (77.1%) of HPV-positive cancers contained HPV16/18. Thus, only 23% contained non-16 and 18 types alone (15.9% contained HPV 31, 33, 45, 52, or 58, and 7.0% contained another HPV type). The dominance of HPV16/18 was even stronger for cancers in younger

women: 81.6% of cervical cancers diagnosed in those aged 20–39 contained HPV16/18. Compared with international series, the Australian pre-vaccine distribution showed a high proportion of HPV16/18, likely reflecting the effect of cervical screening in preventing squamous cancers and thereby enriching for adenocarcinomas, in which types 16 and 18 predominate⁷⁹. Against this pre-vaccine backdrop, the post-vaccine data show a clear and substantial decline in HPV16/18-only cervical cancers, especially among women under 40. This shift is most pronounced in women under 30, where the proportion of cancers caused by HPV16/18 has dropped by more than half, and non-16 and 18 types now account for the majority of cases. These findings align with vaccine coverage patterns and the natural ageing of vaccinated cohorts into screening.

At the same time, the relative increase in cancers caused by non-16 and 18 types—both as sole infections and in combination with HPV16/18—highlights the residual burden from types not targeted by the original bivalent/quadrivalent vaccines. This trend highlights the value of the nonavalent vaccine, which covers additional oncogenic types, and underscores the importance of continuing to monitor type distribution to confirm its longer-term effects. It is important to note, however, that both HPV vaccination and HPV based cervical screening are efficacious at preventing cervical adenocarcinomas in the population, unlike previous cytology-based screening programs. In interpreting the relative proportion of type grouping causing cancer over time, it is important to note that rising proportions of non-16 and 18 related cancer do not imply a rise in the number of people with cervical cancer⁹⁹. Absolute numbers of cervical cancers diagnosed are reflected in incidence rate trends over time (See Indicator 4.1). It was difficult to identify any trends in the relative proportion of HPV16/18 and other oncogenic types over time among Aboriginal and Torres Strait Islander people with cervical cancer, with year-to-year variation in proportions likely due to small case numbers (typically fewer than 50 cases per year)

Around one in ten cervical cancers in this dataset, for which at least one clinical HPV test result was available in the preceding five years, did not have HPV detected on any clinical HPV test. Some of these are likely to be truly HPV-independent tumours (e.g. rare histological subtypes), but others may reflect limitations of current clinical assays in detecting infection in cancers where HPV DNA has integrated into the host genome and is no longer easily detectable. (i.e. false negative results)⁹⁹.

4.4 Monitor the distribution of HPV genotypes detected in anogenital and oropharyngeal cancers

Tracking HPV prevalence in anogenital and oropharyngeal cancer specimens allows direct monitoring of reductions in cancers caused by vaccine-preventable HPV types. It also helps clarify the causes of remaining cancers, guiding future prevention efforts.



Indicator: Prevalence of HPV, by type, age, sex and vaccination status, in anogenital cancer specimens

Indicator: Prevalence of HPV, by type, age, sex and vaccination status, in anogenital cancer specimens from Aboriginal and Torres Strait Islander peoples

Indicator: Prevalence of HPV, by type, age, sex and vaccination status, in oropharyngeal cancer specimens

Indicator: Prevalence of HPV, by type, age, sex and vaccination status, in oropharyngeal cancer specimens from Aboriginal and Torres Strait Islander peoples

Status of indicator

HPV typing is not routinely performed for anogenital cancers in Australia. For oropharyngeal cancers, however, distinguishing between HPV-positive and HPV-negative tumours has important clinical implications because HPV-positive cancers tend to occur in younger patients, be more chemoradio-sensitive and have a better prognosis¹¹⁶. HPV status can be inferred using p16 immunohistochemistry, a surrogate marker for HPV-associated disease.

Histology codes 8085 (HPV-positive SCC) and 8086 (HPV-negative SCC), based on p16 results, are in use in the Australian Cancer Database for both anogenital and oropharyngeal cancers. The earliest recorded use is a single case coded as HPV-positive in 2006, indicating that these codes have been available since at least that time. However, their use was extremely limited before 2012, with more consistent reporting only evident from 2016 onwards (**Figure 4.10**). For anal SCC, HPV status, based on p16 results, was recorded for 103 of 5,835 cases (1.8%). Of these, 96 (93.2%) cases were HPV positive and 7 (6.8%) were HPV negative. In vaginal SCC, HPV status was recorded for 17 of 1,039 cases (1.6%), all of which (100%) were HPV positive. For vulval SCC, HPV status was available for 39 of 4,705 cases (0.8%), with 17 (43.6%) being HPV positive and 22 (56.4%) HPV negative. No p16 status was available for penile SCC (**Figure 4.10**).

By contrast, HPV status—via p16 testing—was more frequently reported in oropharyngeal SCC, with 1,612 of 17,229 cases (9.4%) having recorded HPV status: 1,425 (88.4%) were HPV positive and 187 (11.6%) HPV negative (**Figure 4.10**).

Data gaps and limitations

- HPV genotyping data to populate these indicators is unavailable, as identifying causal HPV types requires specialised testing, such as laser capture microdissection, which is not routinely performed.
- HPV status based on p16 immunohistochemistry was recorded for only a small fraction of cases. This limits the ability to draw reliable conclusions about HPV prevalence and introduces potential selection bias, as the tested cases may not reflect the broader population.
- While p16 immunohistochemistry is a useful marker for HPV-associated cancers, it does not provide information on the specific HPV genotype involved, limiting insights into the viral types driving disease and the potential impact of HPV vaccines, which are genotype specific.
- Pending MSAC approval, Medicare-funded screening and treatment of precancerous anal lesions may become available in the future. This would increase anal HPV testing in laboratories already involved in cervical screening and support future surveillance efforts for groups at high risk of anal cancer.

Data interpretation

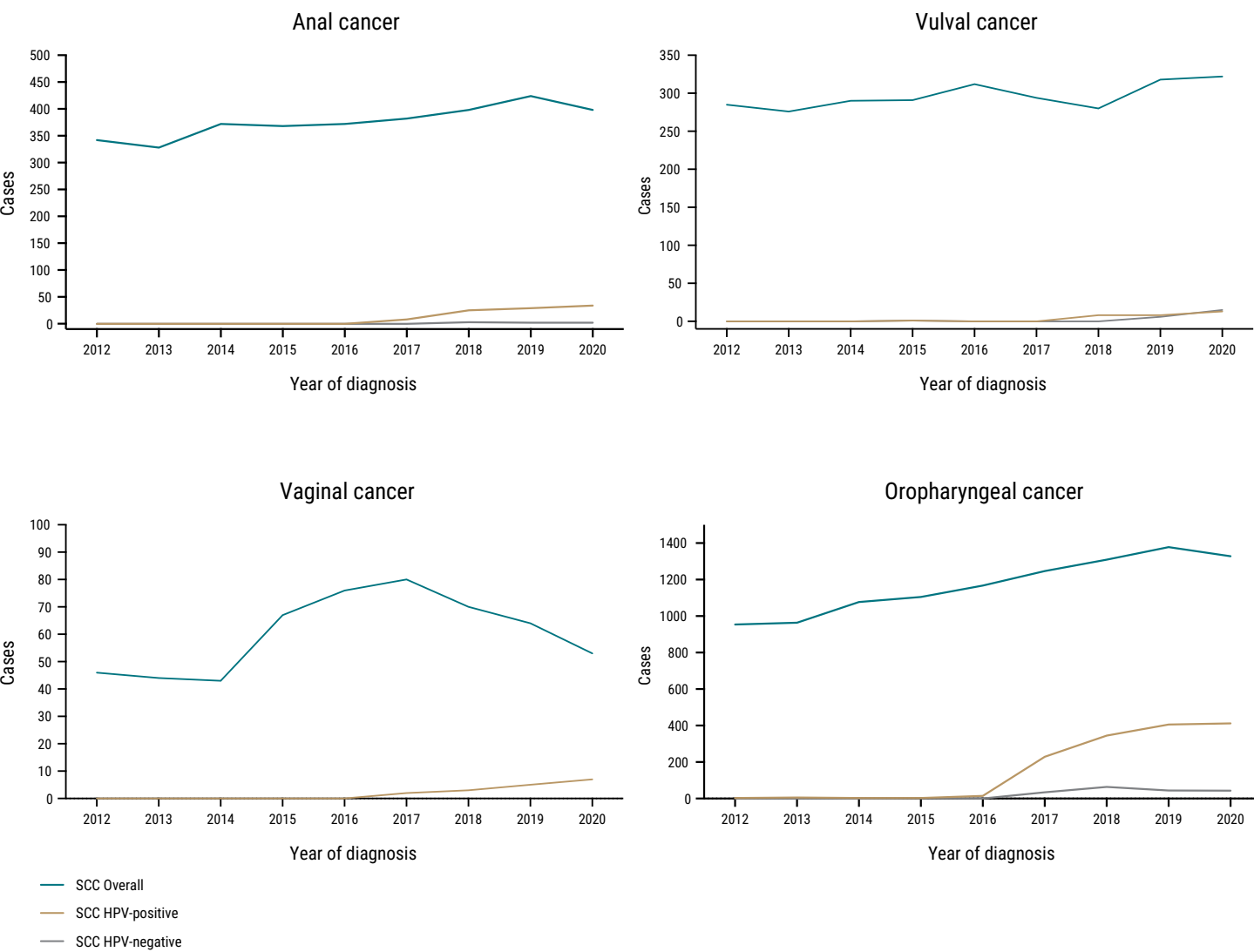
Case numbers recorded under histology codes 8085 and 8086 remain very low compared to the overall number of SCC cases, and no penile cancers have been assigned to either code. For anogenital cancers, this likely reflects both infrequent clinical use and inconsistent reporting. For oropharyngeal cancers, although p16 testing has been part of routine care since 2016, results appear not to be routinely captured in the Australian Cancer Database.

HPV typing studies of anogenital cancers in Australia are challenging to conduct due to the overall rarity of these cancers. Among the available data, an analysis of 112 anal cancer cases diagnosed between 1999 and 2009—comprising 82% males and 18% females—found HPV DNA in 96.4% of cases. HPV type 16 was the most common, detected in 75% of samples and as the sole genotype in 61% of cases. Overall, 90% of cases contained at least one HPV type targeted by the quadrivalent HPV vaccine, and 96% were covered by the nonavalent vaccine.

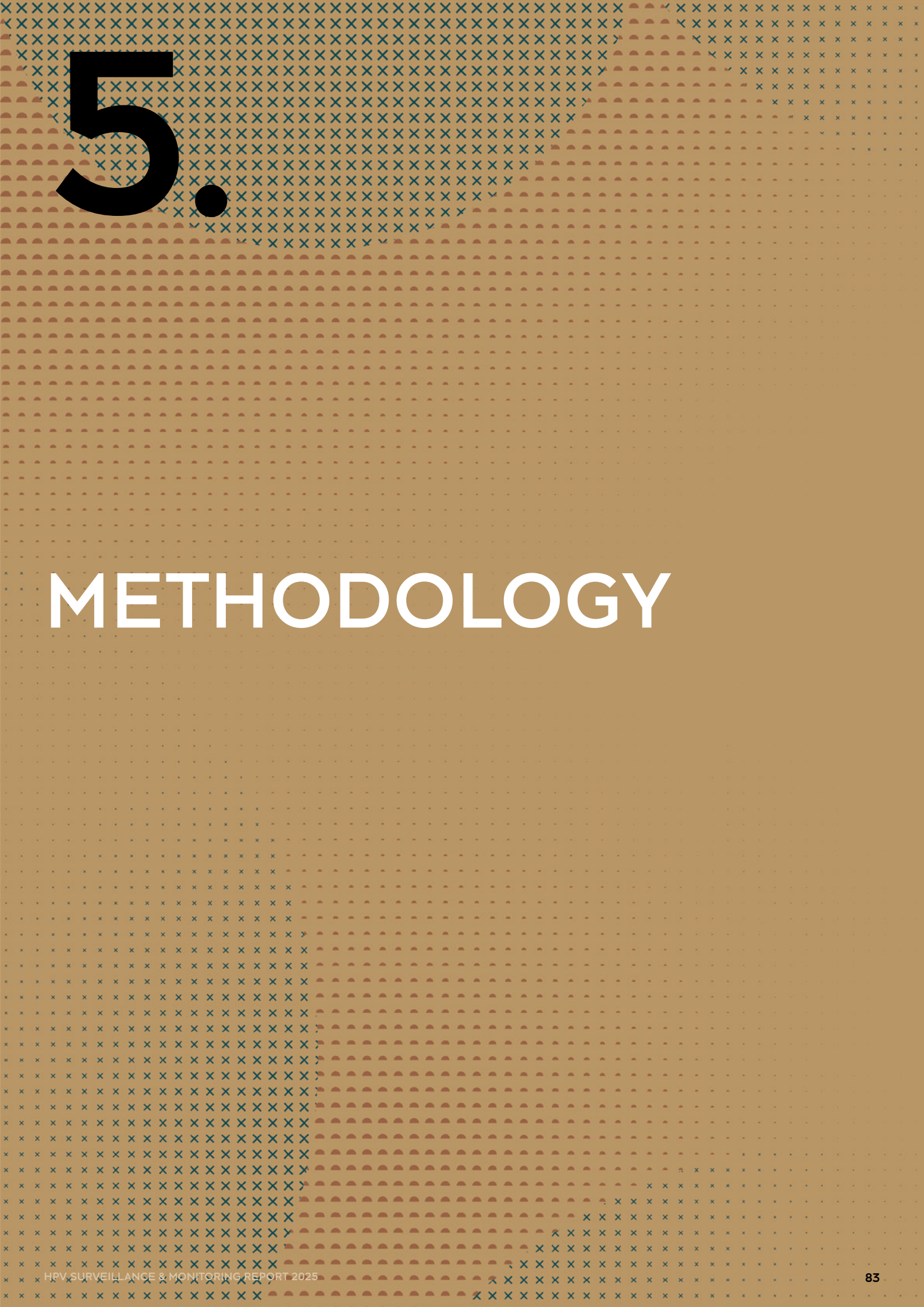
For vulvar cancer, the only significant data come from a documented cluster of SCC cases among Aboriginal and Torres Strait Islander females in the Northern Territory, particularly those from East Arnhem Land. Between 1996 and 2005, 32 women were diagnosed with vulvar cancer in this region. Of these, 21 (66%) were Indigenous, 10 (31%) were non-Indigenous, and the Indigenous status of one woman was unknown¹¹⁷. HPV typing revealed that a substantial proportion of these cancers were associated with high-risk HPV types—100% of cases from Arnhem Land and 80% from other regions—with HPV 16 being the predominant genotype, detected in 70% of cases¹¹⁸. No HPV typing data are currently available for vaginal or penile cancers in Australia.

A study of 302 oropharyngeal cancers diagnosed between 1987 and 2006 found an overall HPV positivity rate of 36%, with types 16 and 18 accounting for 94% of positive cases. The proportion of HPV-related cases increased markedly over time, from 19% between 1987–1990 to 47% between 2001–2005. More recent data from Queensland (2015–2019) show that 96% of newly diagnosed oropharyngeal cancers were SCCs, with 77% testing p16 positive, consistent with the HPV association¹⁰³. These findings align with a single-centre study from New South Wales, which reported a p16/HPV positivity rate of 83% among 244 oropharyngeal SCC cases treated between 2005 and 2017¹¹⁶.

Figure 4.10 Number of incident cases of squamous cell carcinoma (SCC) by histologic codes (8085 – p16 positive; HPV positive, 8086 – p16 negative; HPV status) and overall.



Data source: Australian Cancer Database (see [methodology](#)).



5.

