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HPV VACCINATION AND CERVICAL CANCER PREVENTION: A GLOBAL SYSTEMATIC REVIEW

Dr. Sobia Fatima[1], Dr. Asma Muzaffar[2], Dr. Muhammad Usama Hashmi[3], Dr. Misbah Aziz[4], Dr. Munnawar Haque[5], Dr. Naromal[6], Dr. Amber Shams[7]*

[1] MBBS, MSPH, Dow University of Health Sciences, Karachi, Pakistan; MSPH, SZABIST University, Karachi, Pakistan.
[2] MBBS, FCPS (Obstetrics & Gynaecology), Nishtar Medical University, Multan, Pakistan.
[3] MBBS, Jinnah Sindh Medical University, Karachi, Pakistan.
[4] MBBS, FCPS (Obstetrics & Gynaecology).
[5] BDS, MPH (USA), CHPE; Senior Lecturer, Indus College of Family Medicine and Public Health, Indus University of Health Sciences, Indus Hospital & Health Network, Karachi, Pakistan.
[6] MBBS, MPH; Senior Lecturer, Department of Community Medicine, Liaquat Institute of Medical & Health Sciences, Thatta, Sindh, Pakistan. MBBS: Liaquat Medical College, Jamshoro, Sindh, Pakistan. MPH: Baqai Medical University, Karachi, Sindh, Pakistan.
[7]MBBS – Liaquat University of Medical & Health Sciences (LUMHS), Jamshoro, Pakistan; Professional Diploma in Obstetrics & Gynaecology – Royal College of Physicians of Ireland (RCPI), Ireland.
*Corresponding Author: Email: drambershams@gmail.com

ABSTRACT

Objective: To synthesize global evidence on the effectiveness of prophylactic human papillomavirus vaccination for preventing persistent vaccine-type infection, high-grade cervical intraepithelial neoplasia and invasive cervical cancer and to examine how age at vaccination, dose schedule, programme coverage and socioeconomic context modify population impact.

Study design: Systematic review of randomized evidence, population-based cohorts, registry analyses and programme evaluations.

Place and duration of the study: Global evidence indexed from database inception was reviewed from February 2025 to may 2026.

Methodology: A sensitive MEDLINE/PubMed search was supplemented by citation tracking and authoritative global reports. Eligible reports evaluated prophylactic HPV vaccination and cervical HPV infection, CIN2+, CIN3 or invasive cervical cancer. Two-stage title/abstract and full-text assessment was applied. Results were synthesized by outcome, age at vaccination, dose and setting because designs and estimands were heterogeneous.

Results: The initial MEDLINE/PubMed search identified 12,064 records and 32 publications met the focused eligibility criteria. In Sweden, adjusted invasive cervical cancer incidence was lower among vaccinated women (incidence rate ratio 0.37, 95% CI 0.21–0.57) and was lowest when vaccination occurred before age 17 years (0.12, 95% CI 0.00–0.34). England recorded an 87% (95% CI 72–94) reduction in cervical cancer and a 97% (95% CI 96–98) reduction in CIN3 among women offered vaccination at age 12–13 years. Scotland recorded no invasive cancers among women immunized at age 12–13 years. Recent English follow-up estimated 687 (95% CI 556–819) cervical cancers and 23,192 (95% CI 22,163–24,220) CIN3 cases prevented by mid-2020.

Conclusion: HPV vaccination provides strong real-world protection against cervical precancer and invasive cervical cancer. The largest benefit occurs with vaccination before sexual exposure. High coverage, equitable delivery and continued HPV-based screening remain necessary for global elimination.

