Meeting Report

Human papillomavirus vaccines: WHO position paper

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1. Background

This is the first WHO position paper on vaccines against diseases caused by human papillomaviruses (HPVs). To complement the text and the selected references offered in this document, a comprehensive WHO background paper provides additional information and references reflecting evidence available to the end of September 2008 [1]. The final section of this position paper provides links to 5 grading tables for scientific evidence and their accompanying references.

1.1. The epidemiology of HPV and HPV-related diseases

Genital HPV infections are primarily transmitted by sexual contact, predominantly but not exclusively through penetrative intercourse. HPVs are highly transmissible, and most sexually active men and women will acquire an HPV infection at some time in their lives. Whereas most HPV infections are transient and benign, persistent genital infection with certain viral genotypes can lead to the development of anogenital precancers and cancers.

Diseases caused by HPVs include cancers of the cervix, vagina, vulva, penis and anus; a subset of head and neck cancers; anogenital warts; and recurrent respiratory papillomatosis. In 2005, there were about 500 000 cases of cervical cancer and 260 000 related deaths worldwide. Cervical cancer incidence rates vary from 1 to 50 per 100 000 females; rates are highest in Latin America and the Caribbean, sub-Saharan Africa, Melanesia, and south-central and South-East Asia. Most cases of cervical cancer are diagnosed in women aged >40 years [2].

Countries with well-organized programmes to detect and treat precancerous abnormalities and early stage cervical cancer can prevent up to 80% of these cancers. However, effective screening programmes and follow-up of women with abnormal screening tests have been difficult to implement in low-resource and middle-resource settings. Mortality rates from cervical cancer are therefore much higher in the developing world.

Vulvar, vaginal, penile and anal cancers, and precancerous lesions are relatively rare, and most of these cancers occur in adults aged >50 years. HPVs are estimated to cause at least 80% of anal cancer and at least 40–60% of vulvar, vaginal and penile cancers.

Genital warts are common among sexually active people and usually first occur in adolescence or young adulthood. Estimates of the global incidence of anogenital warts are not available but prevalence is believed to be high in all parts of the world, especially in people infected with HIV.

1.2. The viruses

HPVs are non-enveloped, double-stranded deoxyribonucleic acid (DNA) viruses in the family of Papillomaviridae. The HPV genome is enclosed in a capsid shell comprising major (L1) and minor (L2) structural proteins.

More than 100 HPV genotypes are known. Certain HPV genotypes are associated with cell immortalization and transformation related to carcinogenesis. Of these, at least 13 may cause cervical cancer or are associated with other anogenital and oropharyngeal cancers. HPV types 16 and 18 cause about 70% of all cases of invasive cervical cancer worldwide, with type 16 having the greatest oncogenic potential. The distribution of HPV types varies among geographical regions, but the dominant oncogenic type in all regions is HPV-16 [3]. Some genotypes rarely cause cancer but may cause benign or...
low-grade changes in cervical cells that may be indistinguishable by cytology or histology from those caused by HPV types with higher oncogenic potential. The low-risk HPV types 6 and 11 are responsible for about 90% of anogenital warts and almost all recurrent respiratory papillomatosis.

1.3. Immunology, pathology and diagnosis

HPV infections are restricted to the intraepithelial layer of the mucosa and do not induce a vigorous immune response. Approximately half of all women infected with HPV develop detectable serum antibodies, but these antibodies do not necessarily protect against subsequent infection by the same HPV type. The best characterized and most type-specific HPV antibodies are those directed against the L1 protein of the virus. The median time from infection to seroconversion is approximately 8–12 months, although immunological response varies by individual and HPV type.

Persistent HPV infection may lead to cervical intraepithelial neoplasia (CIN) of moderate (2) grade or severe (3) grade or to adenocarcinoma in situ (AIS), a precancerous lesion involving cervical glandular cells. If untreated, CIN2–3 has a high probability of progressing to squamous cell cancer, and AIS has a high probability of progressing to adenocarcinoma. The time between initial HPV infection and development of cervical cancer averages 20 years.

HPV-induced changes in the cervical epithelium can be detected by cytology using a microscopic examination of exfoliated cells, which is also known as a Papanicolaou (Pap) test. Persistent HPV infection can be diagnosed by repeated tests for HPV DNA. Cytology or testing for HPV DNA, or both, is used for cervical cancer screening and diagnostic follow-up in many countries. In low-resource settings that lack a complex health infrastructure, visual inspection of the cervix with acetic acid or Lugol’s iodine is used to identify cervical lesions, which can be immediately treated by cryotherapy.