METHODOLOGY

5. METHODOLOGY

Monitoring HPV vaccination coverage (Objective 1.1)

Data source (secondary use of data from the 2025 Cervical Cancer Elimination Report⁷⁶): Australian Immunisation Data as at 31 March 2021 for 2020 coverage, 3 April 2022 for 2021 coverage, 2 April 2023 for 2022 coverage, 4 Feb 2024 for 2023 coverage, and 2 Feb 2025 for 2024 coverage. Data provided by NCIRS.

Area-based socioeconomic status quintiles based on an individual's postcode as recorded on AIR and assigned according to SEIFA IRSD for 2016 for the years 2020 and the SEIFA Index of IRSD for 2021 for the years 2021–2024¹¹⁹.

Remoteness area based on an individual's postcode as recorded on AIR and assigned according to ASGS for 2016, for 2020, and the ASGS for 2021, for 2021–2024¹²⁰.

Aboriginal or Torres Strait Islander identification was based on whether a person had ever self-reported their status, as recorded in the AIR.

Monitoring coverage of cervical screening (Objective 1.2)

Data source (secondary use of data from the 2025 Cervical Cancer Elimination Report⁷⁶): Analysis of NCSR data (NCSR RDE 11/07/2025); data prepared by the Screening Analysis and Monitoring Unit of the Australian Institute of Health and Welfare¹¹⁵. Remoteness areas and socioeconomic areas were allocated to records at the time of the HPV test, primarily using an SA2 2021 correspondence, with postcode correspondence used where SA2 was unavailable¹¹⁵.

Participants with an HPV test include individuals who received an HPV test for any reason within a 5.5-year period (6 months longer than the recommended screening interval) and who were aged 25–74 years at the time of the test. The eligible population for screening comprises the estimated resident female population aged 25–74 years, minus estimates of the number of females who have had a hysterectomy, consistent with routine AIHW reports^{119,121–123}. ABS population estimates have been updated to use those based on the 2021 Census^{119,121,123}. Hysterectomy prevalence from AIHW analysis of the National Hospital Morbidity Database (NHMD)¹²².

Age and residence were determined at the time of the test. Individuals with more than one HPV test in the 5.5-year period contributed only one test; the last test in the period was selected for the purpose of calculating age and place of residence at the time of the test in these analyses,

consistent with the 2025 methodological update (with the first test retained for sub-analyses in other reports). Inclusion of those with an HPV test within a 5.5-year period (6 months longer than the recommended interval) aligns with international screening program methodologies, and allows flexibility for attendance slightly beyond 5 years, including following reminders. See the National Cervical Screening Program Monitoring Report prepared by AIHW for additional information¹¹⁵.

Percentages were age-standardised using the Australia 2001 standard population¹¹⁹.

Monitoring HPV infection prevalence (Objective 2.1)

Data Source (HPV prevalence in cervical screening program attendees - secondary use of data from the 2025 Cervical Cancer Elimination Report⁷⁶): Analysis of NCSR data (NCSR RDE 11/07/2025) prepared by the Screening Analysis and Monitoring Unit of the Australian Institute of Health and Welfare¹¹⁵.

- Results were derived using the standard method for calculating population prevalence (positivity): the sum of females with at least one positive result for a given HPV group divided by the total count of participants with a primary screening HPV test
- Aboriginal or Torres Strait Islander identification was based on whether a person had ever self-reported their status, as recorded in the National Cancer Screening Register

Data Source (other populations): National HPV Monitoring Program (IMPACT). Pathology laboratories across Australia provided de-identifiable residual specimens from key populations for HPV testing²³. Basic demographic data of patients were collected for data linkage to HPV vaccination data held in the AIR, by the AIHW Data Linkage Unit (EO2021/4/1297)¹²⁴.

Population: 1) Females attending chlamydia screening aged 16–24 years; 2) Gay and bisexual men aged 16 years or older. Male rectal swabs used as a proxy for GBMSM population²⁴.

Exclusion criteria: Repeat samples from one individual, specimens from an ineligible age range, unknown sex, or collected in urine.

Primary specimen: Vaginal and cervical swabs (females), rectal swabs (males) collected from seven laboratories in six different states of Australia.

Patient-level data: De-identified information, including age, postcode, specimen type, chlamydia result (females and males), gonorrhoea result (males only), and service type, was collected.

HPV detection and genotyping: All samples were tested for HPV DNA at the Centre for Women's Infectious Diseases Molecular Microbiology Laboratory, Royal Women's Hospital, Melbourne, using the Seegene Anyplex HPV28 assay, as previously described²³.

Data linkage: Participating laboratories (except two that declined) securely transferred approved identifiers for the collected specimens to the AIHW via encrypted, password-protected files. HPV test results from the Royal Women's Hospital were sent directly to AIHW via a secure messaging service. AIHW linked specimen data to HPV vaccination records from AIR. Personal identifiers were removed and replaced with a new project-specific person number. The final linked dataset was stored in a secure research access environment for analysis. Vaccination status was categorised as: confirmed vaccinated (record in AIR with HPV vaccine doses), confirmed unvaccinated (record in AIR without HPV vaccine doses), and unknown (no matching record in AIR).

Data analysis: HPV prevalence estimates were calculated for each HPV type grouping by dividing the number of individuals who tested positive for at least one HPV type within the group by the total number of individuals with valid HPV test results. A person could be counted towards more than one HPV group. Socioeconomic status quintiles and remoteness area, derived according to SEIFA IRSD for 2021¹²⁵ and the Accessibility/Remoteness Index of Australia classification from the 2016 Census, based on the individual's postcode¹²².

Monitor the incidence of genital warts (Objective 3.1)

Data source: Data were extracted from ACCESS, a collaboration between the Burnet Institute, the Kirby Institute, and NRL. This is a national, sentinel surveillance network for monitoring, testing, treatment, and management of sexually transmissible infections and blood-borne viruses¹²⁰.

Data from 2006–2024 were extracted from 49 sexual health clinics participating in ACCESS. Data were de-identified and extracted from the electronic medical record using GRHANITE™, which enabled patient linkage across the sentinel network. Genital wart diagnoses were identified from the clinical diagnosis field recorded within the patient management systems of sexual health

clinics. Data were presented as the proportion of clients who received a genital wart diagnosis at their first-ever visit to an ACCESS sexual health clinic. These proportions were stratified into three groups: females, heterosexual males, and GBMSM. Within each group, data were further disaggregated by the patient postcode's socioeconomic status and remoteness, as well as by age group. SEIFA and ASGS remoteness structure were used to assign socioeconomic status and geographic areas^{120,122,125}.

Aboriginal or Torres Strait Islander identification was based on whether a person had ever self-reported their status, as recorded in the clinic records. Data for Aboriginal or Torres Strait Islander males and females were presented by age group.

Monitor the incidence of recurrent respiratory papillomatosis (Objective 3.2)

Data source (new cases): Incident cases of JoRRP are reported annually in the full here Australian Paediatric Surveillance Unit (APSU) Annual Surveillance Report⁵⁴.

Data source (genotyping of JoRRP cases): Data were obtained from Novakovic et al⁴⁵ and the APSU.

Data source (hospitalisations): AIHW collates clinical and demographic data on hospital admissions in public and private hospitals across Australia, recorded in the NHMD, back to 1993. All hospitalisation episodes with a principal diagnosis of benign neoplasm of the larynx were identified using the International Statistical Classification of Diseases and Related Health Problems, Tenth Revision, Australian Modification (ICD-10-AM) diagnosis code D14.1. As these data are de-identified, they may include repeat admissions for the same individuals¹²⁶.

All hospitalisation episodes recorded in the NHMD between 2011 and 2021 in Aboriginal and Torres Strait Islander children (aged 0–14 years) with a principal diagnosis of benign neoplasm of the larynx were also identified using the ICD-10-AM diagnosis code D14.1. As these data are de-identified, they may include repeat admissions for the same individuals.

Incidence and hospitalisation rates: For the national population of males and females aged 0–14 years, crude annual incidence and hospitalisation rates were calculated for each year from 2001 to 2023 by dividing the number of hospitalisations by the respective population estimates and multiplying by 100,000. National population denominators were sourced from the ABS¹²¹.

For Aboriginal and Torres Strait Islander children, crude annual incidence and hospitalisation rates were calculated using the 'Estimated Resident Population, Aboriginal and Torres Strait Islander Australians, Sex and Age Groups by States and Territories and Australia, 2011 to 2021' dataset from the ABS¹²⁷, which is based on the 2021 Australian Census and demographic projections.

Monitor the prevalence of screen-detected cervical abnormalities (Objective 3.3)

Data source (secondary use of data from the 2025 Cervical Cancer Elimination Report⁷⁶): Analysis of NCSR data (NCSR RDE 11/07/2025); data prepared by the Screening Analysis and Monitoring Unit of the Australian Institute of Health and Welfare¹¹⁵.

Where reported, rates were age-standardised using the Australia 2001 standard population¹¹⁹.

Aboriginal or Torres Strait Islander identification was based on whether a person had ever self-reported their status, as recorded in the NCSR.

Monitor the distribution of HPV genotypes detected in high-grade cervical lesions (Objective 3.4) and cervical cancer (Objective 4.3)

Data source: Analysis of NCSR data (NCSR RDE 13/01/2025); data prepared by the Screening Analysis and Monitoring Unit of the Australian Institute of Health and Welfare.

Objective 3.4:

Denominator: Count of people with high-grade histological abnormalities detected. High-grade abnormality is defined as HSIL NOS (S3.1), HSIL CIN2 (S3.2), HSIL CIN3 (S3.3), or AIS (E3.2, E3.3). If a person has more than one high-grade abnormality, the most serious abnormality is selected. Age is as at the time of the histology result.

Numerators: Count of results from all HPV tests over the 5 years preceding the high-grade histology result (2018–2023), as defined in **Table 5.1**.

Objective 4.4:

Denominator: Count of people with a first cancer histology result. Cancer histology results are defined as squamous cell carcinoma (SCC) (microinvasive & invasive) (HS04.1 or HS04.2), adenosquamous carcinoma (HE04.3), endocervical adenocarcinoma (microinvasive & invasive) (HE04.1 or HE04.2) or carcinoma of the cervix (other) (HE04.4). Age is at the time of the histology result. Excludes people who have ever had a previous cervical cancer histology result recorded (i.e., only the first detection of cervical cancer was included).

Numerator: Count of results from all HPV tests over the 5 years preceding the cancer histology result, as defined in **Table 5.1**. Data were reported as proportions nationally and by age group and type of high-grade abnormality. Aboriginal or Torres Strait Islander identification was based on whether a person had ever self-reported their status, as recorded in the NCSR.

Table 5.1 Definitions of HPV groups

Category	Definition
No tests available	No satisfactory HPV test in the five years before histology.
HPV never detected	At least one satisfactory test; all test results were negative for HPV.
HPV detected	At least one test was positive for any oncogenic HPV type.
Only HPV16/18 detected	At least one test was positive for HPV 16 and/or 18; no tests were ever positive for any other oncogenic type.
Only other oncogenic types detected	At least one test was positive for non-16 and 18 oncogenic types; no tests were ever positive for HPV 16 or 18.
HPV16/18 and other types detected	At least one test was positive for both 16 or 18 and non-16 and 18 types, either in the same test or on different testing occasions.

Monitor cervical, anogenital and oropharyngeal cancer incidence and mortality (Objective 4.1 and Objective 4.2)

Data source: Incidence and mortality data were obtained from the Australian Cancer Database (ACD), supplied by the AIHW, for the years 2001–2020. The ACD was used instead of the National Mortality Database to enable classification of cancer deaths by histological type. Raw counts of cases and deaths were provided by five-year age group and sex for both the national population and the Aboriginal and Torres Strait Islander population.

For Indigenous data, reporting was limited to the years 2008–2020.

Cancer classification

Cancer cases and deaths were classified using ICD-10 site codes and ICD-O-3.2 histology codes:

Cancer type	ICD-10 Codes	ICD-O-3.2 Histology Codes
Cervical cancer	C53	Squamous cell carcinoma: 8050–8086, 8120–8131 Adenocarcinoma: 8140–8149, 8160–8163, 8190–8231, 8250–8552, 8570–8574, 8576, 9110–9111 Adenosquamous carcinoma: 8560 Other types: All other codes excluding 9050–9053, 9140, 9590–9993
Anal cancer	C21	Same histology codes as above for squamous and adenocarcinomas
Vulvar cancer	C51	Squamous cell carcinoma: 8050–8086, 8120–8131
Vaginal cancer	C52	Squamous cell carcinoma: 8050–8086, 8120–8131
Penile cancer	C60	Squamous cell carcinoma: 8050–8086, 8120–8131
Oropharyngeal cancer (HPV-associated sites) ⁹⁸	C01, C02.4, C05.1–C05.2, C09–C10, C14.2	Squamous cell carcinoma: 8050–8086, 8120–8131
Head and neck cancer (non-HPV-associated sites) ⁹⁸	C00.3–C00.5, C02.0–C02.3, C03–C04, C05.0, C06	Squamous cell carcinoma: 8050–8086, 8120–8131

Note: Squamous cell carcinoma includes basaloid and cloacogenic carcinomas (8123–8124, 8083), grouped due to inconsistent histological classification and low case numbers. “Other” cervical cancer types were included in overall cervical cancer totals but not reported separately.

Rate calculations

- Age-specific incidence and mortality rates were calculated in five-year age groups.
- Age-standardised rates were calculated using the direct method and the 2001 Australian standard population, expressed per 100,000 persons.
- For national data, annual rates were produced for individual calendar years and pooled for 2016–2020.
- For the Aboriginal and Torres Strait Islander peoples, only the pooled 2016–2020 estimate was reported.

Population denominators

Population estimates used in rate calculations were sourced from the ABS:

- National population: **ABS National and State Population**
- Indigenous population: **ABS Indigenous Population Estimates**

Aboriginal and Torres Strait Islander population rates were restricted to states and territories with adequate identification in cancer registries:

- NSW (from 1999), Victoria (from 2008), Queensland (from 1997), Western Australia (from 1982), ACT (from 2000), and NT (from 1982).
- South Australia and Tasmania were excluded.

Rates were reported to one decimal place to mitigate small cell count disclosure risks. A rate of 0.0 does not necessarily indicate zero cases but may reflect a value below the rounding threshold.

Monitor the distribution of HPV genotypes detected in anogenital and oropharyngeal cancers (Objective 4.4)

Data source: Counts of histology codes 8085 (HPV-positive SCC) and 8086 (HPV-negative SCC) were obtained from the Australian Cancer Database, supplied by the AIHW, for the years 2001–2020, by cancer type.

6.