KEYWORDS: Human papillomavirus; HPV vaccine; cervical cancer; cervical intraepithelial neoplasia; vaccine effectiveness; systematic review

INTRODUCTION

Persistent infection with oncogenic human papillomavirus (HPV) is the necessary initiating event for almost all cervical cancers. HPV16 and HPV18 produce most invasive disease although the proportional contribution of individual genotypes varies by region. Cervical cancer remains a preventable cause of premature mortality because exposure occurs early, progression is usually slow and both primary and secondary prevention are available. Recent global analyses continue to show a concentrated burden in countries where organized vaccination, high-performance screening and timely treatment are least accessible [1]. The mismatch between biological preventability and continuing deaths makes vaccine implementation a health-system issue rather than only an immunization question.

Prophylactic vaccines based on virus-like particles prevent acquisition of vaccine-type infection and the precursor lesions that precede cancer. Bivalent, quadrivalent and nonavalent formulations cover progressively broader oncogenic types. Population benefit depends on delivery before sexual exposure, completion or effectiveness of the recommended schedule, sustained programme coverage and screening of cohorts already exposed to HPV [2]. Current evidence also supports single-dose strategies for immunocompetent adolescents in many settings. These

strategies may reduce cost, cold-chain pressure and missed second doses. Long-term protection and programme surveillance remain important because invasive cancer appears years after infection [3].

The evidence base has moved beyond immunogenicity and cervical infection. Large registry-linked cohorts now demonstrate reductions in CIN3 and invasive cancer. Yet evidence is unevenly distributed: high-income countries contribute most direct cancer endpoints whereas low- and middle-income countries carry the greatest burden and often report coverage, acceptability or modelling outcomes. Age at vaccination, deprivation and programme structure further influence observed effectiveness [4]. Pakistan and similar settings face an additional evidence gap because national rollout, reliable vaccine registries and linkage with cervical cancer surveillance are limited. International effect estimates therefore require cautious translation into local delivery policy [5].

This systematic review assessed the global effectiveness of prophylactic HPV vaccination for preventing cervical HPV infection, high-grade cervical lesions and invasive cervical cancer. Secondary objectives were to examine age and dose effects, identify equity-related differences and define programme conditions required for cervical cancer elimination. The review was designed to preserve reported estimates rather than recalculate incompatible effect measures [6-13].

MATERIALS AND METHODS

A systematic review was conducted and reported according to PRISMA 2020 principles. The review question used a population-intervention-comparator-outcome structure: girls and women eligible for prophylactic vaccination; receipt or programme offer of any licensed HPV vaccine; unvaccinated, historical or differently exposed comparators; and vaccine-type infection, CIN2+, CIN3, adenocarcinoma in situ or invasive cervical cancer. The setting was global and the review work was completed from February 2025 to may 2026.

MEDLINE/PubMed was searched from database inception. The core strategy combined ‘HPV’ or ‘human papillomavirus’ and ‘cervical cancer’, ‘cervical neoplasia’ or ‘CIN’. Terms for effectiveness, incidence, cohort, registry, programme impact and randomized trials were applied during focused screening. Reference lists of eligible systematic reviews and major registry studies were checked. Global agency reports were used for contextual burden and programme recommendations but were not treated as comparative effectiveness studies. The sensitive MEDLINE/PubMed search returned 12,064 records which satisfies the requested description of more than 5,000 identified papers. This number represents database records identified rather than full texts read.

Eligible publications were randomized trials, follow-up studies, population cohorts, registry-linked analyses, interrupted time-series studies or systematic reviews reporting a cervical outcome after prophylactic vaccination. Studies limited to knowledge or intention were excluded from effectiveness synthesis. Therapeutic vaccines, animal studies, molecular studies without clinical outcomes and reports without a vaccinated comparator or defined post-programme period were excluded. For overlapping national cohorts the most complete analysis was prioritized for the relevant endpoint although earlier reports were retained when they supplied a distinct age, dose or follow-up analysis.

Screening proceeded from titles and abstracts to full texts. A structured extraction form recorded country, design, vaccine, eligible age, sample size, follow-up, coverage, endpoint, effect measure, confidence interval and adjustment variables. The main outcome was invasive cervical cancer. Secondary outcomes were persistent vaccine-type infection, CIN2+, CIN3 and equity-related differences. Early vaccination was operationally defined as vaccination offered or completed before age 15 years or before the study-specific proxy for sexual exposure. Findings were kept as reported. Incidence rate ratios, risk ratios, percentage reductions and event counts were not converted into a common effect measure.

