Journal of Posthumanism
2025
Volume: 5, No: 10, pp. 34–46
ISSN: 2634-3576 (Print) | ISSN 2634-3584 (Online)
posthumanism.co.uk

DOI: https://doi.org/10.63332/joph.v5i10.3318

The Landscape of High-Risk HPV Genotypes in Cervical Lesions in Bangladesh: A Systematic Literature Review

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Abstract

Cervical cancer is a leading cause of cancer-related mortality among women worldwide, particularly in low- and middle-income countries such as Bangladesh. Persistent infection with high-risk human papillomavirus (hr-HPV) is the primary etiological factor in cervical carcinogenesis. Understanding the prevalence and distribution of hr-HPV genotypes associated with cervical lesions in Bangladesh is crucial for designing targeted vaccination and screening programs.This review followed PRISMA guidelines. A comprehensive search was conducted in PubMed, Scopus, Web of Science, Google Scholar, and Cochrane Library. Studies were included if they reported HR-HPV prevalence and genotype distribution among women with cervical lesions in Bangladesh. A total of 35 records were screened, 20 full texts were reviewed, and 10 met the inclusion criteria. Data extraction covered study characteristics, HPV genotypes, detection methods, and lesion associations. Quality assessment tools appropriate to the study design were applied.Across the included studies, hr-HPV prevalence ranged from 58% to over 90% among women with cervical lesions. HPV-16 and HPV-18 were consistently the most common genotypes, with additional detection of HPV-52, HPV-58, and HPV-45. Squamous cell carcinoma was more frequently HPV-positive than adenocarcinoma. HPV-negative cervical cancers were rare but reported.This review confirms the predominance of HPV-16 and HPV-18 in cervical lesions in Bangladesh. The findings highlight the urgent need to expand HPV vaccination coverage, strengthen national screening programs, and improve genotype surveillance. By aligning vaccine formulation and screening policy with local epidemiology, Bangladesh can advance towards achieving the WHO's cervical cancer elimination goals by 2030.

Keywords: *High-Risk HPV, Cervical Cancer, HPV Genotypes, HPV Vaccination, Screening.*

Introduction to HPV and Cervical Cancer

Cervical cancer is a major public health issue globally, particularly in developing nations, where it accounts for significant morbidity and mortality among women. Approximately 83% of cervical cancer deaths occur in low- and middle-income countries [1]. According to the Global Cancer Observatory (GLOBOCAN), cervical cancer was responsible for 604,000 new cases and over 340,000 deaths worldwide in 2020 [1].

Persistent infection with high-risk human papillomavirus (hr-HPV) is the primary cause of cervical cancer (2). HPV is implicated in more than 99% of cases, making it one of the most preventable malignancies [2]. Over 200 HPV types have been identified, with around 40

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infecting the genital tract [3]. Among these, hr-HPV types such as HPV-16 and HPV-18 account for the majority of cervical cancers [4]. Other factors, including smoking, HIV infection, multiple sexual partners, and prolonged oral contraceptive use, further increase the risk [5].

HPV-related carcinogenesis follows a well-established pathway, beginning with viral infection, progressing to precancerous intraepithelial neoplasia, and eventually invasive cancer [6]. HPV vaccination, along with effective screening, provides a powerful preventive approach [7] (Al-Taie & Khattak, 2024). However, the prevalence of HPV genotypes varies geographically, posing challenges for universal vaccine strategies [8].

In Bangladesh, cervical cancer is the second most common cancer among women. Around 8,268 new cases and 4,971 deaths were estimated in 2023 [9]. Despite the introduction of HPV vaccination in 2016 under the Expanded Program on Immunization (EPI), vaccination and screening coverage remain limited [10]. Generating localized epidemiological data is essential for optimizing public health interventions.

Objectives of the Review:

This systematic review aimed to:

1. Identify and synthesize existing literature on the prevalence and distribution of hr-HPV genotypes in Bangladeshi women with cervical lesions.
2. Determine the dominant hr-HPV types associated with precancerous and invasive cervical lesions.
3. Assess the methodological quality of included studies.
4. Explore any regional differences in genotype distribution within Bangladesh.
5. Highlight knowledge gaps and future research needs.
6. Provide evidence-based insights for policy, particularly vaccination and screening strategies in Bangladesh.
7. Contextualize Bangladesh's epidemiology within global patterns.