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6. REFERENCES

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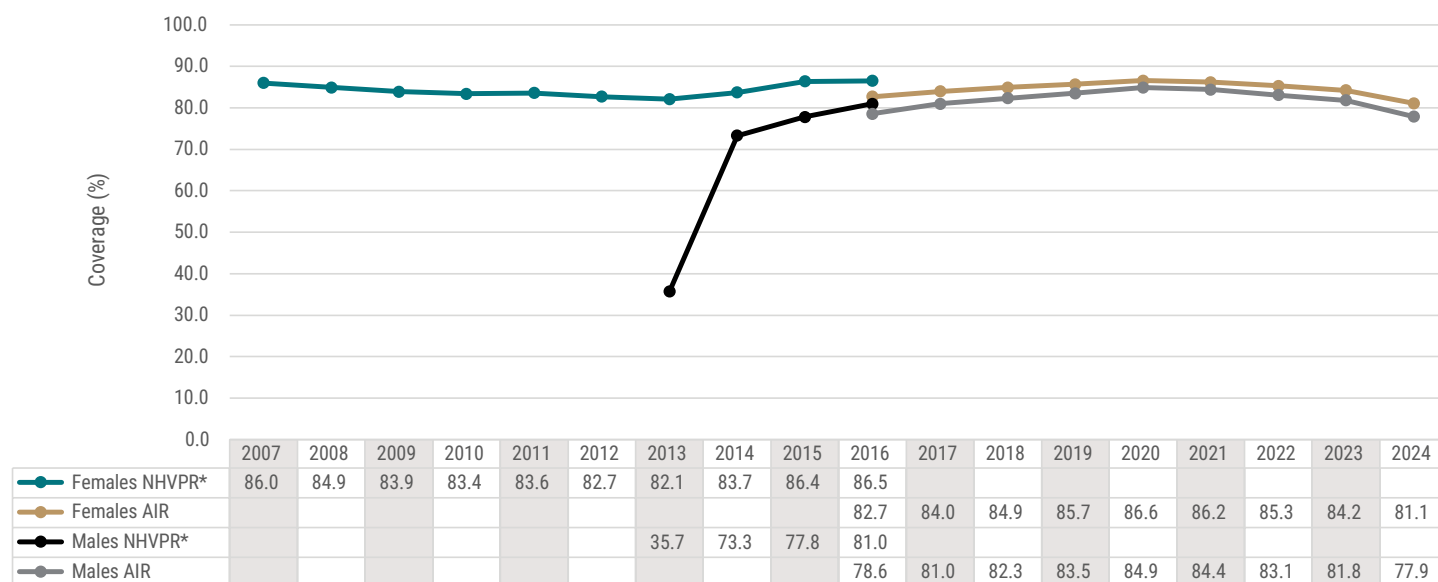
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APPENDIX

7. APPENDIX

Figure A.1 Historical HPV vaccine coverage estimates (≥ 1 dose) for females and males by age 15 in Australia, 2007–2024



Source: Brotherton et al. 2017 analysis of National HPV Vaccination Program Register (NHVPR) data (2007–2016)¹²⁸ and Australian Immunisation Register (2016–2024).

Table A3.1 Historical rates of histologically confirmed high-grade cervical abnormality (HGA) per 1,000 females screened nationally, 1997–2024

	High grade ^a (AS) (data from table s4.8)	High grade ^a (AS) (data from 2025 cervical cancer elimination report)	CIN3 (AS) (data from 2025 cervical cancer elimination report)	AIS (crude)(data from 2025 cervical cancer elimination report)
1997	6.4	NA	NA	NA
1998	6.2	NA	NA	NA
1999	6.9	NA	NA	NA
2000	6.9	NA	NA	NA
2001	6.9	NA	NA	NA
2002	7.5	NA	NA	NA
2003	7.5	NA	NA	NA
2004	7.7	NA	NA	NA
2005	7.7	NA	NA	NA
2006	7.8	NA	NA	NA
2007	7.7	NA	NA	NA
2008	8.3	NA	NA	NA
2009	8.1	NA	NA	NA
2010	8.5	NA	NA	NA
2011	8.4	NA	NA	NA
2012	8.4	NA	NA	NA
2013	8.5	NA	NA	NA
2014	8.1	NA	NA	NA
2015	7.8	NA	NA	NA
2016	7.4	NA	NA	NA
Jan–Jun 2017	7.1	NA	NA	NA
2018*	-	8.4	4.9	0.3
2019*	-	9.8	5.7	0.4
2020	-	16.8	9.6	0.6
2021	-	16.8	9.8	0.7
2022	-	14.4	8.6	0.5
2023	-	7.8	4.6	0.3
2024	-	8.4	4.3	0.2

Source: [2025 Cervical Cancer Elimination Progress Report](#) for 2018–2024 and [Cervical screening in Australia 2019](#) for 1997–2017 (Table S4.8). 1997–2017: for 20–69 year olds. 2018–2024: for 25–74 year olds. Age-standardised (AS) rate is the number of participants with a high-grade abnormality detected on histology per 1,000 participants screened, age-standardised to the 2001 Australian population. *Note: There was likely under-notification of these data to the NCSR in 2018 and 2019. *For data from 1997–2017: High-grade abnormality detection is defined as the number of women who have a high-grade abnormality detected per 1,000 females screened; a high-grade abnormality is defined as a histology result of HS03 (CIN NOS, CIN II and CIN III) or HE03 (endocervical dysplasia and adenocarcinoma in situ). *For data from 2018–2024: High-grade cervical disease was defined as a histology result of S3.1 (High-grade intraepithelial lesion (HSIL) (CIN NOS)), S3.2 (HSIL (CIN 2)), S3.3 (HSIL (CIN 3)) or E3.1 (Endocervical dysplasia), E3.2 (Adenocarcinoma in situ) or E3.3 (Mixed carcinoma in situ/adenocarcinoma in situ).

Table A3.2 Prevalence (%) of HPV by type among people with a cervical high-grade histological abnormality, overall and by histology type, 2018–2023

	Overall	2018	2019	2020	2021	2022	2023
All ages	Known HPV status	87.9	98.1	98.9	98.7	98.7	98.7
	Unknown HPV status	12.1	1.9	1.1	1.3	1.3	1.3
25–74	Known HPV status	88.2	98.1	98.9	98.8	98.8	98.9
	Unknown HPV status	11.8	1.9	1.1	1.2	1.2	1.1
All ages	HPV detected	97.5	98.6	98.7	98.5	98.4	98.2
	HPV never detected	2.5	1.4	1.3	1.5	1.6	1.8
25–74	HPV detected	97.7	98.6	98.8	98.6	98.5	98.2
	HPV never detected	2.3	1.4	1.2	1.4	1.5	1.8
All ages[†]	Only HPV16/18	43.4	36.6	26.8	26.4	24.0	22.6
	Only non HPV16/18 types	51.5	57.6	63.1	61.7	63.6	66.0
	Both HPV16/18 and other	5.1	5.8	10.1	12.0	12.4	11.4
25–74[†]	Only HPV16/18	46.4	38.5	27.8	27.2	24.7	23.2
	Only non HPV16/18 types	48.3	55.5	61.9	60.6	62.6	65.2
	Both HPV16/18 and other	5.2	6.0	10.3	12.2	12.6	11.6
	HSIL	2018	2019	2020	2021	2022	2023
All ages	Known HPV status	87.8	98.1	98.9	98.7	98.7	98.7
	Unknown HPV status	12.2	1.9	1.1	1.3	1.3	1.3
25–74	Known HPV status	88.1	98.1	98.9	98.8	98.8	98.9
	Unknown HPV status	11.9	1.9	1.1	1.2	1.2	1.1
All ages	HPV detected	97.5	98.5	98.7	98.5	98.4	98.2
	HPV never detected	2.5	1.5	1.3	1.5	1.6	1.8
25–74	HPV detected	97.7	98.6	98.8	98.6	98.5	98.2
	HPV never detected	2.3	1.4	1.2	1.4	1.5	1.8
All ages[†]	Only HPV16/18	41.7	34.9	25.2	24.7	22.3	21.0
	Only non HPV16/18 types	53.3	59.4	64.8	63.5	65.4	67.6
	Both HPV16/18 and other	5.0	5.7	10.0	11.9	12.2	11.3
25–74[†]	Only HPV16/18	44.7	36.7	26.2	25.4	23.1	21.6
	Only non HPV16/18 types	50.1	57.4	63.6	62.4	64.5	66.9
	Both HPV16/18 and other	5.2	5.9	10.2	12.1	12.4	11.5

Source: National Cancer Screening Register (see [methodology](#)). (a) Some rates based on numerators <10 or denominators < 20 and should be interpreted with caution; [†]Among those with a known HPV result over the 5 years preceding the cancer histology result; HSIL: high-grade squamous intraepithelial lesions; AIS: adenocarcinoma in situ.

Table A3.2 – Continues

	AIS	2018	2019	2020	2021	2022	2023
All ages	Known HPV status	90.2	97.6	98.3	98.9	97.8	98.2
	Unknown HPV status	9.8	2.4	1.7	1.1	2.2	1.8
25–74	Known HPV status	90.1	97.6	98.4	98.9	98.0	98.4
	Unknown HPV status	9.9	2.4	1.6	1.1	2.0	1.6
All ages	HPV detected	97.6	99.0	98.6	97.5	98.9	98.6
	HPV never detected	2.4	1.0	1.4	2.5	1.1	1.4
25–74	HPV detected	97.7	99.1	98.6	97.9	98.9	98.6
	HPV never detected	2.3	0.9	1.4	2.1	1.1	1.4
All ages¹	Only HPV16/18	83.9	77.8	67.5	67.6	69.1	64.4
	Only non HPV16/18 types	9.3	14.9	19.8	18.4	13.7	21.2
	Both HPV16/18 and other	6.8	7.2	12.7	14.0	17.1	14.4
25–74¹	Only HPV16/18	84.5	78.6	67.9	67.9	69.7	64.6
	Only non HPV16/18 types	8.9	14.3	19.4	18.0	13.4	21.1
	Both HPV16/18 and other	6.6	7.1	12.7	14.1	16.9	14.3

Source: National Cancer Screening Register (see [methodology](#)). (a) Some rates based on numerators <10 or denominators < 20 and should be interpreted with caution;

¹Among those with a known HPV result over the 5 years preceding the cancer histology result; HSIL: high-grade squamous intraepithelial lesions; AIS: adenocarcinoma in situ.

Table A3.3 Prevalence (%) of HPV by type among people with a high-grade histological abnormality, by age group, 2018–2023

		2018	2019	2020	2021	2022	2023
25–29^a	Only HPV16/18	28.3	21.1	13.9	12.7	10.9	10.2
	Only non HPV16/18 types	67.5	73.9	77.9	78.1	79.6	81.6
	Both HPV16/18 and other	4.2	5.0	8.2	9.2	9.6	8.2
30–34^a	Only HPV16/18	48.4	40.6	27.5	25.4	21.4	18.1
	Only non HPV16/18 types	46.0	53.3	62.3	63.1	67.2	73.0
	Both HPV16/18 and other	5.6	6.2	10.1	11.5	11.3	8.9
35–39^a	Only HPV16/18	54.0	43.8	32.9	34.2	31.0	26.3
	Only non HPV16/18 types	41.0	50.9	57.3	54.2	56.7	61.8
	Both HPV16/18 and other	4.9	5.2	9.9	11.6	12.3	11.9
40–44^a	Only HPV16/18	55.9	47.0	38.0	37.0	34.5	30.9
	Only non HPV16/18 types	38.8	46.0	50.8	49.8	52.5	56.1
	Both HPV16/18 and other	5.3	7.0	11.2	13.2	13.0	12.9
45–49^a	Only HPV16/18	54.5	47.3	36.9	36.5	36.1	35.1
	Only non HPV16/18 types	39.4	45.3	51.3	46.1	48.3	49.1
	Both HPV16/18 and other	6.1	7.4	11.9	17.4	15.7	15.9
50–54^a	Only HPV16/18	54.4	47.0	35.1	35.2	33.2	34.2
	Only non HPV16/18 types	38.6	45.4	50.4	47.8	48.8	47.3
	Both HPV16/18 and other	6.9	7.5	14.5	17.0	18.1	18.5
55–59^a	Only HPV16/18	58.9	47.8	34.8	30.5	30.5	35.4
	Only non HPV16/18 types	34.9	44.1	50.9	52.8	50.7	47.2
	Both HPV16/18 and other	6.1	8.1	14.3	16.7	18.8	17.3
60–64^a	Only HPV16/18	56.2	50.9	33.7	34.6	30.4	29.6
	Only non HPV16/18 types	38.2	43.6	53.2	49.6	49.2	51.7
	Both HPV16/18 and other	5.6	5.6	13.1	15.8	20.4	18.7
65–69^a	Only HPV16/18	51.4	49.8	35.9	33.2	34.4	32.6
	Only non HPV16/18 types	39.1	45.4	50.8	54.5	46.4	52.4
	Both HPV16/18 and other	9.4	4.8	13.4	12.3	19.1	15.0
70–74^a	Only HPV16/18	54.8	47.4	39.5	28.3	34.8	41.0
	Only non HPV16/18 types	38.7	48.3	51.9	55.9	50.8	47.0
	Both HPV16/18 and other (a)	6.5	4.3	8.5	15.7	14.4	11.9

Source: National Cancer Screening Register (see [methodology](#)). (a) Some rates based on numerators <10 or denominators < 20 and should be interpreted with caution.

^aAmong those with a known HPV result over the 5 years preceding the high-grade histology result.

Table A3.4 Prevalence (%) of HPV by type among Aboriginal and Torres Strait Islander peoples with a cervical high-grade histological abnormality, overall and by age group, 2018–2023

		2018	2019	2020	2021	2022	2023
All ages	Known HPV status	86.9	98.6	99.0	99.4	99.0	99.6
	Unknown HPV status (a)	13.1	1.4	1.0	0.6	1.0	0.4
25–74	Known HPV status	87.0	98.7	98.9	99.3	99.2	99.6
	Unknown HPV status (a)	13.0	1.3	1.1	0.7	0.8	0.4
All ages	HPV detected	96.7	98.3	98.7	98.7	98.8	97.7
	HPV never detected (a)	3.3	1.7	1.3	1.3	1.2	2.3
25–74	HPV detected	97.0	98.5	98.8	98.8	98.8	97.6
	HPV never detected (a)	3.0	1.5	1.2%	1.2%	1.2%	2.4%
All ages[†]	Only HPV16/18	37.5	34.1	26.6	26.7	24.8	23.6
	Only non HPV16/18 types	58.3	60.2	61.9	63.0	66.7	65.9
	Both HPV16/18 and other	4.2	5.7	11.5	10.2	8.5	10.5
25–74[†]	Only HPV16/18	41.1	36.6	28.5	27.6	25.2	23.9
	Only non HPV16/18 types	54.9	57.9	60.1	61.7	66.4	65.4
	Both HPV16/18 and other	4.0	5.6	11.5	10.7	8.5	10.7
25–34[†]	Only HPV16/18	31.0	32.9	19.4	21.8	19.2	12.4
	Only non HPV16/18 types	64.8	64.1	72.1	69.6	75.8	79.2
	Both HPV16/18 and other	4.2	3.1	8.5	8.5	5.0	8.5
35–44[†]	Only HPV16/18	55.9	34.3	35.2	35.5	31.1	32.1
	Only non HPV16/18 types	40.2	56.2	46.9	53.3	57.4	58.2
	Both HPV16/18 and other (a)	3.9	9.5	17.9	11.2	11.5	9.7
45–54[†]	Only HPV16/18	57.1	52.1	44.8	33.8	38.6	43.1
	Only non HPV16/18 types	40.2	56.2	46.9	53.3	57.4	58.2
	Both HPV16/18 and other (a)	3.9	9.5	17.9	11.2	11.5	9.7
55–64[†]	Only HPV16/18 (a)	57.6	47.1	43.6	28.6	36.7	48.0
	Only non HPV16/18 types	39.4	44.1	43.6	60.7	40.0	28.0
	Both HPV16/18 and other (a)	3.0	8.8	12.8	10.7	23.3	24.0
65–74[†]	Only HPV16/18 (a)	50.0	57.1	61.5	33.3	25.0	27.3
	Only non HPV16/18 types	50.0	42.9	38.5	33.3	62.5	63.6
	Both HPV16/18 and other (a)	0.0	0.0	0.0	33.3	12.5	9.1

Source: National Cancer Screening Register (see [methodology](#)). (a) Some rates based on numerators <10 or denominators < 20 and should be interpreted with caution.

[†]Among those with a known HPV result over the 5 years preceding the cancer histology result.

