National Cancer Prevention Policy
Cervical Cancer
Cervical cancer

About this chapter

This chapter of the National Cancer Prevention Policy combines information about human papillomavirus immunisation and cervical screening, which were two separate chapters in previous editions of the NCPP. It will be updated as significant new literature is published or changes in the policy environment occur. We will review the content and policy priorities as information from the current Renewal of the National Cervical Screening Program\(^1\) is released.

The chapter was revised in March/April 2012 in consultation with Associate Professor Karen Canfell (Program Leader, Cancer Modelling Program and NHMRC Career Development Fellow, Cancer Research Division, Cancer Council NSW and Clinical Associate Professor in Public Health, University of Sydney). Associate Professor Kristine Macartney (Deputy Director of Government Programs, National Centre for Immunisation Research & Surveillance) provided advice about HPV immunisation. It was externally reviewed in July 2012 by Professor Ian Frazer, Professor Ian Hammond and Dr Marion Saville.

Contact: Paul Grogan\(^2\)

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Overview
Cervical cancer is one of the most preventable cancers. Cervical screening with Pap tests enables early detection and treatment of cervical abnormalities prior to the development of cancer. In Australia, cervical cancer incidence and death rates have halved since the introduction of a national cervical screening program in 1991\cite{AIHW2011}.

It is now established that persistent infection with human papillomavirus (HPV) causes nearly all cervical cancers. This knowledge has led to two important clinical advances: a vaccine for primary prevention of cervical cancer, and HPV testing to improve secondary prevention. The introduction of a national HPV vaccination program in 2007, delivering the prophylactic vaccine to adolescent girls, is expected to further reduce cervical cancer in Australia.

Given these developments it is time to consider the optimal combination and integration of vaccination, HPV testing and screening, as well as the role of conventional and new methods of cytology, and new screening technologies, into the future. New approaches to screening will be needed as the proportion of vaccinated women participating in screening increases. A review of Australia’s cervical screening program (presently underway) – including screening tests, intervals and age ranges – and improved surveillance of HPV prevalence, cytology and cervical cancer incidence, are essential to optimise prevention of cervical cancer.

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Impact

Cervical cancer is the 13th most common cancer affecting Australian women. Australia has one of the lowest rates of cervical cancer and incidence in the world. In 2008 in Australia there were 778 new cases of cervical cancer\textsuperscript{AIHW2012}. In 2007 (the latest year for which we have mortality data) there were 208 deaths attributable to cervical cancer\textsuperscript{AIHW\_AACR2010}. The lifetime risk of a woman developing cervical cancer before the age of 85 years is one in 158\textsuperscript{AIHW\_AACR2010}.

Cervical cancer mortality rates are four to five times higher for Aboriginal and Torres Strait Islander women than for non-Indigenous Australian women\textsuperscript{AIHW\_AACR2010}. This has been attributed to low participation in cervical screening\textsuperscript{Condon2005}. In some Australian states and territories Aboriginal and Torres Strait Islander women’s participation rates have improved in response to efforts to increase accessibility and provide more culturally-acceptable programs.

Worldwide, cervical cancer is the seventh most common cancer, and the third most common cancer in women, with an estimated 530,000 new cases in 2008. The burden is greater in developing countries, where cervical cancer accounts for 13\% of all female cancers. Cervical cancer caused 275,000 deaths worldwide in 2008, 88\% of which occurred in developing countries\textsuperscript{IARC2008}.

Almost all cases of cervical cancer are attributable to human papillomavirus (HPV) infection. HPV infection is highly prevalent: the estimated lifetime risk for women of one or more genital HPV infections is 80\%\textsuperscript{Bekkers2004}. It is estimated that around 291 million women worldwide are infected with HPV, almost a third of whom are infected with the high-risk types HPV16 or HPV18 or both\textsuperscript{deSanjose2007}, which are present in about 70\% of cervical cancers.

In Australia cervical cancer incidence rates halved between 1991, when Australia’s National Cervical Screening Program commenced, and 2008\textsuperscript{AIHW2012}. Australia’s cervical cancer mortality rate also halved, from 4.0 per 100,000 population in 1991 to 1.8 in 2007\textsuperscript{AIHW\_AACR2010}, and is now among the lowest in the world\textsuperscript{Jemal2011}.

However in recent years, incidence rates appear to have stabilised. The majority of the decline in invasive cervical cancer in developed countries is due to a reduction in squamous cell carcinoma (SCC). In contrast, the incidence of adenocarcinomas has not declined, largely attributed to difficulties in detecting these types of cancer through cervical screening\textsuperscript{Sasieni2009 #Ault2011}. In Australia this previously rare cancer now comprises around a quarter of all cervical cancers diagnosed\textsuperscript{AIHW2012}. Trends in age-standardised incidence for different types of cervical carcinomas in Australia are shown in Figure 1.

Figure 1. Incidence of carcinoma of the cervix (squamous cell carcinoma, adenocarcinoma, adenosquamous carcinoma and other carcinoma) in women aged 20–69, by year, 1982 to 2008
Number of new cases per 100,000 women


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Causes

Persistent infection with human papillomavirus (HPV) causes an estimated 5% of the total global cancer burden#Parkin2006. HPV has been identified in 99.7% of cervical cancer specimens#Walboomers1999, and a significant proportion of some anal, vaginal, vulval, penile and head and neck cancers.

HPV is a common, and usually asymptomatic, sexually transmitted infection. Almost all individuals become infected with HPV within two to five years of becoming sexually active#Wright2006.

Of the more than 40 anogenital HPV types, 15 are classified as 'high risk' based on the strength of their association with cervical cancer. HPV types 16, 18 and 45 are responsible for nearly all cases of cervical cancer worldwide#Guan2012. HPV types 16 and 18 are detected in more than 70% of cases of cervical cancer in Australia#Brotherton2008.

While HPV infection is necessary for the development of cervical cancer, it is certainly not sufficient#Walboomers1999. Worldwide, there are estimated to be about 100 million adult women who are infected with high-risk HPV types#Giles2006. This compares with approximately 530,000 new cases of cervical cancer worldwide each year#IARC2008. Cervical cancer is a very rare outcome in relation to the high prevalence of HPV infection. However, the risk of developing cancer increases significantly with persistent HPV infection#Koshiol2008.

