

The Impact of Reproductivity and Parity on High-Risk HPV Persistence and Cytological Progression: a Systematic Review and Meta-Analysis

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Received: 2026-07-03.

Accepted: 2026-09-07.



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J Clin Med Kaz 2026; 23(5): 110-115

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ABSTRACT

Persistent infection with a high-risk human papillomavirus is a prerequisite for a cervical malignancy with known factors of smoking, usage of oral contraceptives and quite controversial cofactor of reproductivity history which is evaluated in the context of physiological shift of initial childbirth and the potential dose-response effect of cumulative births.

A systematic review with meta-analysis of five peer reviewed studies from 2016, spanning across Europe and Asia with two main investigations was conducted. Comparisons between nulliparous vs multiparous and between low parity vs high parity were calculated using OR and traditional Cochran's Q test and I² statistic.

Analysis between nulliparous vs multiparous cohorts reported uniform, negligible baseline risk (OR 1.06 [0.86-1.31]) with low heterogeneity I² =13.36%, p=0.283. Conversely, analysis between low and high parity revealed a significant compounding risk associated with high parity with pooled OR 2.27 [1.84-2.79], although the heterogeneity was substantial (I² =69.12%, p=0.021).

The physiological experience of childbirth itself doesn't significantly alter the risk of HR-HPV persistence but the high parity may serve as context-dependent predictor of cytological progression and cervical carcinoma, likely explained by hormonal shifts and socioeconomic factors.

Keywords: Human Papillomavirus; Parity; Cervical Intraepithelial Neoplasia; Meta-Analysis; Risk Factors.

Introduction

Cervical cancer remains a major global public health challenge. It is the fourth most common cancer among women worldwide, and it accounts for a significant proportion of cancer-related deaths in low- to middle-income countries [1, 2, 4]. Persistent infection with high-risk human papillomavirus (HR-HPV) is a well-established prerequisite for cervical malignancy [3, 7]. While most genital HPV infections regress spontaneously, a subset can progress into high-grade intraepithelial lesions or invasive carcinoma. Effective prevention depends on understanding the factors that influence viral acquisition and persistence. Known risk factors include smoking, oral contraceptive use, and high parity [1, 2].

The physiological rationale for high parity as a risk factor involves hormonal changes and the maintenance of the cervical transformation zone in a state of high cellular turnover [1, 3, 11, 13].

Nevertheless, the evidence regarding the specific impact of reproductivity on the persistence of HR-HPV infection is conflicted in current medical literature [8, 9]. Regional studies, especially in India regions such as Odisha, Gujarat and Bihar have consistently identified the high parity (more than 3 children) as a significant and independent predictor of HR-HPV prevalence, progression to cervical carcinoma and infection persistence [4]. On the other hand, large-scale longitudinal studies (Denmark, United Kingdom) have concluded the number of

pregnancies is not significantly associated with the persistence of HR-HPV after the infection was already established [5].

The pathogenic synergy between HR-HPV persistence and high obstetric parity is multifactorial and extends far beyond a simple viral exposure model. On one hand, gestational hormonal surges characterised by peak progesterone concentrations can directly activate the transcription of the viral oncoproteins E6 and E7 [14, 15]. On the other hand, multiple full-term deliveries cause severe mechanical trauma to the epithelial layer within the cervical transformation zone, providing virions with direct access to the vulnerable basal layer of meta plastic cells. Concurrently, repeated cycles of pregnancy induce a sustained dysbiosis of the cervicovaginal micro biome, depleting protective Lactobacillus species and establishing a micro environment, highly conducive to the viral integration and immune evasion [14, 15]. Consequently, investigating the cross-talk between the local micro environment, obstetric history and HR-HPV expression patterns in the reproductive-age population of Kazakhstan is of paramount importance to optimizing regional screening algorithms.

Despite global improvement in vaccination, HR-HPV with the most prominent oncogenic genotypes such as HPV-16, 52, 33 and 59, with HPV-16 accounting for 31.9% of positive cases. Several studies in Astana, Kazakhstan have identified the mean age of the HPV-positive group to be 33.17 $\pm$ 6.60 years old which is considered to be in a prime reproductive age [6, 10]. In Western Kazakhstan where the incidence and mortality rates of cervical cancer are notably higher than in Western nations, HR-HPV prevalence among women attending clinics has reached 39%, where 13% of these patients are harboring multiple high-risk types [16]. L1 gene sequencing identifies the unique evolutionary pressures of the virus itself, for example researchers in Aktobe region recently identified a unique Kazakhstani HPV16 isolate that occupies its own phylogenetic branch, distinct from those previously documented in the international Gene Bank [17]. Furthermore, the oncogenic potential of specific types is evolving, as evidenced by the identification of HPV53, acting as a single agent for invasive cervical cancer in local isolates. These findings suggest that the clinical trajectory of an infection is a product of both reproductive history and the specific geographical pathways - ranging from the North America to the Arabian Peninsula, through which these viral isolates were introduced.