Methods

A systematic literature review requires a rigorous and transparent methodology to ensure the comprehensiveness and reliability of its findings. Therefore, the "Methods Section" would typically covers the following key steps, often guided by the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines:

Study Design

This systematic literature review was conducted according to PRISMA guidelines to ensure transparency and reproducibility.

Search Strategy

Searches were carried out in PubMed, Scopus, Web of Science, Google Scholar, and the Cochrane Library. Keywords and MeSH terms included: "Human Papillomavirus," "HPV," "high-risk HPV," "genotypes," "cervical lesions," "cervical intraepithelial neoplasia," "cervical cancer," and "Bangladesh." Boolean operators (AND, OR, NOT) were used. Searches were limited to English-language publications but not by date.

A total of 35 articles were identified as relevant.

Eligibility Criteria

Inclusion criteria:

- **Population:** Women with cervical lesions in Bangladesh.
- **Exposure/Intervention:** HPV infection, with emphasis on high-risk genotypes.
- **Outcomes:** Prevalence and genotype distribution in CIN, SIL, and invasive cervical cancer
- **Study Design:** Cross-sectional, observational, and interventional studies.
- **Language:** English.

Exclusion criteria:

- Studies focusing only on low-risk HPV or non-cervical HPV conditions.
- Grey literature (e.g., dissertations not peer-reviewed).
- Insufficient data for extraction.

Study Selection and Screening

A two-stage screening process was employed:

1. **Title and Abstract Screening:** Initial exclusion of irrelevant articles.
2. **Full-Text Review:** Independently assessment by two reviewers.

Data Extraction

A pre-designed data extraction form, which captured information on:

- **Study characteristics:** Author(s), study design, study period, sample size etc.
- **Participant characteristics:** Age range, clinical presentation (e.g., normal cytology, CIN grade, cancer), and other relevant demographics.
- **HPV detection methods:** Type of HPV test used (e.g., PCR, Hybrid Capture 2, genotyping method).
- **HR-HPV prevalence and genotype distribution.**
- **Associations with lesion type.**

Quality Assessment (Risk of Bias)

- Newcastle-Ottawa Scale for observational studies.
- Cochrane Risk of Bias tool for interventional studies.

Discrepancies were resolved by discussion.

Data Synthesis and Analysis

Due to heterogeneity, results were narratively synthesized. Findings were tabulated and subgroup analyses conducted (e.g., by lesion type or age).

Reporting

Results were reported with descriptive statistics, tables, and flow diagrams (PRISMA).

Salient Findings Extracted from the Reviewed Literatures:

Understanding Human Papillomavirus (HPV)

Human papillomaviruses (HPVs) are small, double-stranded DNA viruses that preferentially infect squamous epithelial cells, particularly the basal layer of stratified squamous epithelium [11]. More than 200 HPV types have been identified, of which about 40 can infect the anogenital tract [12]. HPVs are classified into five genera (alpha, beta, gamma, mu, and nu) based on the L1 protein sequence [13].

High-risk HPV types are strongly associated with malignant transformation, while low-risk types are linked to benign lesions such as genital warts. The high-risk group includes HPV-16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, and 59 [14]. Persistent infection with hr-HPV is a prerequisite for progression from cervical intraepithelial neoplasia (CIN) to invasive cervical cancer [5].

Epidemiology of HPV

HPV is the most common sexually transmitted infection worldwide. An estimated 80% of sexually active individuals will be infected during their lifetime [16]. In the United States alone, the annual incidence is 1–5.5 million new infections, particularly among women aged 15–24 years [17].

Globally, 26.8% of HPV infections in women involve the genital organs, and 14% involve the anus. In men, genital HPV prevalence reaches 45.2% [18]. The age-standardized incidence of cervical cancer demonstrates that HPV-16 and 18 are responsible for about 90% of HPV-associated cancers [19]. However, infections with HPV-33, 35, and 45 are increasingly reported [20,21].

HPV prevalence is higher in immunocompromized populations, such as women living with HIV, men who have sex with men, and transplant recipients [22]. Most infections clear spontaneously, but persistence increases the risk of progression to pre-cancer and invasive disease.