Table A4.1 Incidence rates of cervical cancer in females nationally per 100,000 estimated resident population, 2001–2020

		2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020
All cervical cancer	All ages	7.5	6.9	7.2	7.0	7.1	6.8	7.0	7.2	6.9	7.3	7.0	7.5	6.9	7.4	6.7	7.2	6.7	7.3	7.3	7.5
	25–74 years	10.7	9.9	10.4	9.9	10.2	9.9	10.4	10.4	10.2	10.7	10.5	11.2	10.3	11.2	10.1	10.9	10.2	11.3	11.3	11.8
	Age group																				
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.3	0.0	0.1	0.4	0.3	0.0	0.1	0.1	0.0	0.0	0.0	0.3	0.0	0.1	0.0	0.0	0.0	0.3	0.0	0.0
	20–24	1.3	2.2	0.9	2.4	1.9	1.5	0.7	1.6	1.6	2.8	1.5	1.9	1.9	1.5	1.3	1.0	0.5	0.5	0.8	0.1
	25–29	6.0	5.9	5.5	6.1	6.0	6.6	5.9	8.6	7.5	8.0	8.4	8.9	9.3	8.4	6.2	6.3	4.9	4.7	4.1	3.3
	30–34	8.0	10.1	8.5	11.8	10.9	10.7	9.7	10.7	11.9	10.0	12.8	12.4	12.6	12.3	12.2	12.3	11.6	13.0	14.3	12.2
	35–39	11.7	10.7	11.9	11.2	11.6	10.7	12.9	12.6	11.4	13.1	11.7	14.7	13.1	13.0	14.3	14.5	14.7	15.3	15.8	17.8
	40–44	9.2	10.2	10.8	10.2	14.2	10.6	11.4	12.6	10.1	10.1	11.9	13.3	12.9	15.5	11.7	13.6	14.8	14.8	15.6	18.2
	45–49	15.3	11.3	12.9	13.8	10.3	11.3	11.5	9.3	9.9	11.9	11.8	12.0	9.6	10.8	11.5	13.9	11.7	16.2	12.9	12.8
	50–54	12.0	10.7	11.1	8.5	8.9	9.1	11.0	9.8	11.8	9.6	8.5	10.1	9.1	10.2	8.5	8.7	8.7	9.5	8.8	13.7
	55–59	11.2	8.3	9.2	8.7	8.2	7.6	9.5	10.7	8.2	11.8	10.2	8.7	8.4	8.8	8.6	7.8	7.4	9.5	10.5	9.0
	60–64	11.6	10.1	11.5	7.6	11.2	11.3	10.4	7.2	10.2	9.9	8.3	7.5	7.3	10.0	8.6	9.7	7.9	8.2	9.1	7.8
	65–69	12.8	12.8	11.4	10.8	11.5	12.8	11.4	12.3	8.3	10.9	9.0	8.9	9.2	10.5	7.2	9.3	7.7	7.9	8.4	9.8
	70–74	11.4	10.3	13.2	8.7	9.0	10.2	10.8	9.4	12.0	12.2	10.5	11.2	7.0	10.1	8.3	9.7	6.8	7.3	9.4	7.4
	≥75	16.7	14.4	15.2	15.5	14.0	13.3	12.2	14.3	11.6	11.5	9.3	11.0	9.8	9.3	9.5	9.5	10.1	8.3	7.3	6.5
Cervical squamous cell carcinoma	All ages	5.1	4.9	4.9	4.8	4.7	4.3	4.5	4.7	4.5	4.8	4.6	4.9	4.5	4.8	4.6	4.6	4.4	4.5	4.5	4.8
	25–74 years	7.3	7.0	7.0	6.6	6.8	6.3	6.7	6.8	6.6	7.1	7.0	7.4	6.9	7.2	6.9	7.0	6.6	7.1	7.1	7.6
	Age group																				
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.4	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	1.3	1.9	0.8	1.8	1.4	0.8	0.3	1.1	1.2	1.8	1.3	1.0	0.7	1.2	1.1	0.7	0.5	0.5	0.6	0.1
	25–29	4.1	4.4	3.1	4.5	4.3	4.7	4.0	6.2	5.7	6.0	6.2	6.7	6.3	5.4	4.3	4.1	2.9	3.2	3.5	2.3
	30–34	6.7	6.3	5.8	7.5	6.9	6.9	6.3	6.2	7.3	7.1	8.7	8.7	8.5	7.9	7.9	8.0	9.0	8.2	8.8	8.0
	35–39	8.0	6.8	8.2	7.0	7.4	6.9	8.8	8.0	7.3	8.8	7.8	9.9	8.1	9.2	10.4	9.1	9.0	9.3	8.8	12.1
	40–44	5.7	6.8	7.3	6.2	10.3	6.4	7.5	8.5	7.2	6.1	6.9	8.8	8.1	10.0	6.9	8.8	9.2	9.8	9.4	11.6
	45–49	10.5	8.6	8.8	10.3	5.6	5.6	7.3	6.1	6.0	7.5	8.0	6.8	6.8	6.0	7.8	8.9	7.6	9.7	8.7	7.5
	50–54	8.7	7.5	7.7	5.0	6.2	6.3	6.4	7.2	8.3	6.8	6.2	6.2	6.3	7.0	5.7	5.1	5.8	5.7	5.7	8.4
	55–59	6.7	5.6	5.8	5.8	5.2	4.5	5.9	6.4	5.2	7.9	7.0	5.7	5.7	5.4	6.1	5.3	5.5	6.1	6.9	6.0
	60–64	7.2	7.7	8.7	5.8	6.9	6.6	6.3	5.0	5.0	6.4	5.9	4.9	4.5	5.8	6.3	7.3	4.7	5.9	5.6	5.5
	65–69	7.8	9.7	7.8	7.3	7.9	9.7	7.4	8.4	6.0	6.6	5.2	5.6	6.6	7.3	5.7	6.1	4.8	4.8	5.7	6.0
	70–74	8.4	9.1	9.2	5.9	7.5	7.1	7.5	5.6	7.1	8.3	6.2	7.8	4.8	6.7	5.3	5.3	3.7	4.0	4.2	4.2
	≥75	12.0	10.0	10.6	11.7	9.1	7.6	7.2	8.8	7.4	7.6	6.0	7.1	6.7	5.7	6.6	5.9	6.3	4.5	4.3	3.9

Source: Australian Cancer Database (see [methodology](#)). Overall age categories standardised to the 2001 Australian population.

Table A4.1 – Continues

		2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020
Cervical adeno- carcinoma	All ages	1.4	1.4	1.5	1.6	1.5	1.7	1.8	1.8	1.7	1.6	1.7	1.9	1.7	1.9	1.6	2.1	1.8	2.0	2.1	2.1
	25–74 years	2.1	2.2	2.2	2.4	2.4	2.6	2.7	2.7	2.7	2.5	2.7	2.9	2.6	2.9	2.5	3.3	2.8	3.2	3.4	3.3
	Age group																				
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.3	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.0	0.3	0.2	0.3	0.1	0.3	0.1	0.4	0.4	0.6	0.3	0.5	0.5	0.1	0.2	0.2	0.0	0.0	0.2	0.0
	25–29	1.4	1.0	1.8	1.2	0.9	1.3	1.1	1.9	1.4	1.2	2.1	1.2	2.3	2.3	1.7	2.0	1.6	1.1	0.6	0.6
	30–34	1.2	3.1	2.0	3.7	2.6	3.1	3.3	4.0	3.9	2.5	2.7	2.9	3.0	3.4	3.4	3.8	2.3	4.3	4.9	3.4
	35–39	2.5	3.3	2.7	2.7	2.8	2.9	3.1	3.1	3.3	3.5	3.2	4.0	4.1	3.1	3.3	5.0	4.8	4.7	6.2	4.1
	40–44	2.2	2.4	2.7	3.6	2.9	2.8	2.8	3.2	2.2	2.8	4.2	3.8	4.1	4.2	3.5	4.5	5.1	4.1	4.5	5.9
	45–49	2.9	2.0	3.0	2.5	3.8	4.2	3.0	2.3	3.1	3.1	2.6	3.7	1.7	3.8	2.8	3.8	3.0	4.6	3.8	4.5
	50–54	2.2	2.6	1.7	1.8	1.8	1.9	2.7	1.3	2.4	1.8	2.0	3.1	2.0	2.3	2.0	3.2	1.9	3.1	2.2	4.0
	55–59	2.0	1.9	2.1	2.0	2.3	1.9	2.7	3.6	2.3	2.9	2.1	1.4	2.3	1.8	1.8	1.9	1.2	1.8	2.0	2.3
	60–64	3.2	1.9	1.4	1.3	2.4	3.3	3.4	1.8	3.5	2.5	1.3	1.8	2.4	2.7	1.8	2.2	2.6	1.7	2.5	2.0
	65–69	2.3	2.3	3.0	1.6	2.1	1.8	2.5	3.1	1.4	1.5	2.5	2.7	1.3	2.5	1.2	2.0	1.8	2.1	2.4	3.1
	70–74	1.8	0.6	1.2	2.5	0.9	1.9	2.4	2.1	2.9	2.2	3.2	3.1	1.0	1.7	1.4	2.4	2.5	2.3	2.8	1.6
	≥75	1.9	1.3	2.7	2.1	1.5	2.9	2.4	2.7	1.7	1.4	1.2	1.6	1.2	2.1	1.7	1.9	2.1	1.9	1.4	0.9
Cervical adeno- squamous carcinoma	All ages	0.4	0.2	0.3	0.3	0.2	0.2	0.2	0.2	0.2	0.3	0.3	0.2	0.2	0.3	0.1	0.1	0.2	0.2	0.2	0.2
	25–74 years	0.6	0.3	0.5	0.4	0.4	0.3	0.4	0.3	0.4	0.4	0.4	0.3	0.3	0.4	0.2	0.2	0.3	0.3	0.3	0.2
	Age group																				
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.0	0.0	0.0	0.1	0.0	0.3	0.0	0.0	0.0	0.3	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	25–29	0.3	0.1	0.1	0.1	0.4	0.3	0.3	0.3	0.1	0.2	0.1	0.2	0.5	0.5	0.0	0.2	0.3	0.4	0.0	0.0
	30–34	0.0	0.7	0.5	0.4	0.1	0.4	0.0	0.1	0.4	0.1	0.8	0.4	0.4	0.7	0.5	0.4	0.1	0.2	0.2	0.6
	35–39	0.5	0.0	0.5	1.2	0.7	0.3	0.6	0.5	0.6	0.6	0.5	0.5	0.3	0.1	0.4	0.1	0.4	0.3	0.3	0.2
	40–44	0.7	0.5	0.3	0.3	0.3	0.5	0.8	0.5	0.3	0.3	0.5	0.1	0.2	0.7	0.4	0.1	0.5	0.1	0.9	0.2
	45–49	1.3	0.1	0.4	0.1	0.3	0.5	0.3	0.4	0.1	0.5	0.5	0.6	0.4	0.5	0.3	0.5	0.2	0.9	0.1	0.1
	50–54	0.2	0.3	0.8	0.5	0.5	0.1	0.6	0.7	0.4	0.4	0.1	0.3	0.3	0.5	0.0	0.1	0.1	0.1	0.3	0.3
	55–59	1.4	0.4	0.4	0.5	0.0	0.5	0.5	0.0	0.3	0.3	0.6	0.4	0.3	0.4	0.0	0.3	0.4	0.4	0.1	0.5
	60–64	0.5	0.5	0.9	0.0	0.6	0.0	0.2	0.2	0.9	0.3	0.2	0.2	0.2	0.3	0.3	0.0	0.0	0.1	0.1	0.0
	65–69	0.6	0.0	0.0	1.1	0.8	0.3	0.5	0.2	0.2	1.3	0.4	0.0	0.0	0.4	0.0	0.0	0.7	0.2	0.0	0.0
	70–74	0.6	0.0	0.6	0.0	0.0	0.0	0.0	0.3	0.3	0.6	0.8	0.0	0.5	0.0	0.5	0.2	0.0	0.2	0.7	0.4
	≥75	0.3	0.1	0.4	0.0	0.5	0.1	0.1	0.5	0.3	0.0	0.4	0.1	0.4	0.3	0.1	0.0	0.0	0.1	0.0	0.2

Source: Australian Cancer Database (see [methodology](#)). Overall age categories standardised to the 2001 Australian population.

Table A4.2 Mortality rates from cervical cancer nationally per 100,000 estimated Australian residential population, 2001–2020

		2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020
All cervical cancer	All ages	2.6	2.1	2.2	1.9	2.0	2.0	1.7	2.0	2.0	1.9	1.8	1.8	1.8	1.7	1.8	1.9	1.7	1.6	1.6	1.5
	25–74 years	3.2	2.4	2.7	2.2	2.4	2.4	2.1	2.5	2.3	2.4	2.3	2.1	2.3	2.2	2.3	2.5	2.1	2.1	2.2	2.0
	Age group																				
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.3	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.0	0.3	0.0	0.0	0.1	0.3	0.1	0.0	0.0	0.0	0.3	0.0	0.1	0.0	0.1	0.1	0.0	0.0	0.0	0.0
	25–29	0.1	0.3	0.7	0.6	0.7	0.4	0.1	0.5	0.9	0.5	0.4	0.4	1.2	0.5	0.8	0.3	0.5	0.3	0.3	0.1
	30–34	1.5	0.8	1.7	0.9	1.1	0.9	0.8	1.2	0.3	0.3	0.7	0.9	0.9	1.3	0.5	1.2	0.7	1.4	1.2	0.8
	35–39	1.5	1.4	1.6	1.2	0.1	1.8	1.4	1.0	0.9	2.5	1.9	2.0	1.5	1.3	1.3	2.2	1.4	1.4	1.0	1.4
	40–44	2.3	1.6	1.8	1.6	2.5	3.1	2.0	1.6	1.3	2.1	2.1	1.7	1.4	2.1	1.9	2.8	2.1	2.7	2.5	3.0
	45–49	3.2	2.3	3.0	2.5	3.1	1.3	1.7	3.0	2.6	1.7	2.4	1.3	3.5	1.8	3.1	2.7	2.5	2.3	2.7	3.2
	50–54	3.6	4.8	2.5	2.3	3.6	2.4	3.9	3.3	3.6	3.4	1.7	3.1	2.2	2.2	3.5	2.8	3.2	1.4	2.9	2.8
	55–59	4.5	2.4	3.2	3.6	3.3	2.2	1.6	3.8	3.5	4.9	4.2	3.6	3.0	3.9	2.3	3.9	2.5	3.1	3.4	2.1
	60–64	6.4	4.6	4.9	3.4	4.5	5.7	5.3	3.8	4.2	3.0	4.7	4.4	3.0	4.5	2.9	2.7	3.1	2.9	3.2	3.0
	65–69	6.7	4.3	5.5	4.8	3.7	4.9	3.2	5.0	5.5	4.6	4.4	2.9	5.2	2.7	3.1	4.0	3.9	3.9	2.5	1.7
	70–74	8.7	4.9	7.4	5.0	4.0	4.6	4.2	5.6	4.3	5.0	4.9	3.6	4.0	5.5	7.1	5.7	3.7	4.8	3.9	3.3
	≥75	12.8	10.9	10.6	10.3	9.8	10.1	8.1	9.4	10.4	7.6	7.1	8.4	6.9	5.6	6.5	6.9	8.2	5.7	5.0	4.6
Cervical squamous cell carcinoma	All ages	1.2	1.1	1.1	1.0	1.1	1.1	1.3	1.2	1.2	1.3	1.3	1.2	1.1	1.1	1.2	1.2	1.2	1.0	1.0	0.9
	25–74 years	1.5	1.3	1.4	1.2	1.5	1.3	1.7	1.6	1.5	1.7	1.7	1.5	1.4	1.5	1.4	1.6	1.5	1.3	1.4	1.3
	Age group																				
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.0	0.3	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.3	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0
	25–29	0.0	0.3	0.6	0.3	0.4	0.3	0.1	0.3	0.5	0.1	0.4	0.2	0.7	0.1	0.4	0.1	0.2	0.3	0.2	0.1
	30–34	0.5	0.7	0.8	0.5	0.7	0.3	0.7	0.7	0.1	0.1	0.3	0.8	0.7	1.1	0.2	0.9	0.4	1.0	1.1	0.5
	35–39	0.8	0.4	0.8	0.8	0.0	0.4	1.7	0.5	0.7	2.0	1.3	1.0	0.8	1.2	0.9	1.5	1.1	1.0	0.6	0.9
	40–44	1.2	0.7	1.0	1.2	1.3	2.2	1.7	0.8	0.7	1.9	1.6	1.1	0.7	1.1	1.4	1.8	1.7	1.4	1.6	1.6
	45–49	1.2	1.5	2.1	1.7	1.9	0.7	1.7	2.1	2.3	1.1	1.8	1.3	2.3	0.9	2.0	1.5	1.9	2.2	1.9	2.2
	50–54	2.2	2.6	1.1	1.4	2.1	1.6	2.6	2.4	2.8	2.6	1.3	1.9	1.3	1.9	2.4	1.9	2.3	0.5	2.2	2.1
	55–59	1.8	1.3	1.1	1.7	1.8	1.9	1.3	2.7	1.4	3.9	2.8	2.5	2.0	2.1	1.6	2.4	1.4	1.8	2.7	1.8
	60–64	3.7	1.9	2.1	1.8	2.8	2.5	3.4	2.0	3.5	2.0	3.7	2.9	2.1	2.5	1.4	2.1	2.3	2.0	1.5	1.6
	65–69	2.9	2.6	1.9	2.4	2.6	2.6	2.2	4.6	3.0	1.7	2.9	2.3	2.8	2.0	2.2	2.3	2.8	1.8	1.7	0.6
	70–74	3.6	2.4	4.9	1.9	3.4	2.5	2.7	2.9	3.1	2.2	3.2	2.6	2.8	4.8	3.5	3.3	2.5	2.9	1.7	1.2
	≥75	4.8	5.8	5.4	5.0	4.6	5.7	4.7	5.0	5.5	5.4	4.4	4.9	3.7	4.2	5.0	4.4	4.8	2.8	2.5	3.2

Source: Australian Cancer Database (see [methodology](#)). Overall age categories standardised to the 2001 Australian population.