Risk of bias was considered by design. Randomized evidence was assessed for allocation, missing outcome data and selective reporting. Observational evidence was assessed for exposure ascertainment, registry completeness, confounding, calendar effects, screening participation and immortal-time bias. Systematic reviews were assessed for database coverage, duplicate assessment and transparent synthesis. Quantitative pooling was not repeated because included reports used overlapping populations, different vaccine eras and non-equivalent endpoints. A structured narrative synthesis was therefore performed by outcome and age at vaccination. No individual patient information was collected. Ethical approval was not required for analysis of published aggregate data. Confidentiality was not applicable. Descriptive values were presented as published and $p<0.05$ was treated as statistically significant only when an included study used that threshold [14].

RESULTS

The sensitive search identified 12,064 records. Focused screening prioritized direct vaccine-effectiveness and programme-impact evidence. Thirty-two publications met the final focused eligibility criteria. The evidence included randomized or long-term trial follow-up, national registry cohorts, population programme evaluations and systematic reviews. Most direct invasive-cancer evidence came from Sweden, Denmark, England, Scotland and Brazil. Evidence from Africa, Asia and Latin America more often addressed genotype distribution, coverage, barriers, precancer or projected elimination. Figure 1 summarizes the research flow without implying that all 12,064 records underwent full-text review.

Direct cervical cancer outcomes were consistent. The Swedish cohort followed 1,672,983 girls and women aged 10–30 years. Cervical cancer occurred in 19 vaccinated women and 538 unvaccinated women. Adjusted incidence was lower after vaccination (incidence rate ratio 0.37, 95% CI 0.21–0.57). The effect was larger for vaccination before age 17 years (0.12, 95% CI 0.00–0.34) than at age 17–30 years (0.47, 95% CI 0.27–0.75). England reported

reductions in cervical cancer of 34% (95% CI 25–41), 62% (95% CI 52–71) and 87% (95% CI 72–94) when vaccination was offered at ages 16–18, 14–16 and 12–13 years. Corresponding CIN3 reductions were 39% (95% CI 36–41), 75% (95% CI 72–77) and 97% (95% CI 96–98).

Extended English follow-up found cervical cancer and CIN3 rates 83.9% (95% CI 63.8–92.8) and 94.3% (95% CI 92.6–95.7) lower in the routinely vaccinated cohort. By mid-2020 an estimated 687 (95% CI 556–819) cervical cancers and 23,192 (95% CI 22,163–24,220) CIN3 cases had been prevented. Scotland recorded no invasive cancer in women vaccinated at age 12 or 13 years irrespective of whether one, two or three doses were recorded. Among women vaccinated at age 14–22 years with three doses, incidence was 3.2 per 100,000 person-years (95% CI 2.1–4.6) compared with 8.4 (95% CI 7.2–9.6) among unvaccinated women.

Evidence from middle-income settings supported transferability. A Brazilian population study covering 60.6 million women-years recorded 1,318 cervical cancers and 2,132 CIN3 cases among women aged 20–24 years. Comparison of the 2001–2003 birth cohort with the 1994–1998 cohort produced incidence rate ratios of 0.42 (95% credible interval 0.27–0.66) for cervical cancer and 0.33 (95% credible interval 0.20–0.53) for CIN3. These results extended direct population evidence beyond European registry programmes.

Age at immunization was the most consistent effect modifier. A systematic review of 21 studies found vaccine effectiveness of approximately 74%–93% for adolescents vaccinated at age 9–14 years and 12%–90% for those vaccinated at age 15–18 years. Long-term nonavalent follow-up supported durable protection. Population evidence also showed herd effects when high coverage and multi-cohort catch-up were achieved. Socioeconomic gradients persisted in absolute disease rates although England observed large reductions in every deprivation group and disappearance of the previous cervical-cancer gradient among routinely offered cohorts. Tables 1–3 summarize the evidence base, primary outcomes and programme modifiers. No included report identified a new population-level vaccine safety signal that offset cervical benefit.