Human papillomavirus

Species-specific papillomaviruses infect a range of animals including humans. Over 100 different types of HPV have been identified. The majority of HPV types infect only epithelial cells of the skin and mucosa. Most produce no evident disease. Some HPV types have been associated with anogenital and aerodigestive diseases#Baseman2005. Some produce proliferative lesions (warts) of skin and genital skin, and these tend to be grouped as low and high risk based on their potential to cause malignancy.

Anogenital HPVs are the most common sexually transmitted infections. There are more than 40 anogenital HPV types, 15 of which are classified as 'high risk'#IARC2007. High risk types can establish persistent infection and cause abnormalities which, in rare case, progress to cancer. A recent meta-analysis suggests some of these types have relatively low carcinogenic potential, and types 16, 18 and 45 are the cause of virtually all cases of cervical cancer worldwide#Guan2012.
Transmission

Genital HPVs are primarily transmitted through sexual contact (genital-genital or genital-anal), though this need not include penetrative sexual intercourse. Other modes of transmission, including perinatal, digital, oral and autoinoculation, are thought to be less common#IARC2005. HPV infections are thought to be established in the basal epithelium through abrasion or microtrauma of the superficial epithelium#Lowy2006.

Genital HPV infection is common in sexually active adults. A study of cervical HPV prevalence found multiple infections were common in Australian women, with a wide range of HPV types detected (HPV 16 being the most common) and incidence peaking in the years following the start of sexual activity#Garland2011a. International data show prevalence is high even in young women who are with their first partner and are monogamous, with HPV infection rates of 30% within one year of becoming sexually active and 48% within three years#Winer2009. Most infections are self-limited or cleared by the immune system within 1 to 2 years#Stanley2006a. HPV prevalence peaks soon after the average age of first sexual intercourse; prevalence among women aged over 30 years is much lower than among younger women#Franceschi2006.

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HPV infection and cancer

HPV infections can induce the development of either benign or malignant lesions. Benign lesions include non-genital and anogenital skin warts, oral and laryngeal papillomas and anogenital mucosal condylomata#Lowy2006.

Persistent infection with high-risk HPVs are generally subclinical, but can result in the development of a range of anogenital tumours including cancers of the cervix, anus, penis, vulva and vagina#Lowy2006. HPV infection is associated with squamous cell carcinomas of the head and neck, particularly oropharyngeal cancers#Kreimer2005. There is continuing research into the possible role of HPV infection in the development of cancers in other sites.

In 2007 the International Agency for Research on Cancer published an analysis of the current evidence linking HPV infection to specific cancers#IARC2007. Grouping the links according to whether the evidence was sufficient, limited or inadequate, the IARC concluded:

- There is sufficient evidence in humans for the carcinogenicity of HPV 16 in the cervix, vulva (basaloid and warty tumours), vagina, penis (basaloid and warty tumours), anus, oral cavity and oropharynx.
- There is sufficient evidence in humans for the carcinogenicity of HPV 18 in the cervix.
- There is sufficient evidence in humans for the carcinogenicity of HPV types 31, 33, 35, 39, 45, 51, 52, 56, 58, 59 and 66 in the cervix.
- There is limited evidence in humans for the carcinogenicity of HPV 16 in the larynx and periungual skin (squamous-cell carcinoma).
- There is limited evidence in humans for the carcinogenicity of HPV 18 in the vulva (basaloid and warty tumours), vagina, penis (basaloid and warty tumours), anus, oral cavity and larynx.
- There is limited evidence in humans for the carcinogenicity of HPV 6 and HPV 11 in the cervix (squamous-cell carcinoma) and in the vulva, penis and anus (verrucous carcinomas of the latter three sites).
- There is limited evidence in humans for the carcinogenicity of HPV genus-beta types in the skin (squamous-cell carcinoma). In the rare case of patients with epidermodysplasia verruciformis, there is compelling evidence for the carcinogenicity of HPV genus-beta types 5 and 8 in the skin (squamous-cell carcinoma).
- There is limited evidence in humans for the carcinogenicity of HPV in the conjunctiva (squamous-cell carcinoma).
- There is inadequate evidence in humans for the carcinogenicity of HPV in the oesophagus, lung, colon, ovary, breast, prostate, urinary bladder and nasal and sinonasal cavities#IARC2007.
HPV and cervical cancer

The association between HPV and cervical cancer was not determined until the 1970s, and it was not until the mid-1990s that the primary role of HPV in the development of cervical cancer was definitely confirmed#Eurogin2003. HPV has been identified in 99.7% of cervical cancer specimens#Walboomers1999.

Research has distinguished between high-risk (oncogenic) and low-risk (non-oncogenic) types of HPV. Cervical cancer and its immediate precursor lesion (CIN3) only develop after many years of persistent infection with a high-risk type#Nobbenhuis1999. More than 70% of squamous cell carcinomas and about 78% of adenocarcinomas are caused by the high-risk types 16 or 18#ICESCC2007. HPV type 16 is the most carcinogenic, accounting for about 55% to 60% of cervical cancers; HPV 18 accounts for a further 10% to 15% of cervical cancers#Munoz2003 #deSanjose2010.

The four major steps in cervical cancer development are HPV infection/acquisition, viral persistence (vs clearance), progression to cervical precancer and invasion#Schiffman2007b. It is estimated that it takes an average of 10 years from HPV infection to malignant progression#Schiffman2003.

It previously was thought that the development of cervical cancer involved progression from low to moderate to high-grade intraepithelial lesions (HSIL), but studies have shown that low and high-grade cervical lesions are distinct HPV processes#Baseman2005. It is now accepted that low-grade squamous intraepithelial lesions represent acute HPV infection (high- or low-risk) rather than cancer precursors#Guan2012, most of which will resolve spontaneously within 12 months#Rodriguez2008. Most high-grade abnormalities also regress over time, but regression takes longer#Schlecht2003.

Pre-cancerous lesions (CIN3) occur when oncogenic HPV is not cleared, infects immature cells and prevents maturation and differentiation, resulting in the replication of immature cells and the accrual of genetic changes that can lead to cervical cancer#Baseman2005. HSILs were indicated in 0.8% of cytology tests in Australia in 2009. Histologically verified HSIL includes the subcategory CIN3 or pre-cancer.