Table A4.2 – Continues

		2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020
Cervical adeno- carcinoma	All ages	0.4	0.3	0.3	0.2	0.2	0.3	0.3	0.3	0.3	0.3	0.3	0.4	0.4	0.4	0.3	0.4	0.4	0.4	0.3	0.3
	25–74 years	0.5	0.4	0.4	0.3	0.2	0.4	0.3	0.4	0.3	0.4	0.3	0.5	0.5	0.5	0.4	0.6	0.5	0.6	0.5	0.4
	Age group																				
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	25–29	0.0	0.0	0.0	0.3	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.1	0.0	0.2	0.0	0.2	0.1	0.0	0.1	0.0
	30–34	0.3	0.0	0.5	0.3	0.0	0.1	0.3	0.3	0.0	0.0	0.1	0.1	0.0	0.2	0.0	0.2	0.1	0.4	0.0	0.1
	35–39	0.4	0.3	0.1	0.1	0.0	0.7	0.1	0.4	0.1	0.2	0.4	0.3	0.6	0.0	0.0	0.7	0.4	0.1	0.4	0.3
	40–44	0.4	0.5	0.0	0.0	0.4	0.3	0.0	0.5	0.1	0.0	0.2	0.4	0.5	0.6	0.4	0.4	0.4	0.9	0.2	0.7
	45–49	0.3	0.3	0.4	0.1	0.1	0.3	0.4	0.1	0.0	0.6	0.4	0.1	0.8	0.5	0.5	0.5	0.6	0.1	0.6	0.2
	50–54	0.5	0.5	0.6	0.3	0.3	0.3	0.7	0.1	0.3	1.0	0.1	0.9	0.5	0.4	0.6	0.6	0.5	0.8	0.6	0.9
	55–59	0.6	0.6	0.5	0.5	0.7	0.2	0.2	0.5	1.1	0.8	0.3	0.9	0.4	1.3	0.5	0.7	0.8	0.8	0.6	0.3
	60–64	0.7	0.7	1.4	0.4	0.6	0.8	0.9	1.3	0.3	0.7	1.0	1.1	0.3	1.1	0.8	1.0	0.6	0.7	1.1	0.8
	65–69	2.3	0.3	1.1	0.5	0.5	1.0	0.5	0.7	1.6	0.9	0.2	0.6	1.5	0.7	0.3	1.0	0.8	1.6	0.3	0.6
	70–74	1.2	0.9	0.0	1.2	0.0	0.6	0.9	1.5	1.1	0.3	0.5	1.6	1.0	1.0	1.4	0.7	1.2	1.2	1.5	0.9
	≥75	0.9	1.0	1.0	0.8	1.0	1.7	1.6	1.4	1.9	1.1	1.3	1.1	1.4	1.5	1.0	1.3	1.4	1.3	0.6	1.1
Cervical adeno- squamous carcinoma	All ages	0.2	0.1	0.1	0.1	0.1	0.0	0.1	0.1	0.1	0.1	0.0	0.1	0.1	0.1	0.1	0.1	0.1	0.0	0.1	0.1
	25–74 years	0.3	0.2	0.1	0.1	0.1	0.0	0.1	0.2	0.2	0.2	0.0	0.1	0.1	0.1	0.1	0.1	0.1	0.0	0.1	0.1
	Age group																				
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	25–29	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.1	0.1	0.1	0.1	0.0	0.0	0.0
	30–34	0.4	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.1	0.0	0.0	0.1	0.1
	35–39	0.0	0.3	0.0	0.3	0.0	0.0	0.0	0.1	0.0	0.1	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.1
	40–44	0.4	0.3	0.0	0.1	0.3	0.1	0.0	0.1	0.3	0.1	0.0	0.1	0.0	0.1	0.1	0.2	0.0	0.1	0.2	0.1
	45–49	0.6	0.3	0.0	0.0	0.3	0.0	0.0	0.1	0.1	0.0	0.0	0.0	0.3	0.3	0.1	0.4	0.1	0.0	0.0	0.0
	50–54	0.2	0.3	0.2	0.3	0.3	0.0	0.3	0.1	0.3	0.1	0.3	0.1	0.3	0.1	0.4	0.0	0.1	0.0	0.1	0.1
	55–59	0.6	0.2	0.2	0.0	0.2	0.0	0.2	0.2	0.5	0.3	0.0	0.3	0.3	0.1	0.0	0.3	0.1	0.1	0.1	0.1
	60–64	0.2	0.2	0.0	0.0	0.2	0.2	0.2	0.0	0.5	0.3	0.0	0.5	0.3	0.2	0.2	0.1	0.3	0.1	0.0	0.1
	65–69	0.0	0.0	0.6	0.0	0.0	0.0	0.7	0.2	0.5	0.7	0.0	0.2	0.2	0.0	0.2	0.2	0.2	0.0	0.3	0.2
	70–74	0.6	0.0	0.3	0.0	0.3	0.0	0.0	0.6	0.0	0.0	0.3	0.3	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0
	≥75	0.1	0.0	0.3	0.1	0.3	0.1	0.0	0.3	0.4	0.0	0.1	0.4	0.1	0.3	0.0	0.1	0.0	0.1	0.2	0.0

Source: Australian Cancer Database (see [methodology](#)). All ages category standardised to the 2001 Australian population.

Table A4.3 Anal cancer incidence rates in females per 100,000 estimated resident population, 2001–2020

		2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2016–2020 period
Anal squamous cell carcinoma	All ages	0.8	1.0	1.1	1.0	1.3	1.2	1.2	1.2	1.4	1.6	1.4	1.7	1.6	1.7	1.6	1.6	1.7	1.7	1.9	1.7	1.7
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	25–29	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.1	0.0	0.1	0.1	0.0	0.0	0.1	0.0	0.1	0.0	0.0	0.0	0.0
	30–34	0.0	0.4	0.0	0.0	0.1	0.3	0.3	0.1	0.0	0.0	0.1	0.1	0.2	0.0	0.2	0.1	0.0	0.3	0.1	0.0	0.1
	35–39	0.4	0.5	0.4	0.7	0.5	0.3	0.1	0.0	0.5	0.2	0.4	0.4	0.5	0.3	0.4	0.4	0.4	0.3	0.2	0.2	0.3
	40–44	0.7	0.4	1.2	0.8	1.2	1.2	0.9	1.2	1.1	1.7	1.0	0.6	0.8	1.1	0.7	1.3	1.7	0.9	0.9	0.4	1.0
	45–49	1.3	1.7	1.1	1.5	1.8	1.9	1.8	1.8	3.4	2.2	1.7	2.6	1.9	1.8	2.9	1.2	2.0	1.8	2.6	1.1	1.7
	50–54	1.6	1.6	2.0	1.8	3.3	2.7	3.2	3.1	5.3	3.1	3.0	2.9	2.8	3.7	3.4	3.1	2.7	3.0	3.3	2.6	2.9
	55–59	2.2	3.4	2.5	2.2	3.4	2.5	2.5	3.8	2.9	5.8	3.7	3.6	6.1	6.4	4.1	5.1	4.6	5.1	4.5	3.5	4.5
	60–64	1.7	1.9	3.5	2.7	4.5	3.5	4.4	3.0	2.2	5.2	3.9	4.2	4.5	4.4	6.1	4.0	6.9	5.6	7.7	7.7	6.4
	65–69	3.2	4.0	3.3	2.7	3.1	3.8	4.2	4.6	4.8	4.6	5.8	5.6	4.2	6.2	5.3	6.5	5.8	5.0	6.5	6.4	6.0
	70–74	1.8	3.0	3.1	4.3	4.0	4.0	2.7	2.1	2.0	3.3	4.3	8.1	5.0	7.7	4.4	5.7	6.6	7.5	7.9	6.3	6.8
	≥75	3.0	3.2	5.2	3.1	3.1	3.6	3.3	4.3	4.1	4.5	4.6	6.4	5.1	4.1	5.2	5.1	4.1	6.6	5.7	7.3	5.8
Anal adeno-carcinoma	All ages	0.3	0.2	0.2	0.2	0.3	0.3	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.3	0.2	0.2	0.2	0.2	0.2	0.2	0.2
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	25–29	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.1	0.0	0.0	0.0
	30–34	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
	35–39	0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.1	0.0	0.1	0.0	0.2	0.0	0.1	0.1	0.0	0.1
	40–44	0.3	0.1	0.1	0.1	0.1	0.0	0.3	0.0	0.0	0.0	0.1	0.0	0.1	0.0	0.0	0.0	0.0	0.2	0.1	0.0	0.1
	45–49	0.1	0.1	0.3	0.1	0.1	0.1	0.1	0.1	0.0	0.1	0.3	0.0	0.1	0.4	0.0	0.1	0.0	0.1	0.1	0.1	0.1
	50–54	0.5	0.0	0.0	0.2	0.8	0.4	0.1	0.4	0.3	0.1	0.0	0.4	0.3	0.3	0.4	0.1	0.0	0.0	0.4	0.4	0.2
	55–59	0.2	0.2	0.2	0.0	0.5	0.0	0.5	0.3	0.0	0.3	0.3	0.0	0.3	0.4	0.4	0.7	0.7	0.3	0.4	0.4	0.5
	60–64	1.2	0.2	0.2	0.9	0.2	1.4	0.2	0.5	0.7	0.2	0.8	0.5	0.5	1.1	1.1	0.6	0.1	0.1	0.7	0.8	0.5
	65–69	0.6	1.1	1.1	1.1	1.0	1.5	0.5	0.2	0.9	1.3	0.2	0.8	0.4	0.9	0.5	0.3	0.7	0.5	0.0	0.3	0.4
	70–74	1.8	0.0	0.6	1.2	1.2	0.6	0.6	0.9	1.4	1.1	1.3	0.5	0.5	0.7	0.7	1.3	0.6	0.8	0.0	0.7	0.7
	≥75	1.9	1.2	1.4	1.5	2.0	1.7	1.4	1.4	1.5	1.2	1.3	1.6	1.2	1.9	1.3	1.3	1.5	1.4	1.3	1.6	1.4

Source: Australian Cancer Database (see [methodology](#)). All ages category standardised to the 2001 Australian population.

Table A4.4 Anal cancer incidence rates in males per 100,000 estimated resident population, 2001–2020

		2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2016–2020 period
Anal squamous cell carcinoma	All ages	0.7	0.8	0.8	0.8	1.0	0.8	0.8	0.8	0.8	0.9	0.8	1.0	1.0	1.1	1.1	1.1	1.0	1.0	0.9	0.9	1.0
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0
	25–29	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.1	0.1	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
	30–34	0.6	0.3	0.1	0.0	0.0	0.0	0.3	0.1	0.0	0.5	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.3	0.1	0.0	0.1
	35–39	0.3	0.7	0.6	0.4	0.4	0.4	0.5	0.1	0.3	0.3	0.1	0.4	0.3	0.1	0.1	0.1	0.0	0.2	0.2	0.2	0.2
	40–44	0.3	0.0	0.8	0.5	0.9	1.1	0.7	0.8	0.5	0.5	0.5	0.6	0.5	0.2	0.4	0.1	0.3	0.5	0.4	0.6	0.4
	45–49	0.6	1.0	1.0	0.8	1.7	1.0	1.5	1.3	0.6	1.4	2.0	1.6	1.7	1.4	1.7	1.9	1.2	0.4	1.1	1.1	1.1
	50–54	0.8	1.4	1.9	2.1	1.7	1.2	1.0	2.6	2.4	1.2	2.6	1.6	2.1	3.0	2.2	2.0	2.6	2.7	1.5	1.7	2.1
	55–59	1.2	1.8	2.6	2.5	1.8	1.6	1.3	1.6	1.6	2.3	2.1	2.4	1.2	2.1	3.2	3.0	3.7	2.4	2.8	2.1	2.8
	60–64	3.2	1.9	1.8	3.3	2.6	1.4	2.8	1.4	1.7	4.0	2.6	3.3	2.9	4.3	4.3	3.6	3.2	1.7	3.4	2.9	2.9
	65–69	4.2	3.2	2.3	3.0	2.4	2.9	2.0	1.9	3.5	1.3	1.7	4.1	3.9	5.0	2.1	3.2	3.4	2.5	3.5	3.0	3.1
	70–74	2.0	3.6	1.3	2.0	4.7	3.7	2.3	2.2	3.0	2.9	2.2	2.7	2.9	2.8	5.8	4.3	3.0	5.6	4.0	3.9	4.2
	≥75	2.3	2.4	2.1	2.1	3.4	2.5	3.4	3.7	3.4	2.8	2.0	4.3	4.3	3.7	3.0	4.5	3.4	4.3	3.1	2.8	3.6
Anal adeno-carcinoma	All ages	0.4	0.3	0.2	0.3	0.4	0.3	0.4	0.3	0.4	0.3	0.4	0.4	0.4	0.3	0.4	0.3	0.3	0.3	0.3	0.4	0.3
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	25–29	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0
	30–34	0.3	0.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.0	0.0
	35–39	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.1	0.0	0.0	0.3	0.0	0.0	0.1	0.0	0.2	0.0	0.1	0.1	0.1
	40–44	0.1	0.0	0.0	0.1	0.1	0.0	0.1	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0
	45–49	0.0	0.3	0.0	0.1	0.3	0.1	0.1	0.3	0.4	0.0	0.5	0.1	0.3	0.0	0.1	0.1	0.4	0.0	0.6	0.6	0.3
	50–54	0.3	0.2	0.3	0.3	1.2	0.1	0.4	0.4	0.4	0.4	0.0	0.4	0.4	0.4	0.0	0.3	0.4	0.4	0.3	0.6	0.4
	55–59	1.0	0.2	0.2	0.3	0.3	0.6	0.5	0.8	0.8	0.6	0.9	0.4	1.0	0.6	1.1	0.1	0.8	0.5	0.7	0.3	0.5
	60–64	0.7	1.4	0.9	0.4	0.9	1.2	0.6	0.5	0.7	1.0	0.8	0.5	0.8	0.8	1.8	0.9	0.6	1.1	0.9	0.7	0.8
	65–69	2.1	0.3	1.7	1.1	1.3	1.8	1.3	1.7	1.6	0.9	0.6	1.4	1.9	0.9	1.0	0.8	1.4	1.2	1.2	1.6	1.2
	70–74	2.0	1.7	0.0	1.7	1.3	2.0	2.3	1.9	1.2	2.0	1.4	3.0	1.0	1.5	1.4	1.4	1.3	1.8	0.4	1.3	1.2
	≥75	3.0	2.7	1.7	2.9	3.0	1.5	3.2	1.5	2.9	1.6	3.1	2.6	2.2	1.7	2.7	2.6	1.3	2.6	1.9	2.0	2.1

Source: Australian Cancer Database (see [methodology](#)). All ages category standardised to the 2001 Australian population.