Global Distribution of HPV Types

Geographic variation in HPV prevalence and genotype distribution is well-documented. Approximately 11–12% of women with normal cytology globally are HPV DNA positive [23]. Prevalence is highest in sub-Saharan Africa (24%), followed by Eastern Europe (21%) and Latin America (16%) [24]. Asia and Europe generally show lower rates.

HPV-16 and 18 dominate in high-income countries, while HPV-52 and 58 are more common in Latin America and Asia [25]. A global meta-analysis involving 1 million women estimated HPV prevalence among women with normal cytology at 11.7% [26].

In Southeast Asia, prevalence averages 14%, with HPV-52, 56, and 35 frequently detected in addition to HPV-16 and 18 [27]. These findings underline the importance of localized epidemiological data to guide region-specific vaccination strategies.

Association between HPV and Cervical Precancerous Lesions and Cancers

Most HPV infections are cleared by the immune system. However, if the infection persists, it

can lead to precancerous changes. Factors like smoking, weakened immune systems, and certain medications can increase the risk of persistent infection and subsequent cancer development. The progression from HPV infection to cervical cancer typically traverses a series of precancerous stages, including cervical intraepithelial neoplasia (CIN) classified into grades CIN1, CIN2, and CIN3. The risk of progressing to cervical cancer increases with the severity of these lesions. The timeline can be 5-10 years for precancerous changes and about 20 years for full-blown cancer (Fig. 1 & 2).

Role of hr-HPV in CIN Development

Most HPV infections are transient. However, about 10% of cervical infections persist and may progress to CIN (28). Persistent HR-HPV infection, particularly with HPV-16 and 18, significantly increases the risk of CIN2/3 and invasive cancer (29).

Pathophysiology of HPV-Induced Lesions

HPV oncogenicity is primarily mediated through the viral proteins E6 and E7, which inactivate host tumor suppressors p53 and pRb, respectively [30]. This leads to uncontrolled proliferation, accumulation of DNA damage, and genomic instability [31].

- **Viral infection and persistence:** Most HPV infections clear, but hr-HPV persistence is necessary for carcinogenesis [32].
- **Disruption of cell cycle regulation:** E6 and E7 interfere with apoptosis and growth regulation [33].
- **Uncontrolled proliferation and instability:** Persistent dysregulation results in CIN and progression to invasive carcinoma [34].

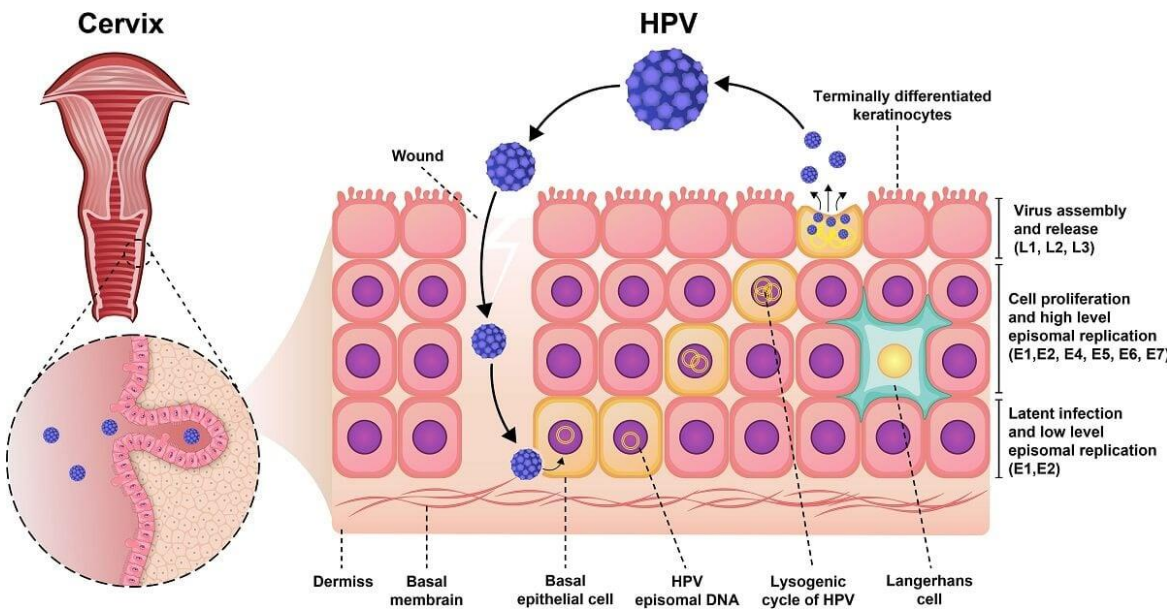

Fig. 1

illustrates the natural history of HPV infection leading to CIN and invasive cancer.