2. HPV vaccines

Currently, 2 HPV vaccines are widely marketed internationally. Using recombinant technology, both are prepared from purified L1 structural proteins that self-assemble to form HPV type-specific empty shells or virus-like particles (VLPs). Neither vaccine contains live biological products or viral DNA, so they are non-infectious. HPV vaccines are designed for prophylactic use only; they do not clear existing HPV infection or treat HPV-related disease [4]. The mechanisms by which these vaccines induce protection have not been fully defined but seem to involve both cellular immunity and neutralizing immunoglobulin G antibodies [5,6]

2.1. The quadrivalent vaccine

The quadrivalent vaccine, which was first licensed in 2006, contains VLPs for HPV types 6, 11, 16 and 18. The vaccine is produced using yeast substrate and includes amorphous aluminium hydroxyphosphate sulfate as adjuvant. Each 0.5 mL dose of this vaccine contains 20 μg of HPV-6 L1 protein, 40 μg of HPV-11 L1 protein, 40 μg of HPV-16 L1 protein and 20 μg of HPV-18 L1 protein adsorbed onto 225 μg of the adjuvant. The formulation contains no antibiotics, thiomersal or other preservatives. This vaccine has been licensed for use in young adolescent girls (as young as 9 years of age in some countries) to prevent cervical precancers and cancers and anogenital warts in females. In addition, the quadrivalent vaccine is licensed for prevention of vulvar and vaginal precancers and cancers as well as anogenital warts in females. In some countries, the vaccine is also licensed for the prevention of anogenital warts in males.

2.2. The bivalent vaccine

The bivalent vaccine, which was first licensed in 2007, contains the VLPs of HPV types 16 and 18. It is produced using a novel baculovirus expression system in Trichoplusia ni cells. Each 0.5 mL dose of the bivalent vaccine contains 20 μg of HPV-16 L1 protein and 20 μg of HPV-18 L1 protein adsorbed onto a proprietary ASO4 adjuvant system containing 500 μg of aluminium hydroxide and 50 μg of 3-O-desacyl-4′-monophosphoryl lipid A. The vaccine contains no thiomersal, antibiotics or other preservatives. This vaccine has been licensed for use in females as young as 10 years of age to prevent cervical precancers and cancers. Registration for indications in males has not been sought.

2.3. Storage, administration and schedules

Both the bivalent and quadrivalent vaccines are available as a sterile suspension in single-use glass vials or single-use pre-filled syringes that should be maintained at 2–8 °C and not frozen. A 2-dose presentation is also available for the bivalent vaccine. The vaccines are to be administered only through intramuscular injections as doses of 0.5 mL each.

Current single-dose presentation and packaging of both vaccines result in a higher per-dose volume than multi-dose vaccines commonly used in childhood.

Both vaccines are intended to be administered to females before the onset of sexual activity — that is, before first exposure to HPV infection. Most countries that have licensed these vaccines, recommend their use in girls aged 10–14 years. Some national programmes also recommend routine or temporary catch-up vaccination of older adolescent females and young women.

The quadrivalent vaccine is given at baseline and again after 2 months and 6 months. A minimum interval of 4 weeks between the first and second dose, and a minimum interval of 12 weeks between the second and third dose, are recommended by the manufacturer if flexibility in the schedule is necessary [7].

The bivalent vaccine is given at baseline and again after 1 month and 6 months. If flexibility in the schedule is necessary, the manufacturer recommends that the second dose be administered between 1 and 2.5 months after the first dose [8].

Alternative schedules are being explored for both the bivalent and quadrivalent vaccines. Restarting the 3-dose series is not necessary if the programme has been interrupted,
but remaining vaccine doses should be administered as close to the recommended schedule as possible. Currently, the manufacturers do not recommend a booster dose following completion of the primary series.

2.4. Immunogenicity studies

With both vaccines, practically all adolescent and young female vaccinees who were initially naive to vaccine-related HPV types developed an antibody response to these antigens after 3 doses [9,10]. Data available up to 5–6.4 years after vaccination have shown that antibody titres peak after the third dose, decline gradually and then level off by 24 months after the first dose. Geometric mean titres (GMTs) of serum antibodies from adolescents aged 10–15 years were higher than titres in sera from older females (aged 16–23 years for the quadrivalent vaccine and 15–25 years for the bivalent vaccine).