Table A4.5 Vulval and vaginal squamous cell carcinoma (SCC) incidence rates in females per 100,000 estimated resident population, 2001–2020

		2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2016–2020 period
Vulval squamous cell carcinoma	All ages	2.0	1.5	1.6	1.7	2.0	1.8	1.9	1.9	2.0	1.9	2.0	2.2	2.1	2.1	2.1	2.2	2.0	1.9	2.1	2.0	2.0
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0
	20–24	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	25–29	0.3	0.3	0.1	0.0	0.1	0.1	0.1	0.1	0.1	0.1	0.2	0.4	0.2	0.0	0.0	0.4	0.1	0.1	0.0	0.1	0.2
	30–34	0.4	0.3	0.1	0.8	0.4	0.3	0.7	1.0	0.5	0.4	0.4	0.1	0.2	0.1	0.3	0.3	0.3	1.0	0.1	0.5	0.4
	35–39	0.9	0.7	0.8	1.2	1.5	0.7	0.9	1.2	1.0	0.9	0.9	0.3	1.5	0.6	0.8	0.6	0.8	0.6	1.3	0.6	0.8
	40–44	2.2	1.2	0.9	1.4	1.2	1.4	1.3	1.5	1.3	1.8	1.1	1.2	1.0	1.7	1.6	0.9	1.2	1.1	1.2	0.2	0.9
	45–49	1.9	1.2	1.8	1.0	1.8	2.5	2.0	3.0	2.3	2.9	1.7	2.5	2.5	3.1	2.1	3.1	2.7	1.6	2.7	1.9	2.4
	50–54	1.2	2.2	1.7	2.9	2.7	2.4	3.0	2.7	4.2	3.7	3.6	4.2	2.8	2.8	4.0	3.4	2.9	2.4	3.8	3.4	3.2
	55–59	2.2	2.3	1.2	3.7	2.8	3.5	2.7	2.5	3.4	3.3	2.5	3.8	2.6	3.8	2.2	3.6	3.6	2.2	2.5	3.6	3.1
	60–64	3.2	2.4	3.3	1.8	4.3	2.9	3.0	3.4	4.3	3.5	3.7	3.9	4.1	5.1	4.7	5.4	4.5	4.4	3.8	4.9	4.6
	65–69	5.5	2.8	3.6	5.7	3.7	3.1	6.4	3.8	5.7	4.2	5.2	5.6	5.7	5.5	7.0	7.1	5.4	4.5	4.9	5.1	5.4
	70–74	8.4	6.7	4.6	6.2	5.3	5.3	4.2	5.6	5.7	6.7	7.6	6.0	8.0	8.9	6.2	7.7	8.2	5.6	8.1	8.1	7.5
	≥75	13.5	9.6	12.0	9.6	14.3	12.1	12.2	11.2	9.5	9.4	12.5	14.0	12.2	11.0	11.9	11.1	10.3	12.5	12.4	11.9	11.7
Vaginal squamous cell carcinoma	All ages	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.3	0.3	0.5	0.4	0.3	0.3	0.3	0.5	0.5	0.5	0.4	0.4	0.3	0.5
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	25–29	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.1	0.0	0.2	0.1	0.0	0.0	0.0	0.0	0.0
	30–34	0.1	0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.1	0.0	0.1
	35–39	0.0	0.1	0.0	0.3	0.0	0.1	0.1	0.0	0.2	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.2	0.1	0.1	0.1
	40–44	0.5	0.4	0.0	0.1	0.1	0.1	0.1	0.0	0.0	0.3	0.4	0.2	0.1	0.2	0.5	0.6	0.1	0.1	0.6	0.0	0.3
	45–49	0.0	0.1	0.1	0.4	0.3	0.4	0.0	0.1	0.3	0.1	0.5	0.1	0.1	0.1	0.5	0.2	0.5	0.1	0.2	0.2	0.3
	50–54	0.6	0.5	0.2	0.3	0.8	1.5	0.1	0.6	0.3	0.7	0.5	0.6	0.0	0.5	0.8	1.5	1.2	0.1	0.1	0.6	0.7
	55–59	0.2	0.6	0.7	0.7	0.3	0.2	1.1	0.5	0.2	1.5	0.9	0.9	0.4	0.4	1.0	1.2	0.5	0.9	0.9	0.3	0.7
	60–64	1.0	1.2	1.2	0.9	0.2	0.6	1.7	1.3	0.5	1.2	0.8	1.3	1.4	0.9	1.7	0.9	1.6	1.0	0.4	1.2	1.0
	65–69	1.5	0.6	0.3	1.6	1.8	0.8	0.7	1.2	1.6	1.3	1.7	1.2	1.8	1.2	1.5	1.7	1.0	1.9	2.5	0.8	1.6
	70–74	2.1	2.7	1.2	0.9	0.6	1.9	1.2	2.1	0.9	2.5	0.5	0.5	0.5	0.5	0.7	2.0	2.3	2.9	2.8	1.8	2.3
	≥75	2.2	2.0	3.4	3.2	3.4	2.1	2.9	1.8	1.7	2.2	2.0	1.9	2.0	2.1	2.4	2.3	3.5	2.6	1.3	1.9	2.3

Source: Australian Cancer Database (see [methodology](#)). All ages category standardised to the 2001 Australian population.

Table A4.6 Penile squamous cell carcinoma (SCC) incidence rates in males per 100,000 estimated resident population, 2001–2020

		2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2016–2020 period
Penile squamous cell carcinoma	All ages	0.8	0.7	0.6	0.6	0.7	0.8	0.7	0.8	0.6	0.6	0.9	0.8	0.8	0.9	0.8	0.9	0.9	1.1	0.8	0.9	0.9
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	25–29	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0
	30–34	0.1	0.1	0.3	0.5	0.4	0.1	0.0	0.1	0.1	0.0	0.0	0.1	0.1	0.4	0.1	0.1	0.2	0.2	0.1	0.1	0.2
	35–39	0.1	0.3	0.3	0.3	0.0	0.1	0.4	0.5	0.1	0.3	0.3	0.6	0.1	0.3	0.1	0.5	0.5	0.1	0.5	0.4	0.4
	40–44	0.4	0.4	0.9	0.4	0.5	0.4	0.7	0.1	0.3	0.4	0.5	0.2	0.2	0.6	0.2	0.6	0.5	0.3	0.4	0.4	0.4
	45–49	0.7	0.4	0.9	0.0	0.0	1.0	0.3	1.0	0.1	0.3	1.3	0.5	0.3	1.1	0.7	0.6	0.9	1.1	0.5	1.0	0.8
	50–54	1.1	0.6	0.9	0.5	0.6	0.6	0.1	0.7	0.7	0.7	0.5	0.8	0.7	1.2	1.6	0.8	0.9	0.9	1.3	1.0	1.0
	55–59	0.8	1.6	0.3	2.3	1.1	1.0	0.6	1.1	0.8	0.6	2.6	0.7	1.0	1.6	1.5	0.8	0.9	1.6	1.4	1.4	1.3
	60–64	2.4	2.6	1.6	2.2	1.5	0.8	1.3	1.1	1.6	1.5	1.3	2.1	1.5	1.6	1.8	1.6	2.2	2.7	2.4	1.7	2.1
	65–69	3.6	2.1	2.3	1.4	2.4	2.1	3.3	2.9	2.1	0.9	1.5	1.6	2.2	1.8	1.6	1.7	2.2	4.7	2.8	2.8	2.9
	70–74	3.6	4.0	1.3	1.0	2.4	3.3	2.6	4.7	2.1	3.2	5.0	4.1	3.9	4.0	2.9	3.7	3.2	3.0	1.7	2.8	2.8
	≥75	4.6	3.1	3.0	2.9	5.0	6.8	5.8	3.9	5.0	4.9	5.3	5.3	5.9	5.3	4.9	6.5	6.8	8.2	4.8	6.1	6.5

Source: Australian Cancer Database (see [methodology](#)). All ages category standardised to the 2001 Australian population.

Table A4.7 Oropharyngeal cancer (frequently associated with HPV*) incidence rates per 100,000 estimated resident population, 2001–2020

		2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2016–2020 period
Females – Squamous cell carcinoma	All ages	1.0	1.1	1.1	1.1	1.2	1.0	1.0	1.1	1.1	1.1	1.3	1.5	1.2	1.4	1.4	1.3	1.4	1.4	1.4	1.5	1.4
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.2	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	25–29	0.0	0.1	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.3	0.0	0.1	0.0	0.0	0.0	0.1	0.0
	30–34	0.1	0.0	0.0	0.0	0.0	0.3	0.0	0.0	0.3	0.1	0.3	0.6	0.2	0.2	0.2	0.0	0.2	0.1	0.0	0.2	0.1
	35–39	0.0	0.3	0.4	0.4	0.0	0.3	0.3	0.4	0.1	0.4	0.4	0.1	0.5	0.4	0.1	0.0	0.1	0.1	0.4	0.3	0.2
	40–44	0.4	0.4	0.8	0.6	0.4	0.5	0.8	0.4	0.4	1.2	0.9	0.7	0.5	0.8	0.6	0.6	1.0	0.5	0.5	1.5	0.8
	45–49	1.2	1.5	1.8	1.1	1.4	0.9	2.6	2.4	1.8	1.1	1.9	3.2	2.5	1.9	2.1	1.7	2.4	1.4	1.6	2.1	1.9
	50–54	2.0	2.5	1.7	2.3	2.6	2.8	2.6	2.0	2.6	3.1	3.4	3.6	2.3	3.5	3.3	3.2	3.5	2.3	3.8	2.8	3.1
	55–59	4.1	3.8	2.5	1.7	2.9	1.9	2.5	3.0	3.4	3.3	3.3	5.1	4.3	3.9	4.4	5.1	3.6	5.5	4.2	4.3	4.5
	60–64	3.7	2.9	4.0	5.2	4.5	4.3	2.7	2.5	4.5	3.5	2.3	3.2	4.5	5.5	6.7	3.7	2.9	6.0	4.5	5.7	4.6
	65–69	2.9	4.3	4.2	3.0	4.7	3.6	3.0	4.8	3.9	2.8	5.4	5.3	2.9	5.5	5.8	4.3	6.1	5.5	5.0	4.7	5.1
	70–74	4.2	4.2	2.8	4.0	5.0	2.2	1.8	3.5	3.7	4.2	5.4	4.4	4.5	3.4	3.2	4.2	4.5	4.4	4.8	4.0	4.4
	≥75	2.8	3.3	3.4	3.1	3.3	3.3	2.0	3.9	2.0	2.9	2.8	3.1	2.6	2.2	2.8	2.7	3.5	3.7	3.6	3.5	3.4
Males – Squamous cell carcinoma	All ages	3.6	4.1	4.1	3.9	4.3	4.5	4.5	4.7	5.0	5.3	5.8	6.2	6.4	6.9	6.9	7.4	7.6	7.8	8.1	7.5	7.7
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.0	0.0	0.1	0.0	0.1	0.0
	25–29	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.2	0.0	0.0	0.1	0.0	0.0	0.1	0.1	0.1	0.0	0.1	0.0	0.1
	30–34	0.1	0.0	0.1	0.0	0.1	0.1	0.3	0.1	0.0	0.5	0.4	0.1	0.1	0.1	0.3	0.3	0.2	0.0	0.0	0.2	0.2
	35–39	0.7	1.0	0.8	1.0	1.1	0.8	0.4	1.0	0.8	1.1	1.3	0.5	0.5	0.3	0.4	0.7	0.8	0.5	1.1	0.4	0.7
	40–44	3.2	4.0	3.3	3.0	3.2	2.9	3.6	2.8	1.9	3.7	3.3	3.7	4.3	4.6	3.2	3.2	2.9	1.9	2.6	3.1	2.8
	45–49	6.1	7.0	5.8	7.1	7.9	7.1	6.8	6.0	6.6	6.9	8.5	8.6	9.6	5.9	11.3	9.5	9.5	8.2	8.7	8.2	8.8
	50–54	7.4	10.4	9.6	10.6	10.6	12.1	12.9	12.1	16.4	15.8	15.0	17.0	18.6	17.6	18.5	17.3	16.8	18.0	17.5	17.2	17.4
	55–59	12.0	12.5	11.8	12.5	15.8	13.5	15.3	14.9	17.5	21.9	21.3	23.9	23.1	27.7	24.1	28.3	28.8	28.3	29.9	23.4	27.7
	60–64	11.7	11.1	15.2	12.4	10.2	16.3	13.8	17.3	17.1	19.8	20.3	23.6	22.9	27.7	27.1	31.4	30.6	37.1	37.0	31.4	33.5
	65–69	13.2	15.2	18.2	13.3	15.6	15.7	14.6	16.1	16.7	16.4	21.7	20.7	22.2	26.1	25.8	27.0	30.7	29.8	34.0	31.3	30.6
	70–74	10.3	12.9	11.7	12.1	11.8	11.7	13.0	14.5	12.1	11.6	17.9	16.8	17.6	20.6	17.1	20.1	25.7	24.6	26.6	26.8	24.9
	≥75	7.4	8.6	7.3	6.8	8.8	9.5	8.3	10.5	10.8	7.9	8.3	10.1	9.9	12.1	12.3	13.8	14.0	15.5	14.4	16.8	14.9

Source: Australian Cancer Database (see [methodology](#)). All ages category standardised to the 2001 Australian population. *Frequently HPV-associated: Base of tongue, lingual tonsil, soft palate, uvula, tonsil, oropharynx, Waldeyer ring.