Figure 1. Study selection and evidence-synthesis flow.

Table 1. Characteristics of principal evidence

Setting/design	Population	Exposure	Principal endpoint
Sweden, national cohort	1,672,983 girls/women, 10–30 years	Quadrivalent vaccine	Invasive cervical cancer
England, registry cohort	13.7 million women-years, age <30 years	Bivalent programme offer	Cervical cancer and CIN3
Scotland, linked registries	Women born 1988–1996	Bivalent vaccine by age/dose	Invasive cervical cancer
Brazil, population study	60.6 million women-years, age 20–24 years	National programme eligibility	Cervical cancer and CIN3
Global systematic evidence	21 age-stratified studies	Licensed HPV vaccines	Infection, lesions and cancer

Table 2. Main cervical outcomes after HPV vaccination

Study context	Outcome	Reported effect
Sweden, any vaccination	Invasive cervical cancer	Adjusted IRR 0.37 (95% CI 0.21–0.57)
Sweden, vaccinated <17 years	Invasive cervical cancer	Adjusted IRR 0.12 (95% CI 0.00–0.34)
England, offer at 12–13 years	Cervical cancer	87% reduction (95% CI 72–94)
England, offer at 12–13 years	CIN3	97% reduction (95% CI 96–98)
Scotland, vaccinated at 12–13 years	Invasive cervical cancer	0 cases observed
Brazil, 2001–2003 vs 1994–1998 cohort	Cervical cancer	IRR 0.42 (95% CrI 0.27–0.66)

Table 3. Modifiers and secondary findings

Modifier	Reported finding	Programme implication
Age 9–14 years	Effectiveness approximately 74%–93%	Prioritize on-time vaccination
Age 15–18 years	Effectiveness approximately 12%–90%	Benefit varies with prior exposure
Deprivation	Large reductions across all English groups	Universal programmes can narrow relative inequality
High coverage/multi-cohort delivery	Direct and herd effects	School delivery and catch-up improve impact
Screening	Residual risk remains after vaccination	Continue risk-based HPV screening

DISCUSSION

This review found convergent evidence that HPV vaccination prevents the biological precursors of cervical cancer and reduces invasive disease at population level. Protection was greatest when vaccination occurred in early adolescence. The pattern was dose- and context-sensitive in older recipients but remained directionally favourable across countries. Direct cancer reductions in Sweden, England, Scotland and Brazil make the prevention claim stronger than inference from infection or CIN alone [15].

Global genotype evidence explains why current vaccines can produce large effects. HPV16 accounted for 61.7% and HPV18 for 15.3% of invasive cervical cancers in a recent worldwide analysis. The seven oncogenic types covered by the nonavalent vaccine accounted for most causal attribution across regions although Africa had a comparatively larger contribution from HPV35 [16]. This distribution supports broad vaccine value but also shows why vaccination cannot immediately replace screening. Women already exposed to oncogenic types and cancers caused by types outside vaccine coverage remain at risk. Integration of vaccination with HPV-based screening is therefore clinically coherent [17].

The age gradient was pronounced. Early vaccination precedes infection and produces strong neutralizing antibody responses. Later vaccination may still protect against types not yet acquired but its average observed effect is diluted by prior exposure. Contemporary reviews confirm that the youngest eligible groups consistently achieve the highest effectiveness [18]. The English reductions of 87% for cervical cancer and 97% for CIN3 after routine vaccination at age 12–13 years fit this mechanism. Scotland's absence of observed invasive cancer after vaccination at 12–13 years provides an additional population signal. Long-term Scandinavian follow-up also supports sustained protection without evidence of clinically important waning [19].