Women with persistent infections, especially with HPV 16, are at significantly higher risk of CIN3 and cervical cancer#Trimble2009 #Kjaer2010 #Brotherton2011b. Co-factors that increase the risk of cervical cancer progressing in women who have a persistent high-risk HPV infection include:

• multiparity (more than five full-term pregnancies)#ICESCC2006;
• early age at first full-term pregnancy#ICESCC2006; and
• the use of oral contraceptives#Appleby2007.

Current cigarette smoking is associated with a significantly increased risk of squamous cell carcinoma, but not of adenocarcinoma#Appleby2006. Immunodeficiency (acquired through chronic immunosuppresion, for example HIV infection) also contributes significantly to persisting HPV infection and cervical cancer risk#Grulich2007.

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Prevention

Following HPV infection being determined as necessary for the for the development of cervical cancer, vaccines have been developed to prevent infection with high-risk types of HPV. Vaccination offers the potential to further the decrease in cervical cancer rates and deaths that has been seen with the introduction of screening programs, particularly among groups with lower participation in such programs.

By preventing infection with high risk HPV types 16 and 18 (and potentially other types) prophylactic vaccines could prevent up to 70% of cervical cancers#Munoz2003. As nearly 80% of adenocarcinomas are associated with the most common HPV types#ICESCC2007, prophylactic HPV vaccination is expected to be effective in preventing these cancers#Ault2011, which has not been achieved by cervical screening.

Second-generation prophylactic vaccines that aim to prevent a greater number of HPV types are in development. The efficacy of the vaccines is likely to enable less intensive screening of vaccinated women in the future.

Internationally, prophylactic vaccination could have greatest impact in developing countries that have the highest burden of HPV-related disease and lack organised screening programs, with the potential to prevent the deaths of millions of women in the next decade#Goldie2008. Given the prevalence of HPV infection worldwide, development of a therapeutic vaccine is also a priority.

Vaccines to prevent HPV infection

Two prophylactic HPV vaccines have been developed commercially and are approved for use in Australia. Cervarix is a bivalent HPV 16/18 vaccine developed by GlaxoSmithKline. It is given as an intra-muscular injection in a three-dose course, generally at zero, one and six months#Stanley2006b. Gardasil, developed by Merck and Co. Inc., is a quadrivalent HPV 16/18/6/11 L1 VLP vaccine delivered by an intra-muscular injection at zero, two and six months#Stanley2006b. Both vaccines have been approved by the Therapeutic Goods Administration for use in Australia.

For maximum efficacy, prophylactic vaccines must be administered to individuals prior to HPV exposure#Herrero2011. Current generation vaccines do not have a therapeutic effect in those already infected with HPV. It is recommended that HPV vaccines be provided before sexual activity commences. In Australia, the National Immunisation Program targets the vaccine to adolescent/pre-adolescent girls, aged 11 to 13 years.

For further information about registered use of HPV vaccines and the National Immunisation Program see Policy context.

Second-generation, expanded valency vaccines are currently in Phase III clinical trials. These vaccines aim to prevent a broad spectrum of HPV infections, increasing the proportion of cervical cancers that would be prevented.
Safety

Large-scale studies have shown the HPV vaccines to be safe and well tolerated. Gardasil, the quadrivalent vaccine distributed via the National Immunisation Program, has been assessed as safe and effective by Australia’s Therapeutic Goods Administration and by the US Food and Drug Administration and the European Medicines Agency.

From the introduction of the National HPV Vaccination Program to June 2010 more than 6 million doses of Gardasil had been distributed and a very low number of adverse events following administration had been reported. The most common side effects after HPV vaccination are mild such as pain, swelling or redness at the injection site. Commonly reported events such as headache or feeling unwell may be equally common in people who have not received the vaccine, hence establishing a causal link to the vaccine is difficult. About one in five reported events were events associated with injection procedures generally, including dizziness, syncope (fainting) and panic attacks. A recent review of notifications of syncope following HPV vaccination in Australia found rates were similar to those reported internationally.

Anaphylaxis (severe allergic reaction) has occurred rarely following administration of Gardasil. The estimated rate of anaphylaxis following HPV vaccination in Australia is 2.6 per million. International studies show anaphylaxis occurs at similar rates (ranging from 0 to 3.5 per million doses) following administration of other vaccines to children and adolescents.

No other serious or unusual events have been causally related to the HPV vaccines in large-scale trials (with up to four years of follow-up) and clinical usage to date.

For further information about HPV vaccine safety and efficacy, as well as dosage and administration, please refer to the Australian Immunisation Handbook.

Efficacy

Results demonstrate that both quadrivalent and bivalent vaccines are highly effective in preventing persistent infections and cervical disease associated with HPV types 16 and 18 when given to females who are not already infected with these HPV types. The quadrivalent vaccine confers additional protection against HPV 6/11-induced mucosal and cutaneous genital disease. Both vaccines have demonstrated some cross-protection against other HPV types, but further studies are needed to confirm the size of the impact and efficacy against diseases due to other HPV types.

The duration of protection conferred has been shown in clinical trials to be over 8.4 years for the bivalent vaccine, and beyond five years for the quadrivalent vaccine. Studies have measured the levels of neutralising antibodies against the targeted HPV types, but it is unknown whether antibody levels reflect clinical immunity and thus whether higher antibody levels will result in longer duration of protection.

Based on present knowledge, both HPV vaccines are anticipated to provide long-term protection against the high-risk HPV types, but ongoing monitoring is essential. If future research identifies waning protection, individuals who have been vaccinated will need to be recalled for a booster. The National HPV Vaccination Register was established to record details of all individual vaccinations in case such recall is required. Recent studies show that for both the bivalent and quadrivalent vaccines two doses may be sufficient for long-term efficacy, but further data are needed.

In the short term, the impact of the HPV vaccines is projected to be a reduction in cervical dysplasia. Following the introduction of population-wide HPV vaccination in Australia there has been a significant decline in new cases of genital warts in young women and a modest but significant decrease in high-grade cervical lesions in girls under 18 years in Victoria. There has also been a decline in genital wart presentations in males presenting at sexual health clinics, suggesting herd immunity due to vaccination of girls and young
women#Donovan2011. These ecological data need to be validated by data linkage, but are likely to be early markers of the impact of vaccination.