To date, information on the immune response to HPV vaccination in HIV-infected individuals is limited to a study of 120 children aged 7–11 years in the United States, some of whom used antiretroviral therapy. Of these children, >99.5% developed antibodies against HPV types 6, 11, 16 and 18 when immunized with the quadrivalent vaccine (Weinberg A et al., unpublished data, 2008). GMTs for all 4 HPV types were lower among HIV-infected children than among non-HIV-infected historical controls of similar age, but differences were statistically significant only for HPV types 6 and 18. Data on the immunogenicity of the bivalent vaccine in young people infected with HIV are not yet available.

Co-administration of the quadrivalent vaccine with a recombinant hepatitis B vaccine (in females aged 16–23 years) or a combined diphtheria—tetanus—pertussis—inactivated poliomyelitis vaccine (in females and males aged 11–17 years) and co-administration of the bivalent vaccine with a combined diphtheria—tetanus—pertussis—inactivated polio-myelitis vaccine (in females aged 10–18 years) did not significantly impair the immune response to any of the involved antigens. Studies of the co-administration of both HPV vaccines with other vaccines are ongoing.

2.5. Clinical efficacy and duration of protection

Since the immunological correlates of vaccine protection are unknown and the development of cervical cancer may occur decades after HPV infection, regulatory authorities have accepted the use of CIN grade 2 or 3 (CIN2–3) and AIS as clinical end-points in vaccine efficacy trials instead of invasive cervical cancer [11]. Also, using cervical cancer as the outcome in such trials is precluded for ethical reasons. Precancerous lesions usually develop <5 years after HPV infection.

Multicentre, randomized, double-blind phase II and III trials that examined the clinical end-points CIN2, CIN3 and/or AIS were conducted in females aged 15–26 years for the quadrivalent vaccine [12] and in females aged 15–25 years for the bivalent vaccine [13]. Phase II and III trials of the quadrivalent vaccine also examined the clinical end-points of anogenital warts and vulvar and vaginal intraepithelial neoplasia in females aged 15–26 years [14]. Collecting cervical specimens from girls or young adolescents is usually considered unethical or impractical. Results of immunobridging studies comparing vaccine immunogenicity in females aged 9–14 years with that in females aged 15–26 years were therefore used to infer clinical efficacy in the younger age group (see “Immunogenicity studies” above).

Data on the efficacy of the HPV vaccines against CIN2–3 outcomes are not yet available for HIV-infected individuals.

Both vaccines appear to have partial efficacy against infections caused by HPV types 31 and 45, which are genetically related to types 16 and 18 [15,16]. The protective efficacy of the 2 vaccines has been maintained throughout their respective observation periods, currently extending to 6.4 years (bivalent vaccine) [16] and 5 years (quadrivalent vaccine) [14].

Differences among the efficacy trials of the quadrivalent and bivalent vaccines in terms of choice of placebo recipients or control subjects, immunological assays and populations analysed preclude direct comparison of results for the 2 vaccines.

2.6. Data on the quadrivalent vaccine

An investigation that included 5455 women aged 16–24 years studied the protective efficacy of the quadrivalent vaccine against CIN2 or CIN3 and AIS caused by HPV-16 or HPV-18. Among females naive to HPV-16 or HPV-18 for up to 1 month following the third dose of vaccine, protection against these combined end-points was 100% (95% confidence interval [CI], 94–100%) after a mean follow-up of 3 years [17]. Another phase III study of women aged 15–26 years followed for a mean of 3 years after the first dose found efficacy against CIN2 or CIN3 and AIS caused by HPV-16 or HPV-18 of 98% (95% CI, 86–100%) [18]. The results of 2 phase III studies that enrolled a total of 17 622 females aged 15–26 years who were naive to 1–3 vaccine-related types at baseline showed that after 3 doses and an average observation period of 3 years, the quadrivalent vaccine was 100% effective (95% CI, 79–100%) against the combined end-point of CIN2 or CIN3 and AIS caused by the HPV type or types for which the women were negative at enrolment [12]. In a phase II study that was extended through to 5 years after enrolment, vaccine efficacy against CIN1–3 caused by HPV types 6, 11, 16 or 18 and anogenital warts among 241 women naive to these 4 types at enrolment was 100% (95% CI, 12–100%) [14].