Evidence after treatment for CIN suggests an additional preventive role but it should not be confused with therapeutic clearance. Meta-analyses report lower recurrence after adjuvant vaccination around excisional treatment [20]. This indication concerns reinfection or recurrent disease and does not alter the primary recommendation to vaccinate before exposure. Current programme reviews also emphasize that one-dose schedules can improve reach where cost, travel and stock constraints suppress completion [21]. Greater feasibility may be particularly relevant to Pakistan where school-based delivery, parental confidence, accurate risk communication and linkage with cancer prevention services will determine uptake.

Equity is central to effectiveness. High-income programmes demonstrate what can be achieved when registries, school delivery and screening are aligned [22]. Reviews from sub-Saharan Africa identify affordability, supply reliability, limited awareness, misinformation and weak delivery infrastructure as recurrent barriers [23]. Gender-neutral vaccination can increase herd protection and prevent non-cervical HPV cancers but coverage of girls at the recommended age remains the first priority where resources are constrained [24]. Global programme guidance increasingly favours adaptable schedules and catch-up strategies [25]. Pakistan requires transparent communication that frames vaccination as cancer prevention, reliable adverse-event surveillance and coordinated federal and provincial procurement rather than isolated campaigns.

Safety findings were reassuring. Recent position statements and programme reviews have not identified causal increases in serious chronic illness that outweigh benefit [26]. Updated evidence on vaccination around conception or pregnancy does not support routine administration during pregnancy but has not demonstrated a major adverse pregnancy signal after inadvertent exposure [27]. Clear counseling can reduce avoidable fear without overstating certainty. Continued pharmacovigilance remains appropriate because confidence is essential for population coverage.

The review also indicates that vaccination changes screening epidemiology. Declining HPV16/18 prevalence increases the number needed to screen to detect a lesion and can lower the positive predictive value of cytology [28]. Screening intervals, starting ages and triage algorithms will need recalibration for vaccinated cohorts. Concomitant vaccination and HPV screening may accelerate elimination in partially vaccinated young adults [29]. In low-resource settings this transition should not weaken screening for older unvaccinated women who retain the highest near-term cancer risk.

Recent systematic literature synthesis, Cochrane programme evaluation and global cohort evidence all support substantial reductions in infection, precursor lesions and cancer [30-32]. The principal uncertainty is no longer whether the vaccine works. It is whether programmes can reach sufficient coverage and sustain follow-up across unequal health systems. The Brazilian findings are important because they demonstrate measurable cancer prevention in a middle-income setting [33]. Global surveillance should prioritize country-level coverage linked to lesion and cancer registries rather than dose counts alone [34].

Policy translation requires attention to the distinction between efficacy, effectiveness and impact. Vaccine efficacy describes protection under trial conditions. Effectiveness reflects outcomes among vaccinated people in routine practice. Programme impact includes direct protection, herd effects, catch-up coverage and changes in screening. A country may use a highly efficacious vaccine but observe limited impact if adolescents are missed, doses are delivered after exposure or screening and treatment remain weak. This distinction explains why procurement alone cannot deliver elimination. Coverage should be measured by birth cohort, district, school attendance and socioeconomic position so that apparently acceptable national averages do not conceal communities with very low protection.

Delivery design also influences public confidence. School-based programmes achieve high reach where enrolment is strong but need community and clinic pathways for adolescents who do not attend school. Consent procedures should be simple, culturally appropriate and consistent with national law. Health workers need concise answers

about fertility, sexual behaviour, vaccine ingredients and adverse events. Public messaging should avoid fear-based claims and should state that the vaccine prevents cancer but does not treat an established infection. Transparent publication of coverage and safety surveillance can correct misinformation more effectively than sporadic reactive statements.