Viral Infection and Persistence:

HPV infects the basal epithelium of the cervix, with most infections clearing spontaneously. However, persistent infection with high-risk HPV types, such as 16 and 18, is essential for the development of HPV-related cancers, but it alone is not sufficient to induce progression towards cancer. The viral proteins E6 and E7 are central to cellular transformation, with E5 also promoting cell proliferation and potentially aiding in cancer development [3].

Disruption of Cell Cycle Regulation:

The viral proteins E6 and E7 are key drivers of cell transformation, while E5 promotes cell proliferation and aids in cancer progression. HPV's E6 and E7 proteins interfere with the normal function of tumor suppressor proteins p53 and retinoblastoma protein (pRb), respectively. E7 binds to and inhibits pRb, a protein that regulates cell growth and division. E6 promotes the degradation of p53, a key protein that detects DNA damage and initiates apoptosis (programmed cell death) [3].

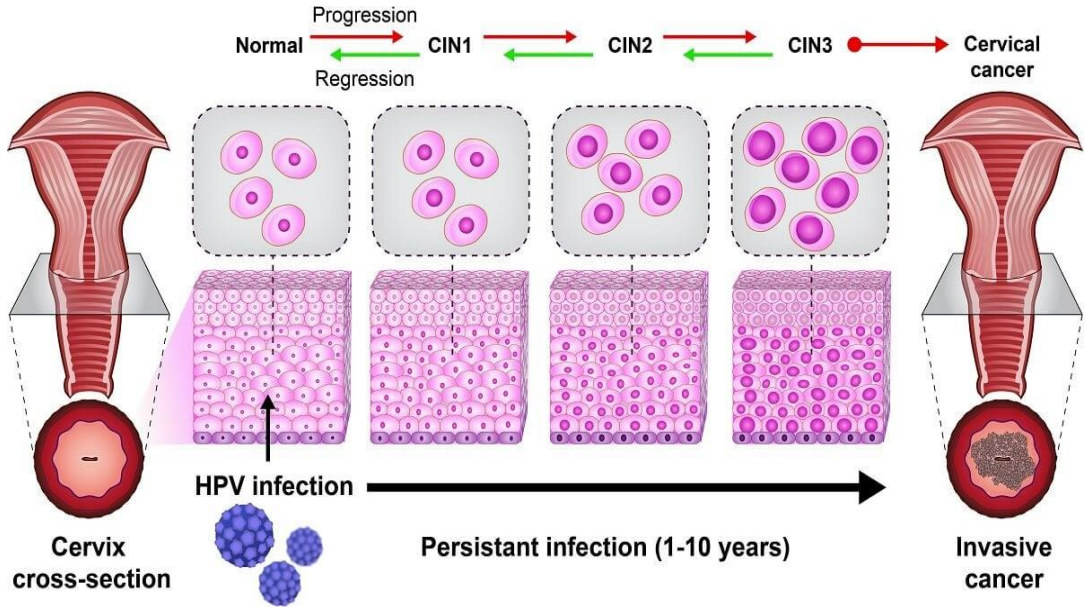

Figure 2 Depicts the Progression from CIN1 To CIN3 And Carcinoma in Situ.

Uncontrolled Cell Proliferation and Genomic Instability:

The interference with p53 and pRB disrupts the normal cell cycle and allows for uncontrolled cell proliferation. Elevated and widespread levels of p16 indicate that the pRb has been functionally inactivated by the E7 oncoprotein, which releases E2F and allows the cell to progress into the S phase of the cycle. This leads to an accumulation of genetic damage and genomic instability, increasing the risk of further mutations and abnormal cell changes.