The expected long-term impact is a reduction in the incidence of cervical cancer and associated mortality. Taken together, this translates to a reduction in treatment costs as well as psychological and medical morbidity#Lowy2006. As the vaccine does not protect against all types of HPV associated with cervical cancer, and may not be effective in women exposed to HPV prior to receiving the vaccine, vaccinated women should continue to have regular Pap tests. Confusion about the protection of HPV vaccination and the need for continuing participation in cervical screening has the potential to lessen compliance with cervical screening in the vaccinated population. Effective communication strategies will be required to ensure vaccinated women still participate in the National Cervical Screening Program.

In the future, as the incidence of cervical abnormalities declines as a result of population-based HPV vaccination, it may be safe to reduce the screening intensity of vaccinated women. See Impact of HPV vaccination on screening.

HPV immunisation in males

Immunising males is expected to further reduce HPV infection rates in women through improved “herd immunity” – an overall reduction in infection rates across the wider community#Georgousakis2012.

In July 2012, the Australian Government announced a plan to include boys on the National HPV Vaccination Program from 2013 #DOHA2012. See Vaccination of boys in the Policy context section for more information.

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Prevention of other cancers

Studies of the potential impact of the prophylactic vaccines on other cancers that are caused by HPV infection are ongoing.

For more information on the link between HPV and cancers other than cervical cancer, see the section on HPV infection and cancer.

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Maximising vaccine uptake

The impact of the HPV vaccination on future cervical cancer incidence and mortality will be influenced by uptake of the vaccine by the population. National HPV vaccination data for girls aged 15 in 2009 shows 70.8% of girls had completed the full course of the HPV vaccine#DoHA2011. Evidence shows higher coverage is possible through similar school-based vaccination programs in the UK, demonstrating room for improved coverage rates#Garland2011b.

The uptake of the HPV vaccines is influenced by the target population’s perceptions of benefits and risks as well as health professionals' attitudes and advice. Therefore communication strategies targeting health professionals, parents, women and adolescents – which are sensitive to culture, religion and age – are required to support uptake. Specific and targeted strategies are necessary to maximise uptake of HPV vaccination by girls in population groups that are at higher risk of cervical cancer and/or have low participation in cervical cancer screening, particularly Aboriginal and Torres Strait Islander women and women of culturally and linguistically-diverse backgrounds.

HPV vaccination has the greatest potential to reduce cervical cancer incidence and associated mortality in under-screened populations with a higher incidence of cervical cancer. There is a strong association between living in areas with a high average socioeconomic status and cervical screening, with the lowest screening rates evident in the in the most disadvantaged areas#Barbaro2012. Conversely, data from Victoria has shown that HPV vaccination coverage has a much less pronounced socioeconomic gradient, with coverage rates only slightly lower in women and girls from the most disadvantaged areas#Barbaro2012.
In Australia, Aboriginal and Torres Strait Islander women are over four times more likely to die of cervical cancer than non-Indigenous women#AIHW_AACR2010. This higher risk is, in part, due to lower participation in the National Cervical Screening Program#Condon2005. Vaccinating Aboriginal girls and women has the ability to reduce the incidence of adenocarcinomas, and incidence of and mortality from cervical cancer#Budd2010. Effective prevention in this population will require a strong understanding of barriers to participation in the immunisation program.

**National HPV Vaccination Program Register**

The National HPV Vaccination Program Register[^2] is a collaboration of the Australian Department of Health and Ageing and the Victorian Cytology Service. It was established through a 2007 act of federal parliament to monitor and evaluate the vaccination program, by recording information about HPV vaccine doses administered in Australia. Its core functions are to:

- Record vaccination doses and key demographic information;
- Develop systems to support the completion of the three-dose vaccination schedule;
- Collect data to monitor and evaluate program participation rates; and
- Inform women when booster doses are required.

The Register will ultimately work towards developing systematic links between the Pap test, cancer registries and the vaccination program, to evaluate the vaccination program's effect on cervical cancer burden in Australia.

**Second-generation prophylactic vaccines**

Despite their efficacy and potential impact, there are some limitations to the current HPV vaccines, including the need for multiple doses, the lack of protection against some types of HPV that cause cervical cancer and, particularly for developing countries, the cost.

Several second-generation vaccines with expanded valency, providing protection against a broader range of HPV types – including a nonavalent (9 HPV targets) vaccine – are currently in phase 3 clinical trials. If successful, such vaccines could prevent most potentially cancer-causing infections, preventing about 90% of cervical cancers. Once efficacy is established, the additional benefits of next generation vaccines will need to be weighed against the cost. Evaluation will also need to consider the potential to further extend the cervical screening interval and commencement age given the expected large-scale reduction in cervical abnormalities.

**Therapeutic vaccine**

Worldwide it is estimated that there are about 100 million women infected with high-risk HPV types, and that five million women have persistent infections that may result in anogenital cancers#Giles2006. Prophylactic HPV vaccines are ineffective against pre-existing HPV infection thus there is an established need for therapeutic vaccines that could clear an existing HPV infection, prevent the development and progression of lesions, and eliminate existing lesions and possibly cancers#Devaraj2003. Recent studies confirm the potential of therapeutic vaccines to enable regression of the most prevalent high-risk genotypes of HPV#Wick2011.

Therapeutic vaccines have the potential to provide less invasive and disfiguring treatment options for women with pre-existing HPV lesions#Brinkman2005, and to decrease treatment costs and psychosocial impact on women. However such vaccines are at least 10 years from market.
Cervical cancer/Screening

Screening

Cervical cancer is one of the few cancers where screening can detect precancerous cell growth. These abnormalities can be treated, preventing the development of cancer.

In Australia, the National Cervical Screening Program (NCSP) using the Pap test began in 1991. Since then, Australia's cervical cancer incidence and death rates have halved.

Implementation of the HPV vaccination program will significantly reduce the average lifetime risk of cervical cancer. Despite the efficacy of the vaccine against the high-risk HPV types that cause most cervical cancers, cervical screening will remain critical in cervical cancer prevention. The vaccine is not effective against existing HPV infections and does not prevent all high-risk HPV types, meaning a significant proportion of cervical cancers will not be prevented.