A combined analysis of the above phase II trial of the quadrivalent vaccine, 1 phase II trial of a monovalent HPV-16 vaccine and the 2 phase III trials of the quadrivalent vaccine (mentioned above) reported an efficacy of 99% (95% CI, 93–100%) for the composite end-point of CIN2 or CIN3 or AIS after 3 years of follow-up among women naive to the relevant type at baseline who had received all 3 doses [4].

Reports of HPV immunization in males naive to vaccine genotypes showed that the quadrivalent vaccine was 86% effective (95% CI, 75–92%) in preventing persistent infection and 90% (95% CI, 69–98%) effective against external lesions caused by vaccine-related HPV types. These results are based on
a randomized, double-blind, placebo-controlled trial involving 4065 men aged 16–26 years with a 3-year follow-up period [19,20].

2.7. Data on the bivalent vaccine

The efficacy of the bivalent HPV vaccine in the prevention of vaccine-related HPV types CIN2–3 was assessed in a phase III study that included 18 644 women aged 15–25 years. After a mean follow-up period of 14.8 months, vaccine efficacy was 90% (95% CI, 53–99%) in preventing CIN2–3 due to HPV type 16 or type 18 [13]. These interim analyses were done on a modified intention-to-treat basis — that is, they included women who had received at least 1 vaccine dose and who were naive to either vaccine type 16 or type 18 at baseline.

An extended phase II study followed 776 females aged 15–25 years for 6.4 years after the first dose. The bivalent vaccine provided an efficacy of 100% (95% CI, 51–100%) against HPV-16 and HPV-18 related CIN2–3 among women who received at least 1 dose and were naive to the relevant HPV type at baseline [16]. Also, high vaccine efficacy against CIN2–3 caused by HPV-16 and HPV-18 was reported in females aged 15–25 years who were naive to 14 oncogenic types (including HPV types 16 and 18) at baseline. In this post-hoc analysis (in which the type-specific etiology of CIN2–3 lesions that included multiple HPV types was classified according to the type of persistent infection before diagnosis, only lesions in which persistent HPV types 16 or 18 were found before diagnosis were classified as cases), 100% effectiveness against CIN2–3 (95% CI, 67–100%) was found among the subset followed for 15 months after the first dose in the phase III trial; 100% effectiveness (95% CI, 33–100%) was also found among the smaller subset followed for 5.5 years after the first dose in a phase II trial [21].

2.8. Reactogenicity and safety

In clinical trials, mild and transient local reactions at the site of injection (erythema, pain or swelling) were 10–20% more frequent among those who received the current HPV vaccines than in their respective control groups, but no systemic adverse reactions assessed to be causally associated with the HPV immunization have been reported. Limited data do not suggest serious adverse outcomes following immunization of HIV-positive children with quadrivalent vaccine or when either of the HPV vaccines were inadvertently administered to pregnant women. Selecting target ages for HPV vaccination that usually precede the onset of sexual activity reduces the likelihood of inadvertently vaccinating pregnant or lactating females.

In June 2007, WHO’s Global Advisory Committee on Vaccine Safety (GACVS) concluded that both vaccines had good safety profiles [22]. In December 2008, GACVS reviewed data on early post-marketing surveillance of the quadrivalent HPV vaccine. No reports raised sufficient concern to change previous advice given by GACVS [23].

2.9. Contraindications and precautions

HPV vaccines should not be given to people who have experienced severe allergic reactions after a previous vaccine dose or to a component of the vaccine. Several countries recommend that HPV vaccination should be delayed for individuals who have severe acute illness. There is no evidence of elevated risk for syncope following HPV vaccine, but post-licensure findings support an increased occurrence of post-vaccination syncope among adolescent vaccinees. Observation of vaccinees for 15 min after the injection is administered is recommended.

HPV vaccines are not recommended for use in pregnant females. The quadrivalent HPV vaccine may be administered to lactating females because available data do not indicate any safety concerns. Safety data for lactating women are not available for the bivalent vaccine.

2.10. Model projections on impact and cost-effectiveness of HPV vaccination

The population impact of HPV vaccination programmes in preventing cervical precancerous lesions and cancer, abnormal cytology that requires follow-up and utilization of health-care services has been estimated for both vaccines, usually using models that consider a prototype vaccine with VLPs of HPV-16 and HPV-18. Models of the quadrivalent vaccine have also evaluated the impact on outcomes related to HPV-6 and HPV-11, including anogenital warts and low-grade cervical abnormalities.