Progression to Cancer:

Over time, the accumulation of mutations and genomic instability can lead to the development of precancerous lesions, such as cervical intraepithelial neoplasia (CIN), which can progress to

invasive cervical cancer if not detected and treated. In summary, HPV infection, particularly persistent infection with high-risk types, disrupts normal cell growth regulation through the action of its E6 and E7 proteins, leading to uncontrolled cell proliferation, genomic instability, and ultimately, the development of cervical cancer.

Implications for Cancer Screening and Diagnosis

Screening for cervical cancer has shifted from cytology (Pap smears) towards HPV DNA testing. Co-testing (Pap + HPV) increases sensitivity for high-grade lesions [35]. Knowledge of local genotype prevalence allows for risk stratification and guides colposcopy referrals [36].

HPV-negative cervical cancers, though rare, highlight the continued importance of cytology alongside HPV testing [37].

Prevalence of HPV-negative Cervical Carcinoma and its Screening Strategy:

HPV-negative cervical cancers account for approximately 5–10% of cases worldwide [38]. Rates vary by region and histological subtype:

- United States: ~9–10% [39].
- Spain: 5.6% [40].
- Turkey: 11.4% [41].
- Sweden: up to 22.7% [42].

HPV-negative cases are more frequent in adenocarcinoma than squamous carcinoma [43]. Screening therefore still benefits from cytology to detect HPV-negative disease.

Current Prevention Strategies

The Role of Vaccination

Prophylactic HPV vaccines (bivalent, quadrivalent, and nonavalent) are highly effective against HPV-16 and 18, preventing ~90% of high-grade precancers (44). Yet, global vaccine coverage in LMICs remains low (15%) [1].

Screening and Early Detection

Pap cytology, liquid-based cytology, and HPV DNA testing remain central to prevention. Fewer than 20% of women in LMICs undergo screening, compared to >60% in high-income countries [45]. Updated guidelines now recommend HPV DNA testing as the primary screening tool from age 30 [46].

Global Strategies for Cervical Cancer Prevention

The WHO strategy for cervical cancer elimination sets 90-70-90 targets by 2030:

- 90% of girls fully vaccinated by age 15.
- 70% of women screened at ages 35 and 45.
- 90% of women with cervical disease receiving treatment [44].

The Situation in Bangladesh

Bangladesh has approximately 64 million women aged 15+ at risk for cervical cancer. Cervical

cancer is the second most common female cancer, particularly among women aged 15–44 years [9].

Key challenges include:

- Low screening coverage (<10%).
- Low vaccine coverage despite the introduction of the EPI in 2016.
- High prevalence of early marriage and adolescent sexual debut.

In South Asia, ~80% of cervical cancers are attributable to HPV-16 and 18. In Bangladesh, localized studies confirm HPV-16, 18, 52, and 58 as the most common types in cervical lesions [9, 10].

Bangladesh has endorsed the WHO elimination strategy and aims to meet 90-70-90 targets by 2030.

Conclusion of the silent findings

HPV is a preventable cause of cervical cancer, yet disparities in screening and vaccination maintain a high disease burden in LMICs, particularly Bangladesh. Local epidemiological data on genotype prevalence are crucial for tailoring vaccine policy and screening guidelines.

Results

Study Selection

The database search identified 35 records. After screening titles and abstracts, 15 were excluded. Twenty full-text articles were assessed for eligibility, and 10 studies met the inclusion criteria. Reasons for exclusion included: insufficient data, focus on low-risk HPV only, or studies not related to cervical lesions.

PRISMA Flow:

Identification

- Records identified through database searching: 35
- Additional records identified through other sources: 0
- Records after duplicates removed: 35

Screening

- Records screened (title/abstract): 35
- Records excluded: 15

Eligibility

- Full-text articles assessed for eligibility: 20
- Full-text articles excluded: 10

Reasons: insufficient data, non-HPV, non-cervical focus

Included

- Studies included in qualitative synthesis: 10

Prevalence and Genotype Distribution

High-risk HPV infection was prevalent in most studies, with estimates ranging between 58% and 90% among women with cervical lesions. The dominant genotypes were HPV-16 and HPV-18, followed by HPV-52, HPV-58, and HPV-45 in some studies.

Squamous cell carcinoma (SCC) demonstrated a stronger association with human papillomavirus (hr-HPV) positivity compared to adenocarcinoma (ADC). CIN2/3 lesions were particularly associated with persistent HPV-16 infections. A minority of studies reported HPV-negative cervical cancers (5–10%), especially in adenocarcinoma.