While cervical screening remains integral to cervical cancer prevention, the NCSP will need to change as the vaccinated cohort ages to maintain its effectiveness and cost-efficiency. There is a substantial body of evidence that screening women younger than 25 years, and screening more frequently than three yearly, does not substantially reduce the incidence of invasive cervical cancer#IARC2005 #Sasieni2009. Accumulating evidence on the value of primary HPV testing and triage by cytology or other tests will also have implications for the NCSP#Brotherton2011b.

The Renewal of the NCSP currently underway will assess the evidence for screening tests and pathways, the screening interval, age range and commencement for both vaccinated and non-vaccinated women, to determine a cost-effective screening pathway and program model.

References

The Pap test

The Pap test (named after its developer, Dr George Papanicolaou) is the most widely used cancer screening test in the world#Eurogin2003. Typically, cervical cancer takes 10 years or more to develop. The value of the Pap test is as a screening tool in a program of re-screening at regular intervals – to detect pre-cancerous abnormalities during this long pre-invasive stage – rather than a single opportunistic test#Dickinson2002. Abnormalities detected by a Pap test can be monitored, or, if required, further investigated and early treatment initiated.

Cervical screening with the Pap test began in British Columbia (Canada) in 1949. Although no randomised controlled trials evaluating screening have been conducted, a large number of observational studies have shown reductions in cervical cancer incidence and mortality over time attributable to screening programs#IARC2005. In Australia, cervical cancer incidence and death rates halved between 1991, when the NCSP began, and 2007#AIHW2012.

Potential benefits

In Australia, the age-standardised incidence rate for cervical cancer declined by an average of 6.2% each year between the introduction of a national screening program in 1991 and 2002. Incidence, after halving from 17.2 new cases per 100,000 women in 1991, has remained at around nine new cases per 100,000 women from 2002 to 2008, for women aged 20-69#AIHW2012. Mortality rates also fell by an average of 5.2% per year from 1991 to 2002. The historical low of around two deaths per 100,000 women has been sustained from 2002 to 2007#AIHW2012. These gains can be attributed, in part, to the success of the NCSP.

The estimated average cumulative lifetime risk of cervical cancer in Australian women with the current screening program is ~0.68%#Creighton2010. However cytological screening has not been as effective in reducing other types of cervical cancer, particularly adenocarcinomas, because of difficulties in sampling and cytological interpretation of glandular cells#Sasieni2009. A recent population-based cohort study of more than 1200 Swedish women with cervical cancer has shown that cervical screening not only reduced the risk for invasive cervical cancer but that women with screen-detected cancers had improved relative survival#Andrae2012. Among women with symptomatic cancer, those who had been screened according to recommendations (presented within the screening interval) had better outcomes than those overdue for screening.

Potential adverse effects

No screening test is 100% accurate. Like all screening tests, the Pap test is performed on asymptomatic women. False positive results may occur. False negative results are common and vary across settings#Cuzick2006. It should be noted that accurate information on false negative rates for cytology against an independent reference standard is not available for Australia.

Even minor abnormalities can cause anxiety for some women. Women who receive false negative results may experience delays in diagnosis or treatment. False negative results may also create a false sense of security that may cause warning symptoms to be ignored.

Abnormal test results can lead to more frequent testing and invasive diagnostic procedures. Risks associated with procedures such as colposcopy and cervical biopsy include vaginal bleeding, pain, infection and failure to diagnose (due to inadequate sampling)#TOMBOLA2009.

In addition to the inherent risks of surgical therapies, some treatments for cervical abnormalities are associated with adverse pregnancy outcomes including preterm delivery, low birthweight and perinatal death#Kyrgiou2006 #Arbyn2008.

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Screening age range and intervals

The current NCSP policy recommends all women who have ever been sexually active should commence having Pap tests between the ages of 18 and 20 years, or within two years after beginning sex, whichever is later#DoHA2008a. A substantial body of evidence has found that screening in women younger than 25 years of age has little or no impact on the risk of developing invasive cancer#Sasieni2009. The International Agency for Research on Cancer recommends regular cervical screening begin at age 25#IARC2005.

In the post-vaccination era it is expected that the risk of cancer in women aged 25 years and under, who were vaccinated prior to exposure to HPV, will be low enough to make screening such young women unjustifiable. Australia’s two-yearly screening interval is conservative, with many countries recommending three years or more between tests. An Australian evaluation has supported international evidence that three yearly screening is safe and screening more frequently does not substantially reduce the number of cervical cancer cases or deaths#Creighton2010. Similar reductions in cervical cancer incidence and mortality have been achieved in Australia, which has a two-yearly screening policy, and the UK, where the policy is predominantly three-yearly screening#Canfell2006. Having reviewed the international evidence the International Agency for Research on Cancer recommends three-yearly screening for women aged 25 to 49 and five-yearly screening for women aged 50 to 64#IARC2005 – a recommendation adopted by the NHS Cervical Screening Programme in the United Kingdom.

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Screening participation

Cervical cytology registers in Australia provide information on the majority of women who undergo screening, although an estimated 1% to 3% of women choose not to be included on the register#AIHW1999. Recent NSW data showed only 0.8% of women ‘opted off’ the NSW register#CINSW2011.

In the two years 2009–10, 57% of the Australian target population of women aged 20 to 69 years participated in cervical cancer screening#AIHW2012, down from 59% in the previous two years (2007-2008). This represented the first true decline in participation in a decade#AIHW2012.

In 2009-10 participation in screening was highest in women aged 45-49 years (63.4%), followed by those aged 40-44 years and 50–54 years (62.3%), and is lower on either side of these age groups (See Table 1)#AIHW2012.

Table 1. Participation in NCSP by age, 2009–2010#AIHW2012

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<td>Women</td>
<td>337,779</td>
<td>418,495</td>
<td>438,861</td>
<td>480,342</td>
<td>442,089</td>
<td>432,082</td>
<td>370,765</td>
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<td>52.2</td>
<td>58.6</td>
<td>61.4</td>
<td>62.3</td>
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<td>59.8</td>
<td>57.2</td>
<td>49.8</td>
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Source: AIHW 2012#AIHW2012

Note: Crude rate is the number of women screened in 2009–10 as a percentage of the ABS estimated resident population for women aged 20–69, adjusted to include only women with an intact cervix using age-specific hysterectomy fractions derived from the AIHW National Hospitals Morbidity Database.