Models predict that vaccination programmes for young adolescent females (defined as being roughly within the range of 10–13 years) will substantially reduce the incidence of cervical cancers associated with vaccine-related HPV types if coverage is high (>70%) and vaccine-induced protection lasts for ≥10 years. Considerable reductions in incidence may also be expected for the less frequent cancers of the vagina, vulva, anus, and head and neck associated with HPV-16 and HPV-18. Depending on assumptions related to vaccination and screening programmes, vaccination could reduce the lifetime risk of cervical cancer by 35–80%.

Models estimate that the reduction in the incidence of cervical cancer and mortality will be greatest in low-income and middle-income countries where there is no screening or only limited screening for cervical cancer. If vaccine uptake is highest in populations who are most likely to be screened later in life, reductions in cervical cancer attributed to vaccination may be less than expected because the diseases prevented by vaccination would otherwise have been detected and treated [24]. Models also predict that vaccination with the quadrivalent vaccine will substantially reduce the incidence of anogenital warts, low-grade cervical abnormalities caused by HPV-6 and HPV-11 and, possibly, recurrent respiratory papillomatosis, if coverage is high and vaccine protection lasts for ≥10 years.

Since HPV vaccines are prophylactic, the largest impact of vaccination is expected to result from high coverage of young
adolescent girls before first intercourse rather than from vaccinating older females, because a smaller proportion of older females would be naïve to vaccine-related types before vaccination. Most models’ predictions suggest that with either vaccine, male HPV vaccination will have a limited impact on the incidence of cervical cancer [24–27].

Cost-effectiveness models rely on uncertain assumptions and parameters that may strongly influence results and thus should be interpreted cautiously. In general, models show that programmes that achieve high coverage in young adolescent girls may greatly reduce costs associated with cervical cancer screening, follow-up of abnormal screening tests, and diagnosis and treatment of precancers and cancer. A systematic review of articles published before August 2007 related to cost-effectiveness analyses of a wide range of HPV vaccination programmes compared with Pap smear screening was recently conducted. Based on the WHO guideline that compares incremental cost-effectiveness ratios with per capita gross domestic product (GDP), it was concluded that nationwide administration of HPV vaccine would be cost-effective only in countries where GDP is high [28]. Several models indicate that HPV vaccination in low-income and middle-income countries where quality screening is not widespread may be cost-effective if the cost per vaccinated girl (including 3 doses of vaccine and programmatic costs) is <US$ 10–25 — that is, substantially lower than current costs in high-income countries [29]. In high-income settings, quadrivalent HPV vaccination is expected to reduce costs associated with the diagnosis and treatment of genital warts.

3. WHO position on HPV vaccines

WHO recognizes the importance of cervical cancer and other HPV-related diseases as global public health problems and recommends that routine HPV vaccination should be included in national immunization programmes, provided that: prevention of cervical cancer or other HPV-related diseases, or both, constitutes a public health priority; vaccine introduction is programmatically feasible; sustainable financing can be secured; and the cost-effectiveness of vaccination strategies in the country or region is considered.

HPV vaccines are most efficacious in females who are naïve to vaccine-related HPV types; therefore, the primary target population should be selected based on data on the age of initiation of sexual activity and the feasibility of reaching young adolescent girls through schools, health-care facilities or community-based settings. The primary target population is likely to be girls within the age range of 9 or 10 years through to 13 years [30,31].

Programmes introduced to prevent cervical cancer should initially prioritize high coverage in the primary target population of young adolescent girls. Vaccination of secondary target populations of older adolescent females or young women is recommended only if this is feasible, affordable, cost-effective, does not divert resources from vaccinating the primary target population or effective cervical cancer screening programmes, and if a significant proportion of the secondary target population is likely to be naïve to vaccine-related HPV types. HPV vaccination of males is not recommended because vaccination strategies that achieve high coverage (>70%) in the primary target population of young adolescent girls are expected to be more cost-effective in reducing cervical cancer than including the vaccination of males [32].

Data on the safety of HPV vaccination in pregnancy are limited, and HPV vaccination of pregnant women should be avoided. However, no adverse events causally associated with the vaccine have been observed in mothers or their offspring following inadvertent vaccination during pregnancy. Data do not indicate that any safety concerns have arisen following administration of the quadrivalent HPV vaccine to lactating females. Corresponding safety information is not available for the bivalent vaccine. Selecting target ages for HPV vaccination that usually precede the onset of sexual activity reduces the likelihood of inadvertently vaccinating pregnant or lactating females.