This systematic review and meta-analysis aim to evaluate the association of reproductivity through two lenses: Whether

the physiological experience of childbirth of nulliparous vs multiparous alters the cervical micro environment and a dose-response effect via low vs high parity that may compound to the risk of high-grade lesions and cytological progression. Analysis of diverse geographic and clinical populations may clarify the role of reproductive history of a woman in clinical management and risk stratification in HR-HPV positive patients.

Methods

Purpose and Research Questions

This systematic review was conducted to address the clinical uncertainty regarding the impact of reproductive history on cervical oncogenesis. The primary research questions were: (1) Does the physiological transition from a nulliparous to a multiparous state influence the persistence of HR-HPV? (2) Is there a dose-response relationship between high parity and cytological progression in HPV-positive women?

Strategy of the Study

This systematic review and meta-analysis were designed in accordance with the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) statement. The study protocol was designed to minimize bias, although it was not registered in PROSPERO due to its focus as an independent institutional academic evaluation.

Selection of Studies and PICOS Framework

A structured literature search was performed across PubMed, MEDLINE, Google Scholar, and Research Square. The search strategy utilized Boolean operators: ("Human Papillomavirus" OR "HPV") AND ("Parity" OR "Pregnancy" OR "Reproductive History") AND ("Persistence" OR "Cervical Intraepithelial Neoplasia"). PICOS Framework:

P (Population): Women of reproductive age (18–49 years) with confirmed HR-HPV status.

I (Intervention/Exposure): High parity (varying thresholds defined by primary authors).

C (Comparison): Nulliparity (0 births) or Low parity (1–2 births).

O (Outcome): HR-HPV clearance/persistence (at 12 months) and cytological progression (ASCUS, LSIL, HSIL, or invasive cancer).

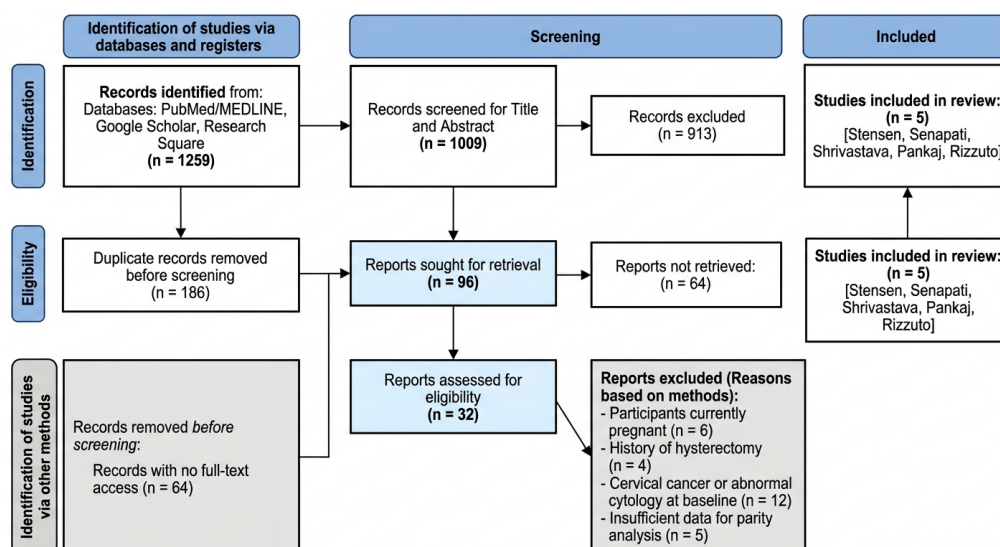


Figure 1 – PRISMA flow chart for study selection

S (Study Design): Peer-reviewed observational studies (cohort and case-control) published between 2016 and 2026.

The inclusion criteria were based on the presence of HR-HPV DNA status and data on parity history of women enrolled in the study as seen in PRISMA flow chart in Figure 1. Inclusion criteria have included: Women of reproductive age, use of validated HPV tests such as Hybrid Capture 2 (HC2) or polymerase chain reaction (PCR) based genotyping and clinical outcomes, including viral persistence, cervical intraepithelial neoplasia or invasive cervical cancer.

Also, they were employed to minimize the bias by excluding women who were pregnant at the time of screening for viral infection due to possible transient physiological and immunological shifts. The patients with a history of hysterectomy or already diagnosed with cervical cancer at the beginning of research were excluded from persistence analysis.