Fourteen per cent of women who received a negative Pap test report in February 2009 were re-screened within 21 months#AIHW2012. Early re-screening increases the cost of the program and reduces cost-effectiveness.

Measuring participation in the NCSP over longer periods reveals higher levels of women having cervical cytology tests. An AIHW analysis of state and territory cervical cytology register data shows that while 57% of the estimated eligible women aged 20–69 years had at least one test in the two years 2009–10, 70.2% had at least one test in the three years 2008–10, and 83.3% participated in the NCSP in the five years 2006–2010#AIHW2012.
**Improving screening rates**
Targeted efforts and better data collection (e.g. routine recording of Aboriginal and Torres Strait Islander status) are needed to further understand barriers to participation in prevention programs and improve outcomes for at-risk populations.

A recent review of studies focused on cancer screening found scant evidence of the effectiveness of particular strategies for targeting 'hard-to-reach' groups of women. For women of culturally and linguistically diverse backgrounds, the most effective strategies appeared to be coaching, community interventions, multi-component interventions and counselling, but the small number of studies focused on engaging Indigenous (Native American, Hawaiian, Canadian and Alaskan) women, women with low income and non-urban women made it difficult to discern trends in effectiveness. Only about half the studies reported a statistically significant increase in uptake. More effort is needed to identify and evaluate strategies, and sustain short-term interventions that appear successful, to increase participation of women in groups at higher risk in Australia.

Self-sampling technologies have proven to be highly acceptable to women and have the potential to improve screening participation rates (see Self-sampling, below).

The NCSP Renewal will consider strategies to improve participation among under-screened women.

**Impact of HPV vaccination on screening**
As successive cohorts of girls are vaccinated, and the vaccinated cohorts mature, the risk of cervical cancer will fall. Prophylactic HPV vaccination is expected to eliminate high-grade intraepithelial lesions and cancers attributable to HPV 16 and 18. Removing the most dangerous (and more cytologically-apparent) cervical abnormalities – and leaving behind the more equivocal ones – may reduce the positive predictive value of cytology. In the era of HPV vaccination, as the population prevalence of HPV 16 and 18 falls, different screening tests and technologies need to be re-evaluated.

Screening will remain necessary, even for vaccinated women. The vaccine does not cover all HPV types that can lead to cervical cancer and may not be effective in women exposed to HPV prior to vaccination. It will take many years for the impact of the vaccine – delivered to adolescent girls – to reduce the incidence of invasive cervical cancer in women in middle age and beyond. Screening policy and protocols, such as age of commencement and screening intervals, will need to be reconsidered as the proportion of vaccinated women increases.

**New screening technologies**
In the past decade, a desire to improve the sensitivity of the Pap test and an increased understanding about the role of HPV has led to the development of new cervical screening technologies. As new technologies are evaluated for adoption, appropriate consideration must be given to how they will improve the cervical screening program in terms of sensitivity, specificity, cost-effectiveness and quality of life. The current Renewal of the NCSP includes assessment of the evidence for various screening tests and pathways.
Cervical cancer/Screening

Liquid-based cytology

Liquid-based cytology is a technique where the cervical cells collected on the sampling instruments are suspended in liquid. At the laboratory the liquid sample is filtered to remove unnecessary material such as blood, bacteria and other matter. The cells are then deposited as a single layer onto a slide, stained and examined under a microscope.

In 2009 the Medical Services Advisory Committee (MSAC) reviewed liquid-based cytology – both automated and with manual reading – and concluded that both were safe and at least as effective as conventional Pap smears, but "not cost effective at the price requested"#MSAC2009a. The evidence suggests that there is no substantial difference between manually read liquid-based cytology and conventional cytology in sensitivity for high-grade disease#Cuzick2006 #Ronco2007 #Siebers2009. However, the MSAC reported that two medium-quality Australian studies have found image read liquid-based cytology (using the ThinPrep Imager system) identified more histologically confirmed CIN2+ lesions than conventional cytology#MSAC2009a.

Sensitivity analyses have demonstrated new technologies are more cost-effective in the context of three-yearly screening. In its assessment report the MSAC noted that as changes due to vaccination are realised "reassessment of the cost-effectiveness of these technologies … would be warranted as part of any review of screening strategies and technologies"#MSAC2009a.

HPV DNA testing

Due to the relationship between persistent infection with high-risk types of HPV and the development of cervical cancer, testing for the presence of (high-risk) HPV DNA in cervical cell specimens has the potential to identify women at increased risk of developing cervical cancer. Research has focused on the application of HPV testing:

- as a 'test of cure' following treatment for high grade precancer;
- in triage of women with possible or low grade cervical abnormalities; and
- in primary cervical screening.

Currently in Australia, HPV DNA testing is recommended for use in women following treatment of a high-grade abnormality. The test is used to determine whether the virus has been cleared from the body#DoHA2008b.

The MSAC assessed HPV triage testing for women with possible or low-grade squamous intraepithelial lesions in 2009, and concluded that compared with repeat recall cytology at one year, it is safe and effective but not cost effective in the Australian setting at the current price of HPV testing#MSAC2009b.

A large body of evidence, including data from randomised trials in developed countries, has shown HPV testing in primary screening is superior to cytology#Franceschi2011, with greater sensitivity but lower specificity for high-grade abnormalities#Cuzick2006 #Bulkmans2007 #Ronco2010, and better reproducibility#Carozzi2005. HPV DNA testing in cervical screening detects high-grade lesions earlier, thus preventing more invasive cervical cancers#Ronco2010 #Rijkaart2012. Using HPV testing also has the potential to improve identification of adenocarcinoma and its precursors#Katki2011. Given HPV types 16, 18 and 45 account for the greatest proportion of infections causing cervical cancer, new screening tests that provide genotyping are expected to improve risk stratification of HPV-positive women in cervical screening programs#Guan2012.

For women who are cytology-negative but HPV-positive, HPV 16-positive or HPV 16/18-positive results are associated with medium-term risk of CIN3 or cancer#Kjaer2010. Women who test HPV negative are at very low risk for CIN3 and cancer for at least five years#Dillner2008 #Katki2011.