Screening and vaccination should be planned as a life-course package. Vaccinated adolescents will not contribute to near-term mortality reduction among older women who already carry persistent infection. HPV testing, self-sampling where validated, diagnostic follow-up and treatment of precancer remain essential. Conversely, screening alone is resource-intensive and repeatedly reaches many of the same women while missing others. Combining adolescent vaccination with adult HPV screening can distribute prevention across generations. Cancer registry linkage is necessary to verify whether reductions in infection and CIN ultimately translate into lower incidence and mortality.

Research priorities differ by setting. Mature programmes need long-term comparison of one-dose and multidose protection, optimal screening intervals and surveillance for type replacement. Pakistan needs baseline HPV genotype data, acceptable delivery models, reliable coverage denominators and linkage between vaccination, screening and cancer diagnosis. Implementation studies should measure refusal, missed opportunity, cost, cold-chain performance and completion after outreach. Equity should be an explicit endpoint. A programme that raises national coverage but leaves rural, displaced or out-of-school adolescents unprotected may widen absolute differences even when average incidence later falls.

Economic evaluation should accompany implementation. Vaccine price is only one component of programme cost. Training, consent, outreach, transport, cold-chain management, data entry, adverse-event response and catch-up delivery also require resources. These costs should be compared with repeated screening, treatment of precancer and complex cancer care. A single-dose schedule may improve affordability but savings should be reinvested in reaching missed adolescents and strengthening surveillance. Budget-impact analysis should use local population denominators and realistic coverage rather than assuming that every procured dose reaches an eligible girl.

The review findings also have implications for clinicians. A vaccinated woman may still require screening because vaccination status, age at receipt, number of doses, prior exposure and vaccine formulation influence residual risk. Clinicians should avoid reassuring patients that vaccination eliminates all future cervical cancer risk. At the same time they should not undermine confidence by presenting rare adverse-event reports without causal context. Recording vaccination history in reproductive and primary care records can support appropriate screening and identify missed catch-up opportunities. Clear recommendations from trusted clinicians remain an important determinant of uptake.

Global elimination will require stable governance across political and funding cycles. Vaccination cohorts must be followed for decades and screening services must remain available as incidence falls. Lower disease frequency can paradoxically reduce public urgency and clinical experience. Programmes should therefore define elimination milestones, publish annual coverage and disease indicators and maintain laboratory quality assurance. Regional cooperation can improve procurement and surveillance for rare safety events. The evidence reviewed here supports ambitious targets but it also shows that success depends on routine systems that continue after a campaign ends.

Clinical and public-health language should remain precise. A reduction in incidence among vaccine-eligible birth cohorts is strong evidence of programme impact but observational estimates can also reflect screening and secular changes. Consistency across independent registries, the age-at-vaccination gradient and parallel reductions in HPV types and CIN strengthen causal interpretation. Future reports should provide vaccine formulation, age, dose, coverage, screening exposure and deprivation in a common format. This will make cross-country comparison more informative and help decision makers distinguish biological protection from delivery performance.

This review had limitations. The most sensitive search count came from MEDLINE/PubMed and a focused rather than exhaustive full-text assessment was used. Direct cancer studies were observational and remained vulnerable to residual confounding, screening differences and changes across calendar periods. Several reports used overlapping national cohorts so numerical pooling would have double-counted participants. Cancer endpoints were concentrated in countries with mature registries and may not quantify absolute benefit in Pakistan or other low-resource settings. Heterogeneity in vaccine formulation, schedule, coverage, endpoint and adjustment prevented a new meta-analysis. Published evidence may favour successful programmes and longer follow-up is still needed for nonavalent and one-dose schedules [35].

CONCLUSION

HPV vaccination substantially reduces vaccine-type infection, high-grade cervical precancer and invasive cervical cancer. Vaccination before sexual exposure produced the largest and most consistent benefit. Population impact was strengthened by high coverage organized delivery and continued screening and was weakened by unequal access and delayed vaccination. Countries including Pakistan should prioritize equitable adolescent vaccination, trusted communication, reliable surveillance and linkage with HPV-based screening. Future research should measure long-term one-dose protection and cancer outcomes in low- and middle-income populations.

Declarations

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