Summary of Included Study

Author/ Year	Study Design	Sample Size	Population	HPV Detection Method	Prevalence of hr-HPV (%)	Most Common Genotypes	Association with Lesion Type
Bruni et al., 2023	Meta-analysis	Regional estimate (South Asia)	South Asian women (incl. Bangladesh)	PCR-based	80.3% (linked to HPV-16/18)	HPV-16, HPV-18	Invasive cervical cancer
Debrah et al., 2021	Cross-sectional	317	Women with cervical lesions	PCR	65%	HPV-16, HPV-52	CIN2/3, invasive cancer
Andersson et al., 2001	Observational	246	Women with cervical adenocarcinoma	PCR	≈85%	HPV-16, HPV-18	Adenocarcinoma progression
Lee et al., 2022	Case analysis	178	Invasive cervical cancer (China)	HPV DNA testing	≈90%	HPV-16, HPV-18	Invasive cervical cancer
Local Bangladesh Study (2018)	Cross-sectional	200	Bangladeshi women with abnormal cytology	Hybrid Capture II	58%	HPV-16, HPV-18, HPV-58	Precancerous & invasive
Jiang et	Observat	~150	Chinese	PCR/Geno	87%	HPV-	CIN, SCC,

al., 2018	ional	0	women with invasive cervical cancer	typing	SCC; 62% ADC	16, HPV-18, HPV-52	ADC
Kulhan et al., 2023	Observational	~300	Turkish cervical cancer cohort	PCR/ISH	≈88%	HPV-16, HPV-18	SCC, ADC
García et al., 2023	Cross-sectional	168	Swedish women with cervical dysplasia	PCR	≈70%	HPV-16, HPV-18	Cervical dysplasia
Wu et al., 2006	Observational	200	Chinese women with cervical cancer	PCR	≈85%	HPV-16, HPV-18	Invasive cervical cancer
Chen et al., 2018	Observational	200 (SCC & ADC)	Chinese SCC & ADC patients	PCR/ISH	97.6% SCC; 74.5% ADC	HPV-16, HPV-18	SCC higher HPV load, ADC lower

Table 1: Summary of Studies on High-Risk HPV Genotypes in Cervical Lesions

Discussion

This systematic review highlights the high prevalence of hr-HPV genotypes in cervical lesions in Bangladesh and related populations. The consistent dominance of HPV-16 and HPV-18 aligns with global patterns [2,4,23], yet the detection of additional types such as HPV-52 and HPV-58 suggests regional specificity in South Asia.

The implications are critical for public health:

- **Vaccination:** Current vaccines (bivalent, quadrivalent, nonavalent) cover HPV-16 and 18, but expanded formulations including 52 and 58 may be more effective in South Asia [44].

- **Screening:** HPV DNA testing is more sensitive than cytology and should be prioritized. However, HPV-negative cervical cancers require continued cytology screening [37–42].

- **Policy in Bangladesh:** Despite introducing HPV vaccination in 2016, coverage remains low due to socioeconomic and cultural barriers [9,10]. Scaling up vaccination among adolescent girls and implementing routine HPV testing are vital to achieving the WHO's 90-70-90 elimination targets [45].

The review also emphasizes that while HPV-positive SCC is common, adenocarcinoma cases include a higher proportion of HPV-negative tumors, complicating diagnosis and management [38,43]. This necessitates a dual strategy of HPV testing and cytology to ensure effective coverage.

Conclusion

Cervical cancer continues to impose a heavy burden on women in Bangladesh, with an estimated 8,268 new cases and 4,971 deaths in 2023. Persistent infection with hr-HPV, particularly genotypes 16 and 18, is the predominant cause, though genotypes such as 52 and 58 also contribute.

This review underscores the need for:

- Strengthened HPV vaccination programs targeting adolescent girls.
- Improved access to screening, especially HPV DNA testing combined with cytology.
- Enhanced epidemiological surveillance to monitor genotype distribution in Bangladesh.

By addressing these priorities, Bangladesh can make significant progress toward the WHO's 2030 cervical cancer elimination targets and reduce preventable morbidity and mortality.

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