There have been some concerns raised about overdiagnosis with HPV testing because it has a lower cross-sectional specificity for high-grade disease when compared to cytology. However, a number of management options have been proposed to deal with this issue. Firstly, HPV testing should not be performed in younger women (under 25 years). Secondly, when used in primary screening, women who are HPV positive can be triaged using either cytology or partial genotyping, so that women are managed on the basis of whether or not HPV types 16, 18 or 45 are present. There are also a variety of newer emerging technologies designed to help triage HPV positive women to
diagnostic evaluation.

Because of its high sensitivity and objectivity, there is growing support for the use of HPV DNA testing in primary screening programs#Bulk2007 #Cuzick2008 #Chen2011. Primary HPV testing could allow future population-based screening to be determined by individual risk assessment#Canfell2010. Adding HPV testing could enable an increase in the recommended age to start screening and a substantial extension of screening intervals#IARC2005 #Dillner2008 #Franceschi2011. Recent research in other developed countries suggests primary HPV DNA testing (for high-risk types) could be cost-effective in unvaccinated women, if an appropriate triage procedure is used in HPV positive women and women are screened at an appropriate age.

**Self-sampling**

Studies show self-collected cervicovaginal samples show promising performance, with sensitivity that may approach that of physician-collected samples for HPV detection#Petignat2007 #Szarewski2007, supporting the potential use of self-sampling in primary cervical cancer screening#Schmeink2011. Self-sampling is highly accepted by women, with studies reporting most women found self-sampling devices easy to use#Jones2008, time saving, less embarrassing and more comfortable#Dzuba2002 #Waller2006.

Self-sampling could increase participation by women who do not participate in current screening programs. Randomised controlled trials and cohort studies in other developed countries have shown that offering self-sampling to women who did not attend regular screening increases participation significantly#Bais2007 #Gok2010 #GiorgiRossi2011 #Szarewski2011 #Virtanen2011 #Wikstrom2011.

Further research is needed to evaluate acceptability of self-sampling by Australian women, effectiveness in increasing participation in screening by women who do not regularly attend screening, and diagnostic pathways for women who are HPV positive.

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**References**

1. Cervical_cancer/References

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Policy context

This section outlines the history and current context of Australian government policy relating to cervical cancer screening and HPV immunisation. It is anticipated that the population-based delivery of the prophylactic HPV vaccine will influence changes to the national screening policy. A Renewal of the National Cervical Screening Program (NCSP) commenced in 2011, including assessment of the evidence for screening tests and pathways, the screening interval and age range. The Renewal is expected to be completed by mid-2014.

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HPV vaccination

Two prophylactic HPV vaccines have been approved by the Therapeutic Goods Administration (TGA) for use in Australia.

In 2006 the TGA approved the quadrivalent HPV vaccine Gardasil for use in women aged nine to 26 and males aged nine to 15. The TGA subsequently approved extension of the age ranges, up to 45 years for females and for males from nine to 26 years. The bivalent vaccine Cervarix was approved by the TGA in 2007 and is registered for use in women aged 10 to 45.

In 2007 Australia commenced the National HPV Vaccination Program to deliver a HPV vaccine (Gardasil) to girls via schools. For the first two years of the program there was also a 'catch-up' program, providing free vaccine to all girls and women up to 26 years of age, through schools as well as general practice and community health centres. Between 2007 and 2009, 72% of girls aged 14 and 15, and nearly 66% of girls aged 16 and 17 received the full three doses need to protect them from HPV. Data from the National HPV Vaccination Catch-up Program shows the coverage rate for women aged 18-19 years who completed the full course was 38% and about 30% of the cohort of women aged 20-26 years completed the full course#DoHA2011.

There is an ongoing school-based vaccination program, offering the vaccine to all girls aged 12-13 years, in the first year of high school or last year of primary school (depending on the state/territory). The Australian Immunisation Handbook [1] provides clinical guidelines for administration of the vaccine. National HPV vaccination data for girls aged 15 in 2009 shows 70.8% of girls had completed the full course of the HPV vaccine#DoHA2011.

The National HPV Vaccination Program Register monitors and reports HPV vaccine coverage. Existing infrastructure such as Pap test registers and cancer registers enables some monitoring of the impact of the HPV vaccination program. However a more comprehensive HPV surveillance program is needed to monitor type-specific HPV infection in the Australian population – to measure reduction in the types targeted by vaccination and any change in other HPV types – as well as incidence of genital warts and recurrent respiratory papillomatosis#Brotherton2010.

See the HPV School vaccination program [1] website for more information about the current vaccination program.
**Vaccination of boys**

In November 2011 the Pharmaceutical Benefits Advisory Committee (PBAC) recommended Gardasil be approved for vaccination of boys aged 12 to 13, plus a two-year catch-up program covering boys in year nine at school.

In July 2012, the Australian Government announced a plan to include boys on the National HPV Vaccination Program from 2013. According to the announcement, the Government will fund the vaccine for 12- and 13-year-old boys through school-based programs under the National Immunisation Program. Year 9 boys and boys aged 14-15 years (who may not be in Year 9) will also be able to get the vaccine through a catch-up program in 2013 and 2014, as per the PBAC recommendations.

**Screening**

Screening for cervical cancer was introduced in Australia on an ad hoc basis in the 1960s. Guidelines on cervical screening programs published in 1986 by the World Health Organization and the International Agency for Research on Cancer were used as a basis for a review of cervical screening in Australia. The review was conducted on behalf of Australian Health Ministers Advisory Council.

Following this review, cervical screening was organised into a structured program known today as the National Cervical Screening Program. The program was implemented in 1991 as a joint initiative of the Australian, state and territory governments, targeting women aged 20 to 69 years.

Women in the target age group are recruited by a variety of initiatives determined mainly at the state/territory level. Recruitment strategies are implemented for particular population sub-groups, such as older women, Australian Aboriginal and Torres Strait Islander women, and women from culturally diverse backgrounds.

State and territory cancer organisations have been involved in a coordinating role in the establishment of state Pap test registries and recruitment of women to the screening program. In some states and territories, Cancer Councils maintain an important role in cervical screening programs.

**Existing recommendations**

The national policy for Australia’s NCSP provides consensus guidelines for which women need screening and how often screening should occur. It states:

- Routine screening with Pap smears should be carried out every two years for women who have no symptoms or history suggestive of cervical pathology.
- All women who have ever been sexually active should start having Pap smears between the ages of 18 and 20 years, or one or two years after first having sexual intercourse, whichever is later.
- Pap smears may cease at the age of 70 years for women who have had two normal Pap smears within the last five years. Women over 70 years who have never had a Pap smear, or who request a Pap smear, should be screened.