Table A4.8 Head and neck cancer (not usually associated with HPV*) incidence rates per 100,000 estimated resident population, 2001–2020

		2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2016–2020 period
Females – Squamous cell carcinoma	All ages	1.9	1.5	1.9	2.0	1.7	1.8	2.0	1.9	1.7	2.3	1.9	1.9	2.1	2.0	2.3	2.0	1.8	1.9	2.1	2.2	2.0
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.1	0.1	0.0	0.1	0.1	0.1	0.1
	20–24	0.2	0.2	0.3	0.1	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.2	0.2	0.1	0.0	0.2	0.1	0.4	0.0	0.1
	25–29	0.6	0.0	0.1	0.6	0.4	0.1	0.1	0.4	0.4	0.1	0.2	0.0	0.7	0.0	0.3	0.2	0.4	0.0	0.2	0.3	0.2
	30–34	0.4	0.3	0.4	0.4	0.1	0.1	0.1	0.4	0.1	0.9	0.4	0.1	0.4	0.5	0.3	0.2	0.1	0.0	0.2	0.4	0.2
	35–39	0.1	0.5	0.5	0.4	0.3	0.1	0.6	0.0	0.2	1.0	0.1	0.5	0.4	0.3	0.4	1.0	0.4	0.3	0.7	0.3	0.5
	40–44	0.5	0.3	1.2	0.5	1.6	1.4	0.7	0.8	0.7	1.0	1.1	1.0	1.7	1.0	0.7	0.6	0.5	0.9	0.2	1.1	0.7
	45–49	1.0	1.0	1.3	1.4	1.4	0.7	1.6	0.8	1.3	1.7	1.0	2.3	1.7	1.5	1.8	1.8	1.1	1.3	1.5	1.8	1.5
	50–54	4.0	1.2	2.2	2.9	1.7	2.1	2.3	2.6	3.1	3.3	1.9	3.0	3.1	1.9	2.8	2.9	2.8	2.7	2.8	3.8	3.0
	55–59	3.9	2.4	3.4	3.9	3.9	4.0	3.0	3.3	3.2	3.9	5.0	2.9	2.8	3.6	3.9	4.4	4.0	4.4	5.3	4.4	4.5
	60–64	5.4	3.8	3.7	3.1	3.9	3.9	4.0	3.9	3.6	6.9	4.4	4.2	5.3	4.4	5.5	4.3	4.5	4.2	5.6	5.3	4.8
	65–69	7.3	8.0	7.2	7.5	5.5	6.9	8.7	6.2	5.1	7.0	3.7	6.8	5.7	7.1	6.2	4.6	6.1	5.5	6.2	4.9	5.5
	70–74	7.5	5.2	6.1	9.6	7.1	9.9	6.6	9.7	7.4	10.8	9.4	8.6	10.1	9.6	10.1	9.7	7.0	10.2	10.1	10.2	9.5
	≥75	8.9	9.3	11.3	10.5	9.6	8.7	13.2	11.2	10.2	10.7	11.7	10.2	10.5	11.9	14.0	10.9	10.4	10.6	10.6	12.8	11.1
Males – Squamous cell carcinoma	All ages	3.3	3.2	3.7	3.6	3.8	3.6	3.6	3.5	3.7	3.9	3.6	3.5	3.4	3.5	3.3	3.2	3.4	3.6	3.4	3.6	3.4
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.1	0.0	0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
	20–24	0.2	0.1	0.0	0.1	0.1	0.1	0.1	0.0	0.0	0.1	0.1	0.0	0.0	0.1	0.0	0.3	0.0	0.0	0.0	0.0	0.1
	25–29	0.9	0.3	0.4	0.4	0.4	0.1	0.3	0.5	0.1	0.6	0.2	0.3	0.5	0.2	0.7	0.1	0.1	0.5	0.3	0.3	0.3
	30–34	0.3	1.2	0.8	0.7	0.7	1.1	0.6	0.0	0.7	1.5	0.9	0.4	0.7	0.4	0.2	0.7	0.4	0.7	0.2	0.6	0.5
	35–39	1.8	1.4	1.4	1.5	1.6	0.9	0.9	1.0	1.8	1.6	0.8	0.8	0.9	0.9	1.0	0.2	1.4	0.8	1.1	0.5	0.8
	40–44	1.9	2.7	1.7	2.5	2.6	1.9	3.2	2.0	2.1	1.8	1.4	2.0	1.7	1.6	2.1	1.6	1.5	1.3	1.6	1.2	1.5
	45–49	2.5	4.0	3.9	3.4	4.2	2.9	3.5	3.3	2.9	5.2	3.0	3.9	2.9	3.3	2.5	2.8	3.5	3.5	2.4	3.2	3.1
	50–54	4.2	5.7	4.8	6.4	5.5	6.1	5.9	6.5	6.6	8.3	5.7	5.8	7.8	6.7	4.5	6.0	6.3	5.8	6.2	4.7	5.8
	55–59	7.5	6.4	9.2	9.4	10.4	9.1	7.0	8.4	9.7	8.9	7.9	8.3	7.9	8.3	7.5	7.7	8.8	8.5	6.7	12.6	8.9
	60–64	12.9	10.2	10.4	7.1	11.5	8.4	10.4	8.8	12.4	10.6	12.4	10.5	10.7	10.3	10.7	10.2	11.4	11.3	11.4	12.2	11.3
	65–69	13.2	10.8	14.8	15.8	16.4	11.5	11.3	10.5	11.6	13.3	15.0	13.2	13.8	14.2	15.7	12.1	12.1	14.7	13.4	11.5	12.7
	70–74	12.9	9.3	12.7	14.8	13.5	18.3	13.6	16.4	13.3	14.5	14.3	17.0	9.2	11.5	13.2	11.0	13.6	14.4	14.3	13.3	13.4
	≥75	11.5	12.0	15.4	12.0	11.4	13.9	15.4	14.1	13.5	12.4	14.5	11.7	13.4	15.2	11.2	12.6	11.0	14.7	13.6	14.5	13.3

Source: Australian Cancer Database (see [methodology](#)). All ages category standardised to the 2001 Australian population. **Not usually HPV-associated: upper lip (inner aspect), lip (unspecified, inner aspect), dorsal surface of tongue, anterior two-thirds of tongue (part unspecified), gum, floor of mouth, hard palate, other and unspecified parts of mouth.

Table A4.9 Anal cancer mortality rates in females per 100,000 estimated resident population, 2001–2020

		2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2016–2020 period
Anal squamous cell carcinoma	All ages	0.2	0.3	0.3	0.2	0.2	0.3	0.3	0.3	0.3	0.4	0.4	0.3	0.3	0.4	0.4	0.4	0.3	0.4	0.3	0.4	0.4
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	25–29	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	30–34	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0
	35–39	0.1	0.0	0.1	0.0	0.0	0.3	0.0	0.0	0.0	0.0	0.0	0.1	0.1	0.0	0.1	0.0	0.0	0.0	0.1	0.0	0.0
	40–44	0.1	0.0	0.0	0.1	0.0	0.1	0.1	0.1	0.1	0.0	0.0	0.1	0.0	0.1	0.2	0.0	0.1	0.1	0.0	0.2	0.1
	45–49	0.0	0.0	0.3	0.1	0.3	0.4	0.3	0.1	0.1	0.1	0.4	0.1	0.1	0.1	0.3	0.2	0.0	0.1	0.0	0.6	0.2
	50–54	0.5	0.8	0.2	0.5	0.2	0.6	0.6	0.7	0.8	1.2	1.3	0.6	0.3	0.9	0.5	0.4	0.6	0.8	0.3	0.3	0.5
	55–59	0.6	0.6	0.5	0.2	0.5	0.5	0.5	0.6	1.1	0.9	0.9	1.2	1.1	1.0	0.8	1.2	0.3	1.2	1.0	0.8	0.9
	60–64	0.5	1.0	0.2	0.2	0.9	0.8	0.8	0.4	0.2	0.3	0.7	0.6	0.6	0.3	0.8	1.3	0.6	1.6	1.3	1.2	1.2
	65–69	0.3	0.9	1.1	0.3	0.0	0.3	0.7	1.2	0.7	1.1	1.5	1.0	0.9	1.2	0.9	0.7	1.0	1.0	0.6	1.4	0.9
	70–74	0.3	0.3	1.2	0.0	0.3	0.9	0.6	0.9	0.6	1.7	1.9	1.6	0.8	1.9	1.6	1.1	1.4	1.7	1.1	1.2	1.3
	≥75	1.5	2.2	1.7	1.5	1.4	1.1	2.2	2.3	2.2	1.7	1.6	0.8	2.4	2.1	2.0	2.7	2.3	2.4	2.5	3.1	2.6
Anal adeno-carcinoma	All ages	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.1	0.0	0.1	0.0	0.0	0.0	0.0	0.0
	25–29	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
	30–34	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	35–39	0.1	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
	40–44	0.1	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	45–49	0.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.1	0.1	0.1
	50–54	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.4	0.0	0.0	0.1	0.0	0.1	0.0	0.3	0.0	0.0	0.0	0.1
	55–59	0.0	0.2	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.3	0.1	0.3	0.0	0.0	0.3	0.1
	60–64	0.2	0.0	0.0	0.0	0.2	0.0	0.2	0.2	0.0	0.0	0.3	0.2	0.0	0.2	0.0	0.3	0.4	0.1	0.1	0.0	0.2
	65–69	0.3	0.3	0.8	0.3	0.0	0.8	0.5	0.5	0.5	0.0	0.0	0.2	0.6	0.2	0.3	0.3	0.7	0.2	0.3	0.0	0.3
	70–74	0.3	0.6	0.3	0.3	0.0	0.3	0.6	0.6	0.0	1.1	0.5	0.0	0.0	0.2	0.0	0.4	0.2	0.2	0.0	0.0	0.2
	≥75	0.3	0.3	1.0	0.6	1.5	0.7	1.0	1.0	1.3	0.9	0.5	1.0	1.4	0.7	0.7	1.0	1.0	1.2	0.9	1.0	1.0

Source: Australian Cancer Database (see [methodology](#)). All ages category standardised to the 2001 Australian population.

Table A4.10 Anal cancer mortality rates in males per 100,000 estimated resident population, 2001–2020

		2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2016–2020 period
Anal squamous cell carcinoma	All ages	0.2	0.2	0.2	0.2	0.2	0.2	0.3	0.3	0.2	0.4	0.2	0.3	0.3	0.3	0.3	0.3	0.3	0.2	0.3	0.3	0.3
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	25–29	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	30–34	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	35–39	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	40–44	0.1	0.0	0.1	0.3	0.3	0.1	0.3	0.1	0.0	0.1	0.1	0.0	0.1	0.0	0.1	0.0	0.0	0.1	0.0	0.0	0.0
	45–49	0.0	0.1	0.1	0.1	0.3	0.1	0.1	0.5	0.1	0.4	0.3	0.4	0.1	0.1	0.1	0.4	0.0	0.2	0.0	0.4	0.2
	50–54	0.0	0.2	0.0	0.2	0.8	0.1	0.3	0.3	0.6	0.8	0.5	0.3	0.4	0.9	0.8	0.1	0.3	0.3	0.9	0.4	0.4
	55–59	0.4	0.0	0.2	0.8	0.3	0.0	0.5	0.8	0.3	1.1	0.3	0.6	0.4	0.6	0.8	0.7	0.3	0.3	0.3	0.5	0.4
	60–64	0.5	0.9	0.7	0.7	0.6	0.2	0.8	0.7	0.3	1.2	0.8	0.7	1.0	0.6	1.1	0.9	0.8	0.9	1.2	0.4	0.8
	65–69	0.3	0.9	0.9	0.6	0.5	0.8	0.8	0.5	0.7	0.4	0.4	1.2	1.7	1.1	1.0	0.8	0.7	1.0	1.0	1.0	0.9
	70–74	1.0	0.7	1.0	0.7	1.0	0.7	1.3	0.9	1.5	1.2	0.8	0.8	0.0	1.3	0.2	1.4	1.7	0.6	1.2	1.7	1.3
	≥75	1.4	1.5	0.6	1.0	0.8	1.7	1.9	1.1	1.1	1.4	1.0	1.6	1.4	2.2	1.6	1.9	2.1	1.4	1.9	2.0	1.9
Anal adeno-carcinoma	All ages	0.2	0.1	0.1	0.1	0.1	0.1	0.2	0.2	0.2	0.2	0.1	0.2	0.2	0.2	0.1	0.2	0.1	0.1	0.1	0.1	0.1
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	25–29	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	30–34	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	35–39	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	40–44	0.0	0.1	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	45–49	0.1	0.0	0.0	0.1	0.0	0.1	0.1	0.0	0.0	0.3	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.1	0.0	0.1	0.1
	50–54	0.2	0.0	0.2	0.0	0.2	0.1	0.1	0.0	0.1	0.1	0.0	0.1	0.1	0.0	0.3	0.0	0.1	0.0	0.0	0.0	0.0
	55–59	0.2	0.2	0.2	0.0	0.0	0.2	0.0	0.3	0.5	0.3	0.3	0.0	0.4	0.0	0.3	0.0	0.0	0.0	0.1	0.0	0.0
	60–64	0.7	0.5	0.2	0.0	0.4	0.0	0.2	0.2	0.2	0.3	0.2	0.7	0.0	0.5	0.3	0.5	0.2	0.6	0.4	0.7	0.5
	65–69	0.6	0.6	0.3	0.3	0.0	0.5	0.0	0.7	0.9	0.2	0.8	1.0	0.4	0.2	0.5	0.7	0.5	1.4	0.3	0.3	0.6
	70–74	1.0	0.3	0.3	0.7	0.7	0.3	2.3	0.9	0.3	1.2	0.0	1.9	0.8	0.8	0.5	0.2	0.6	0.2	0.4	0.9	0.5
	≥75	0.7	0.9	0.9	1.2	0.8	1.2	1.5	1.5	1.4	1.7	0.7	1.2	1.4	1.7	1.0	1.9	1.4	0.8	1.2	0.9	1.2

Source: Australian Cancer Database (see [methodology](#)). All ages category standardised to the 2001 Australian population.

Table A4.11 Vulval and vaginal cancer mortality rates in females per 100,000 estimated resident population, 2001–2020

		2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2016–2020 period
Vulval cancer – Squamous cell carcinoma	All ages	0.4	0.4	0.3	0.3	0.3	0.4	0.4	0.4	0.6	0.5	0.5	0.5	0.6	0.5	0.6	0.6	0.4	0.6	0.5	0.5	0.5
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	25–29	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	30–34	0.0	0.1	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	35–39	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.1	0.0	0.0	0.1	0.3	0.1	0.0	0.0	0.0	0.1	0.1	0.0	0.0
	40–44	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.2	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.0
	45–49	0.1	0.1	0.1	0.1	0.1	0.1	0.0	0.1	0.0	0.1	0.1	0.0	0.4	0.4	0.5	0.2	0.2	0.2	0.1	0.1	0.2
	50–54	0.5	0.0	0.2	0.2	0.3	0.1	0.0	0.1	0.0	0.1	0.4	0.3	0.4	0.3	0.1	0.3	0.1	0.3	0.0	0.1	0.2
	55–59	0.4	0.4	0.0	0.3	0.2	0.2	0.2	0.0	1.2	0.6	0.3	0.4	0.4	0.8	0.7	0.8	0.3	0.8	0.8	0.6	0.6
	60–64	0.7	0.0	0.7	0.7	0.2	0.6	0.6	0.7	0.5	0.5	0.3	0.6	0.5	0.9	0.6	0.3	0.3	0.9	0.0	0.7	0.4
	65–69	0.3	0.3	0.6	0.0	0.3	0.8	0.5	0.7	1.4	1.1	1.5	1.8	1.1	0.5	1.5	2.5	1.3	1.3	1.6	1.7	1.7
	70–74	0.9	1.8	0.6	0.3	0.6	2.2	2.7	0.9	2.3	1.9	3.0	1.8	2.0	3.1	1.8	2.0	1.6	1.9	2.0	1.6	1.8
	≥75	4.0	4.2	2.8	3.5	4.6	4.3	5.2	5.5	6.2	4.7	4.2	4.9	7.1	4.5	6.6	5.4	5.0	5.7	6.1	5.7	5.6
Vaginal cancer – Squamous cell carcinoma	All ages	0.2	0.1	0.2	0.1	0.2	0.1	0.2	0.2	0.1	0.1	0.2	0.1	0.2	0.1	0.2	0.2	0.1	0.2	0.1	0.2	0.2
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	25–29	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	30–34	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0
	35–39	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	40–44	0.1	0.0	0.0	0.0	0.1	0.1	0.1	0.1	0.0	0.0	0.0	0.1	0.1	0.0	0.0	0.1	0.0	0.1	0.1	0.2	0.1
	45–49	0.3	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.3	0.1	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0
	50–54	0.0	0.5	0.2	0.2	0.3	0.1	0.0	0.1	0.3	0.1	0.1	0.1	0.1	0.1	0.0	0.4	0.3	0.1	0.0	0.0	0.2
	55–59	0.2	0.2	0.4	0.5	0.0	0.2	0.3	0.3	0.2	0.0	0.4	0.4	0.1	0.3	0.0	0.3	0.4	0.1	0.0	0.3	0.2
	60–64	0.0	0.7	0.2	0.4	0.0	0.2	0.2	0.2	0.0	0.2	0.3	0.3	0.3	0.6	0.5	0.3	0.1	0.3	0.6	0.3	0.3
	65–69	0.0	0.3	0.0	0.0	0.3	0.3	0.5	0.2	0.2	1.1	0.4	0.2	0.2	0.0	0.3	0.3	0.5	0.3	0.6	0.6	0.5
	70–74	0.6	0.3	0.9	0.9	0.6	0.6	0.0	1.2	0.3	0.6	0.5	0.3	0.5	0.2	1.2	0.9	0.2	0.4	1.1	1.1	0.7
	≥75	2.1	1.0	1.8	1.1	2.4	0.9	1.8	1.5	1.7	0.4	2.2	0.6	1.8	1.2	1.5	1.7	0.3	1.7	0.9	0.9	1.1

Source: Australian Cancer Database (see [methodology](#)). All ages category standardised to the 2001 Australian population.