Little information is available on the safety and immunogenicity of HPV vaccines in people who are immunocompromised due to medications or diseases. Although the immunogenicity and efficacy of HPV vaccines may be reduced in HIV-infected females, the potential benefit of vaccination in this group is particularly great owing to their increased risk of HPV-related disease, including cervical cancer. Most target populations for HPV immunization are likely to include a few HIV-infected individuals, even in areas with a relatively low prevalence of HIV. Concerns about safety or reduced efficacy among females who may be infected with HIV should not defer the initiation of large-scale HPV immunization. HIV testing should not be a prerequisite before routine HPV immunization [33].

Both vaccines should be administered according to their manufacturer’s specifications, schedules and advice on interrupted schedules. Clinical efficacy trials for both vaccines demonstrate that protection lasts for at least 5 years. A need for booster doses has not been established [34].

In settings where both HPV vaccines are marketed, the choice between the 2 should be based on the assessment of a number of factors, including the scale of the prevailing HPV problem (cervical cancer, other anogenital cancers, or anogenital warts); the population for whom the vaccine has been approved (girls aged 9 or 10 years through to 13 years, or older females, women, and/or males); delivery strategies; data on vaccine efficacy against HPV-related diseases; and safety in specific subpopulations eligible for vaccination. The data available to decision-makers differ by vaccine. Decision-makers should also consider unique product characteristics, such as price, supply and cold-chain requirements.

Data are not available on the safety, immunogenicity or efficacy of the 2 marketed HPV vaccines when used interchangeably. These vaccines have different characteristics, components and indications, and in settings where both may be marketed, every effort should be taken to administer the same vaccine for all 3 doses. However, if the vaccine used for prior doses is unknown or unavailable, either of the marketed HPV vaccines can be administered to complete the schedule.
Both HPV vaccines are non-live and non-infectious and can be co-administered with other non-live and live vaccines using separate syringes and different injection sites.

Several delivery strategies are possible. Countries should use approaches that are compatible with their delivery infrastructure and cold-chain capacity; are affordable, cost-effective and sustainable; and that achieve the highest possible coverage. In several countries, school-based delivery appears promising. If countries consider phased introduction, priority should be given to strategies that include populations who are likely to have less access to screening for cervical cancer later in life.

HPV vaccines should be introduced as part of a coordinated strategy to prevent cervical cancer and other HPV-related diseases. This strategy should include education about reducing behaviours that increase the risk of acquiring HPV infection, and information about the diagnosis and treatment of precancerous lesions and cancer. Also, the introduction of HPV vaccine should not undermine or divert funding from effective screening programmes for cervical cancer. HPV vaccination is a primary prevention tool and does not eliminate the need for screening later in life, since HPV types other than 16 and 18 cause up to 30% of all cases of cervical cancer. Opportunities to link the introduction of HPV vaccine to other programmes targeting young people should be sought (for example, through adolescent health services). However, vaccination should not be deferred in countries because at least 1 of these interventions cannot be implemented at the time when vaccination could be introduced.

After HPV vaccination programmes are introduced, coverage by individual, age and district should be measured and records retained for the long term. As with the introduction of any new vaccine, arrangements should be in place to monitor safety. Countries should consider establishing sentinel surveillance to monitor the impact of vaccination on the prevalence of HPV types, the incidence of cervical abnormalities and precancerous lesions, the incidence of and mortality from invasive cancer, and the incidence of anogenital warts. Measuring the impact of vaccination on precancerous lesions and cervical cancer will require monitoring for decades.

Educational messages and notification, approval, or consent of patients or parents should be tailored to local cultural contexts and the information needs of various audiences, including those who are targeted for vaccination, their parents or guardians, educators, community leaders and health-care providers. Messages should emphasize that HPV vaccines do not cure cancer; they prevent some, but not all, HPV-related cancers; they are most effective when given before the onset of sexual activity; they require 3 doses; they are not recommended for pregnant females; and they will not prevent HIV infection, other sexually transmitted infections or pregnancy.

Messages about quadrivalent vaccine programmes could include information about the benefits of preventing genital warts. Vaccinees should be advised to seek cervical cancer screening later in life. Because public knowledge about cervical cancer and its association with HPV is limited in many countries, community education campaigns about cervical cancer and HPV are recommended as a strategy for increasing vaccine acceptance.

References


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