Women with abnormal Pap test results should be managed in accordance with the National Health and Medical Research Council guidelines, 'Screening to prevent cervical cancer: guidelines for the management of asymptomatic women with screen detected abnormalities'.

It is anticipated that the introduction of the HPV vaccine will, in the future, influence changes to this national policy. See Impact of HPV vaccination on screening.
National Cervical Screening Program Renewal

The Australian Government, under the guidance of the Australian Health Ministers' Advisory Council's screening subcommittee, is currently conducting a Renewal of the NCSP\(^2\). The Renewal is multi-tiered, allowing for consideration of:

- screening tests and screening pathways;
- the impact of HPV vaccination;
- cost-effectiveness of different screening tests and pathways;
- use of data systems and quality mechanisms; and
- program acceptability.

Ultimately the Renewal will assess the evidence for screening tests and pathways, the screening interval, age range and commencement for both vaccinated and non-vaccinated women, to determine a cost-effective screening pathway and program model.

The first stages of the Renewal will be conducted by evaluation performed by Australia's Medical Services Advisory Committee (MSAC). A decision analytical protocol\(^3\) for the screening approaches to be reviewed by MSAC is now available.

References

1. Cervical_cancer/References

References

Cervical cancer/Policy priorities

Policy priorities

Cancer Council aims to maximise participation in the cervical screening program of eligible women, encourage maximum uptake of prophylactic HPV vaccination by the target population, and support further research and contribute to improvements in both programs.

Table 1. Strategy for reducing cervical cancer burden in Australia

<table>
<thead>
<tr>
<th>Maximise uptake of the prophylactic HPV vaccine</th>
<th>Policy priority/action</th>
<th>Agency</th>
<th>Estimated cost</th>
<th>Expected benefit</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Promote participation in the HPV immunisation program and compliance with the vaccination schedule among adolescents</td>
<td>Department of Health and Ageing</td>
<td></td>
<td>Higher coverage rates and herd immunity leading to lower HPV infection rates</td>
<td></td>
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</tr>
<tr>
<td>Emphasise strategies to facilitate vaccination of girls in population groups that have a higher incidence of and mortality from cervical cancer</td>
<td>Department of Health and Ageing</td>
<td></td>
<td>As above</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Devise clear, and culturally and age-sensitive communication strategies to inform target audiences about HPV and the importance of vaccination</td>
<td>Department of Health and Ageing</td>
<td>Built into program implementation costs</td>
<td>As above</td>
<td>Particularly important with the addition of adolescent males to the immunisation program. An information campaign has been built into the HPV vaccination program for boys</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Implement a comprehensive, evidence-based cervical screening prevention program</th>
<th>Policy priority/action</th>
<th>Agency</th>
<th>Estimated cost</th>
<th>Expected benefit</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ensure expert advice informs the Renewal of the National Cervical Screening Program (NCSP)</td>
<td>The Renewal Steering Committee, Department of Health and Ageing</td>
<td>Low cost</td>
<td>Safe, effective and cost-effective cervical cancer prevention</td>
<td>The NCSP Renewal undertook consultation with a wide range of partners, including health professionals, scientists and consumers in March 2012</td>
<td></td>
</tr>
</tbody>
</table>
### Timely implementation of Renewal recommendations

Australian Health Ministers’ Advisory Council (AHMAC)  
Dependent on Renewal recommendations. As an example, transition to three-yearly screening could lead to a cost saving of $10-18 million annually#Creighton2010

### Develop communications strategies to ensure target populations and health professionals understand and participate in renewed cervical cancer prevention program

AHMAC  
Built into program implementation costs  
As above

## Maximise screening participation of eligible women

<table>
<thead>
<tr>
<th>Policy priority/action</th>
<th>Agency</th>
<th>Estimated cost</th>
<th>Expected benefit</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Promote and foster participation in cervical cancer screening, particularly among women in under-screened populations, including collaboration with Indigenous communities</td>
<td>AHMAC, Indigenous groups, other representatives of under-screened populations</td>
<td></td>
<td>Improved cervical cancer incidence and mortality rates in high-risk groups</td>
<td></td>
</tr>
<tr>
<td>Develop communication strategies to ensure women are aware of the need and recommended frequency of screening for both HPV-vaccinated and unvaccinated women</td>
<td>AHMAC, health professionals</td>
<td></td>
<td>Improving both under- and over-screening rates, and thus cost-effectiveness of the NCSP</td>
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</table>

## Ongoing monitoring and evaluation of cervical screening and HPV immunisation programs

<table>
<thead>
<tr>
<th>Policy priority/action</th>
<th>Agency</th>
<th>Estimated cost</th>
<th>Expected benefit</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Develop a national (real or virtual) cervical screening register, and linkage to the National HPV Vaccination Program Register</td>
<td>Department of Health and Ageing, AHMAC</td>
<td></td>
<td>Improved program implementation and data on program efficacy</td>
<td></td>
</tr>
<tr>
<td>Implement a national HPV surveillance program, including accurate and internationally comparable HPV DNA genotyping of all CIN3 lesions</td>
<td>Health Protection and Surveillance Branch, Office of Health Protection, Department of Health and Ageing</td>
<td></td>
<td>Evaluate HPV vaccines and impact of vaccination programs</td>
<td></td>
</tr>
<tr>
<td>Encourage routine recording of Indigenous status</td>
<td>AHMAC, health professionals</td>
<td>Low cost</td>
<td>Will enable evaluation of effectiveness of programs and of targeted efforts</td>
<td></td>
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## Further research related to cervical cancer prevention in Australia

<table>
<thead>
<tr>
<th>Policy priority/action</th>
<th>Agency</th>
<th>Estimated cost</th>
<th>Expected benefit</th>
<th>Comments</th>
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</thead>
<tbody>
<tr>
<td>Fund further research to produce the evidence needed to inform practice and policy development in Australia</td>
<td>National Health and Medical Research Council and other funders</td>
<td>Modest, funded on a project by project basis</td>
<td>Evidence informed policy to improve current cervical cancer prevention programs</td>
<td></td>
</tr>
</tbody>
</table>
References


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