Table A4.12 Penile cancer mortality rates in males per 100,000 estimated resident population, 2001–2020

		2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2016–2020 period
Penile cancer– Squamous cell carcinoma	All ages	0.1	0.1	0.1	0.1	0.1	0.2	0.1	0.1	0.2	0.2	0.2	0.1	0.2	0.1	0.1	0.2	0.1	0.3	0.2	0.1	0.2
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	25–29	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	30–34	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	35–39	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.1	0.0	0.1	0.0	0.0	0.2	0.1	0.0	0.1
	40–44	0.4	0.0	0.4	0.1	0.1	0.4	0.0	0.0	0.0	0.3	0.0	0.0	0.1	0.0	0.1	0.2	0.0	0.0	0.1	0.1	0.1
	45–49	0.0	0.0	0.1	0.0	0.3	0.0	0.1	0.0	0.0	0.3	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.1	0.1	0.0	0.1
	50–54	0.2	0.0	0.0	0.2	0.0	0.1	0.0	0.1	0.1	0.0	0.1	0.0	0.0	0.0	0.0	0.4	0.0	0.0	0.1	0.3	0.2
	55–59	0.2	0.4	0.3	0.0	0.0	0.2	0.2	0.3	0.0	0.0	0.2	0.4	0.1	0.0	0.1	0.0	0.1	0.1	0.4	0.8	0.3
	60–64	0.0	0.0	0.5	0.2	0.2	0.2	0.0	0.4	0.0	0.2	0.3	0.3	0.3	0.3	0.3	0.0	0.0	0.9	0.4	0.3	0.3
	65–69	0.3	0.3	0.6	0.8	0.3	0.8	0.5	0.5	0.5	0.9	0.4	0.4	0.2	0.0	0.3	0.2	0.7	0.8	0.3	0.7	0.5
	70–74	1.7	0.7	0.3	0.3	0.3	0.0	0.0	0.6	1.5	0.6	1.4	0.5	0.8	0.3	0.5	0.7	0.2	1.8	0.0	0.0	0.5
	≥75	0.2	0.4	0.6	0.6	0.6	1.2	1.3	0.4	2.2	1.4	1.5	1.0	1.4	1.1	0.9	1.4	1.5	2.0	2.2	0.7	1.6

Source: Australian Cancer Database (see [methodology](#)). All ages category standardised to the 2001 Australian population.

Table A4.13 Oropharyngeal cancer (frequently associated with HPV⁺) mortality rates per 100,000 estimated resident population, 2001–2020

		2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2016–2020 period
Females – Squamous cell carcinoma	All ages	0.3	0.4	0.4	0.3	0.3	0.4	0.4	0.4	0.4	0.3	0.3	0.3	0.3	0.4	0.5	0.4	0.3	0.4	0.3	0.4	0.4
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	25–29	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0
	30–34	0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	35–39	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	40–44	0.0	0.0	0.4	0.3	0.1	0.0	0.3	0.0	0.1	0.0	0.1	0.1	0.0	0.0	0.0	0.0	0.2	0.0	0.1	0.0	0.1
	45–49	0.3	0.4	0.4	0.3	0.0	0.0	0.0	0.3	0.6	0.1	0.0	0.1	0.5	0.4	0.1	0.5	0.1	0.4	0.2	0.1	0.3
	50–54	0.3	0.5	0.0	0.5	0.3	0.7	0.6	0.9	0.3	0.8	0.4	0.0	0.4	0.8	0.6	0.5	0.5	0.8	0.3	0.4	0.5
	55–59	0.8	0.6	0.7	0.3	0.8	0.6	0.6	0.8	0.8	0.8	0.6	1.0	0.4	1.4	0.7	1.2	0.9	1.0	0.5	1.4	1.0
	60–64	1.0	1.2	1.4	1.8	2.1	1.6	1.5	0.9	1.4	0.8	1.3	1.0	0.8	1.1	1.8	1.2	0.7	1.0	1.0	1.5	1.1
	65–69	1.7	0.9	1.9	0.8	0.3	1.3	0.7	1.9	1.4	0.9	0.6	1.4	1.3	1.1	1.2	0.7	1.2	1.3	0.5	1.4	1.0
	70–74	1.2	2.7	1.2	0.9	2.2	1.5	0.9	0.3	1.4	2.2	2.2	0.8	1.8	1.9	3.0	0.9	1.2	1.5	1.1	2.1	1.4
	≥75	1.0	1.6	2.4	1.9	1.4	2.3	2.8	2.5	2.2	1.6	2.1	1.7	1.7	1.2	2.4	2.2	1.8	2.1	2.6	2.1	2.2
Males – Squamous cell carcinoma	All ages	1.4	1.3	1.3	1.4	1.2	1.1	1.5	1.6	1.7	1.5	1.6	1.7	1.7	1.7	1.8	1.6	1.7	1.9	1.9	1.8	1.8
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0
	25–29	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	30–34	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0
	35–39	0.0	0.1	0.1	0.1	0.3	0.1	0.1	0.3	0.1	0.0	0.3	0.1	0.1	0.1	0.0	0.0	0.1	0.0	0.0	0.4	0.1
	40–44	0.7	0.1	0.8	0.7	0.1	0.4	0.0	0.3	0.9	0.0	0.1	0.4	0.4	0.5	0.7	0.4	0.0	0.4	0.1	0.1	0.2
	45–49	1.5	1.5	1.3	1.1	1.0	0.4	1.2	1.0	1.6	1.4	1.3	1.2	1.1	1.2	1.0	1.3	1.0	0.8	1.4	0.2	1.0
	50–54	2.6	1.4	3.1	2.0	2.4	2.5	2.5	2.3	2.7	3.0	2.7	2.8	2.1	2.5	2.3	2.0	2.8	2.7	1.2	2.3	2.2
	55–59	4.3	3.3	2.4	4.7	4.9	2.7	5.3	4.9	4.1	2.8	5.0	4.7	6.6	5.0	5.3	3.0	4.1	4.0	4.7	4.6	4.1
	60–64	3.9	6.4	4.4	5.5	3.2	3.5	5.3	4.6	5.2	6.2	6.1	6.7	7.8	5.3	5.7	6.9	5.1	7.7	7.8	6.3	6.8
	65–69	4.8	6.7	6.6	6.4	6.4	5.5	4.5	7.1	6.3	6.0	7.2	7.9	7.3	9.2	7.9	7.0	8.9	8.0	8.7	8.2	8.1
	70–74	5.0	4.0	6.7	6.0	4.0	5.3	5.5	7.2	7.9	7.0	5.9	7.8	4.5	7.5	6.5	6.2	7.2	9.4	9.8	8.0	8.2
	≥75	5.5	5.1	3.4	3.7	4.0	4.1	6.2	6.4	7.5	6.6	6.6	5.8	7.0	6.6	8.1	7.7	8.0	8.5	9.3	8.7	8.5

Source: Australian Cancer Database (see [methodology](#)). All ages category standardised to the 2001 Australian population. *Frequently HPV-associated: Base of tongue, lingual tonsil, soft palate, uvula, tonsil, oropharynx, Waldeyer ring.

Table A4.14 Head and neck cancers (not usually associated with HPV*) mortality rates per 100,000 estimated resident population, 2001–2020

		2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2016–2020 period
Females – Squamous cell carcinoma	All ages	0.5	0.5	0.5	0.4	0.5	0.4	0.6	0.6	0.7	0.6	0.6	0.7	0.6	0.6	0.6	0.5	0.6	0.5	0.6	0.6	0.6
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.0	0.0	0.0	0.1	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
	25–29	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0
	30–34	0.0	0.0	0.1	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.1	0.1	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
	35–39	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.3	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0
	40–44	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.3	0.0	0.1	0.0	0.2	0.0	0.1	0.2	0.0	0.0	0.1	0.1
	45–49	0.0	0.1	0.1	0.1	0.3	0.3	0.1	0.1	0.4	0.0	0.4	0.4	0.3	0.3	0.4	0.0	0.1	0.1	0.1	0.1	0.1
	50–54	0.6	0.8	0.2	0.2	0.3	0.3	0.4	0.4	0.4	0.5	0.4	0.5	0.1	0.1	0.5	0.5	0.5	0.8	0.3	0.4	0.5
	55–59	0.8	0.8	0.9	1.0	0.7	0.6	0.3	0.9	0.8	0.6	0.9	0.6	0.4	1.0	0.4	0.3	0.7	0.6	1.0	1.1	0.7
	60–64	0.5	0.7	0.2	0.9	1.1	0.8	1.1	0.5	1.0	1.2	1.0	2.3	1.0	0.8	0.8	0.4	0.7	1.3	0.7	0.7	0.8
	65–69	0.9	0.6	1.9	1.9	0.8	2.0	0.7	1.7	2.3	1.1	2.5	1.9	2.6	1.6	2.2	1.2	1.3	1.5	1.6	1.4	1.4
	70–74	1.8	2.1	2.1	0.3	2.5	1.5	3.9	2.9	2.3	2.8	2.7	3.1	1.3	3.6	1.2	2.4	3.1	1.9	2.6	2.6	2.5
	≥75	4.8	3.9	4.4	3.6	3.8	2.9	6.4	5.4	6.1	5.5	4.8	4.9	6.6	5.8	6.6	5.6	5.7	4.1	5.3	5.4	5.2
Males – Squamous cell carcinoma	All ages	1.0	0.8	0.9	0.9	1.0	1.0	1.3	1.2	1.0	1.2	1.1	1.1	1.3	1.0	1.0	1.2	0.9	0.9	1.1	0.9	1.0
	0–14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15–19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20–24	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.0	0.0	0.1	0.0	0.0	0.0	0.0
	25–29	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.1	0.1	0.0
	30–34	0.0	0.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.3	0.0	0.1	0.0	0.0	0.0	0.2	0.0	0.1	0.2	0.0	0.1
	35–39	0.1	0.1	0.3	0.3	0.4	0.5	0.0	0.3	0.1	0.1	0.1	0.0	0.3	0.1	0.3	0.1	0.0	0.0	0.1	0.1	0.1
	40–44	0.1	0.4	0.7	0.0	0.8	0.5	0.3	0.0	0.3	0.4	0.3	0.2	0.1	0.1	0.1	0.0	0.3	0.0	0.4	0.1	0.2
	45–49	0.7	0.1	0.9	0.3	1.4	1.0	0.5	0.9	0.8	1.4	0.7	0.5	0.5	0.8	0.4	0.5	0.2	0.4	1.1	0.6	0.6
	50–54	1.5	0.8	0.8	1.8	1.2	0.1	1.5	1.9	0.8	1.5	0.9	0.7	2.1	1.3	0.8	1.3	1.3	1.2	1.1	1.0	1.2
	55–59	2.2	1.8	2.4	1.5	2.4	2.1	2.2	2.7	1.3	2.6	2.7	2.4	3.1	2.3	1.7	1.9	1.4	2.0	2.9	2.1	2.1
	60–64	3.4	3.5	1.6	3.3	1.9	3.3	1.7	3.0	2.1	2.7	3.8	3.9	2.8	3.2	2.5	3.9	3.7	4.5	2.4	3.0	3.5
	65–69	4.2	3.5	4.3	4.2	6.4	3.4	5.8	4.6	4.4	4.7	4.2	4.7	4.1	4.1	6.1	4.9	3.4	2.9	4.4	3.8	3.9
	70–74	4.6	2.0	3.7	3.0	3.0	3.7	7.8	5.7	6.7	5.8	4.5	6.2	6.3	3.8	3.8	4.8	4.5	2.2	4.4	4.1	4.0
	≥75	3.9	4.2	4.7	4.9	3.6	5.6	8.5	6.1	6.6	5.9	6.8	6.4	7.2	6.2	6.9	7.1	5.3	5.1	6.1	5.2	5.7

Source: Australian Cancer Database (see [methodology](#)). All ages category standardised to the 2001 Australian population. **Not usually HPV-associated: upper lip (inner aspect), lip (unspecified, inner aspect), dorsal surface of tongue, anterior two-thirds of tongue (part unspecified), gum, floor of mouth, hard palate, other and unspecified parts of mouth.

Table A4.15 Prevalence (%) of HPV by type in people with a first cervical cancer histology result, 2018–2023

		2018	2019	2020	2021	2022	2023
All ages	Known HPV status	76.2	86.6	86.7	88.9	88.8	89.0
	Unknown HPV status	23.8	13.4	13.3	11.1	11.2	11.0
25–74	Known HPV status	78.7	88.7	88.1	90.3	91.2	90.2
	Unknown HPV status	21.3	11.3	11.9	9.7	8.8	9.8
All ages	HPV detected	88.8	92.3	90.9	89.1	90.1	88.8
	HPV never detected	11.2	7.7	9.1	10.9	9.9	11.2
25–74	HPV detected	90.0	93.0	91.4	90.2	91.4	90.2
	HPV never detected	10.0	7.0	8.6	9.8	8.6	9.8
All ages[†]	Only HPV16/18	78.1	78.3	70.9	67.3	68.1	64.2
	Only non HPV16/18 types	18.2	17.6	20.6	22.1	23.5	26.5
	Both HPV16/18 and other	3.7	4.1	8.5	10.6	8.4	9.3
25–74[†]	Only HPV16/18	79.3	79.2	71.8	67.3	69.0	64.8
	Only non HPV16/18 types	16.9	16.5	19.5	22.0	22.7	25.9
	Both HPV16/18 and other	3.9	4.2	8.7	10.8	8.3	9.3
<30[†]	Only HPV16/18 (a)	82.9	60.5	40.0	20.7	40.0	36.0
	Only non HPV16/18 types (a)	17.1	36.8	44.0	58.6	44.0	52.0
	Both HPV16/18 and other (a)	0.0	2.6	16.0	20.7	16.0	12.0
30–39[†]	Only HPV16/18	83.2	83.5	74.3	63.4	71.0	59.2
	Only non HPV16/18 types	12.0	10.9	14.5	20.3	18.6	28.8
	Both HPV16/18 and other (a)	4.9	5.7	11.2	16.3	10.4	12.0
40–49[†]	Only HPV16/18	78.8	75.3	78.8	77.1	71.5	71.0
	Only non HPV16/18 types	15.9	21.1	16.3	16.1	21.5	20.2
	Both HPV16/18 and other (a)	5.3	3.6	4.9	6.8	7.0	8.8
50–59[†]	Only HPV16/18	75.9	78.0	70.5	70.2	71.2	73.0
	Only non HPV16/18 types	20.5	16.5	20.5	21.5	21.6	18.0
	Both HPV16/18 and other (a)	3.6	5.5	9.0	8.3	7.2	9.0
60–74[†]	Only HPV16/18	69.8	83.0	58.4	67.0	63.4	60.7
	Only non HPV16/18 types	30.2	16.0	33.7	26.6	29.0	33.6
	Both HPV16/18 and other (a)	0.0	1.0	7.9	6.4	7.5	5.7
75+[†]	Only HPV16/18 (a)	50.0	56.3	45.0	70.0	51.7	53.3
	Only non HPV16/18 types (a)	50.0	43.8	55.0	25.0	41.4	36.7
	Both HPV16/18 and other (a)	0.0	0.0	0.0	5.0	6.9	10.0

Source: National Cancer Screening Register (see [methodology](#)). (a) Some rates based on numerators <10 or denominators < 20 and should be interpreted with caution.

[†]Among those with a known HPV result over the 5 years preceding the cancer histology result.