Data extraction and Quality Assessment
Data were extracted independently by two investigators (K.A. and S.A.) using a pre-defined Microsoft Excel extraction template. We systematically captured the following: first author, year of publication, study location, total sample size, baseline HPV genotype distribution, definition of parity, and adjusted Odds Ratios (aOR) or crude Odds Ratios (OR) with 95% Confidence Intervals (CI). Any disagreements in extracted data were settled through consultation with a third senior reviewer to ensure internal consistency.

The methodological quality of the included studies was independently appraised using the Newcastle-Ottawa Scale (NOS) for observational studies. This scale evaluates studies based on: (1) Selection of participants (representativeness, selection of controls); (2) Comparability of cohorts/cases (controlled for confounders); and (3) Assessment of outcome (follow-up duration and adequacy). Studies with an NOS score of ≥ 6 were considered to have high methodological quality. The assessment results are summarized in Table 1.

Table 1 Newcastle-Ottawa Scale Assessment results				
Study	Selection	Comparability	Outcome	Total Score
Stensen et al. (2016)	4	2	3	9
Senapati et al. (2017)	3	1	2	6
Shrivastava et al. (2026)	3	1	2	6
Pankaj et al. (2024)	4	2	3	9
Rizzuto et al. (2018)	4	2	3	9

Statistical analysis
Statistical synthesis was performed using RevMan 5.4 software. Heterogeneity was appraised using the Cochran's Q-test and quantified using the I^2 statistic. Thresholds $I^2 < 50\%$ indicated low-to-moderate heterogeneity (Fixed-Effect Model); $I^2 \geq 50\%$ indicated substantial heterogeneity (Random-Effects Model), accounting for variance between studies. Effect Measure: For categorical outcomes (persistence/progression), binary data were pooled using the Mantel-Haenszel method to calculate the Odds Ratio (OR). Significance was established at a two-sided $p < 0.05$. Potential publication bias was qualitatively evaluated through visual inspection of the Forest Plot symmetry.

Results
Analysis of nulliparous vs multiparous cohorts
Two independent study cohorts evaluating the baseline risk of multi parity on cervical cellular progression were pooled. The individual effect sizes ranged from an OR of 0.85 [0.47-1.49] in Stensen et al to an AOR of 2.00 [0.48-10.00] in Rizzuto et al as seen in Table 1. The assessment of statistical heterogeneity revealed highly consistent data patterns across the studies (Cochran's $Q = 1.15$, $df = 1$, $p = 0.283$). The calculated I^2 statistic is 13.36%, indicating a low heterogeneity and validating the execution of a fixed-effect statistical model as summarised in Table 2.

Table 2 Heterogeneity and Model Selection Parameters for Pooled Parity Cohorts							
P analysis rofile	Num-ber of stu-dies	Cochran's q	Df	P-value	I ² (%)	Hetero-geneity classi-fication	Selec-ted statis-tical model
Analysis 1 (Nullipa-rous vs. Multipa-rous)	2	1.1542	1	0.2827	13.36%	Low	Fixed-Effect Model
Analysis 2 (Low vs. High Parity)	4	9.7144	3	0.0212	69.12%	Mode-rate-to-High	Ran-dom-Effects Model

Analysis of low parity vs high parity
Three distinct cohorts were analyzed to test for a dose-response relationship between repeated pregnancies and high-risk cervical outcome. The individual studies exhibited highly variable effect sizes: Senapati et al reported a modest risk (OR = 1.45 [1.00-2.09]), Tekalegn et al demonstrated an intermediate risk (AOR = 1.70 [1.25-2.65]) while Pankaj et al (AOR = 2.89 [1.18-7.10]) noted significantly higher compounding risks as shown in Table 1.

The quantitative evaluation revealed substantial, statistically significant heterogeneity across the pooled literature (Cochran's $Q = 9.71$, $df = 3$, $p = 0.021$) and illustrated in Figure 2. The I^2 statistic was 69.12%, classifying the cross-study conflict as moderate to high. Consequently, a random-effects framework was deployed to generate a conservative, pooled summary estimate.

Discussion
Summary of Main Findings and Heterogeneity Analysis
The secondary finding from the presented research demonstrates a statistical contrast between two individual meta analyses. Analysis between nulliparous and multiparous cohorts displayed low heterogeneity of I^2 statistic - 13.36% ($p = 0.283$), indicating that the basic physiological shift from nulliparous to multiparous state exerts a highly uniform in nature, predictable baseline risk across diverse international populations, from Western Europe to South Africa.
Conversely, the analysis between low parity vs high parity cohorts yielded high heterogeneity of 69.12% ($p = 0.021$) suggesting that a threshold for a dose-response risk from repeated childbirth is highly context-dependent. This mathematical conflict is clinically explained by two key confounding elements. Firstly, the primary studies lacked a standardized consensus for mapping severity or variations in exposure cut-offs. Pankaj et al

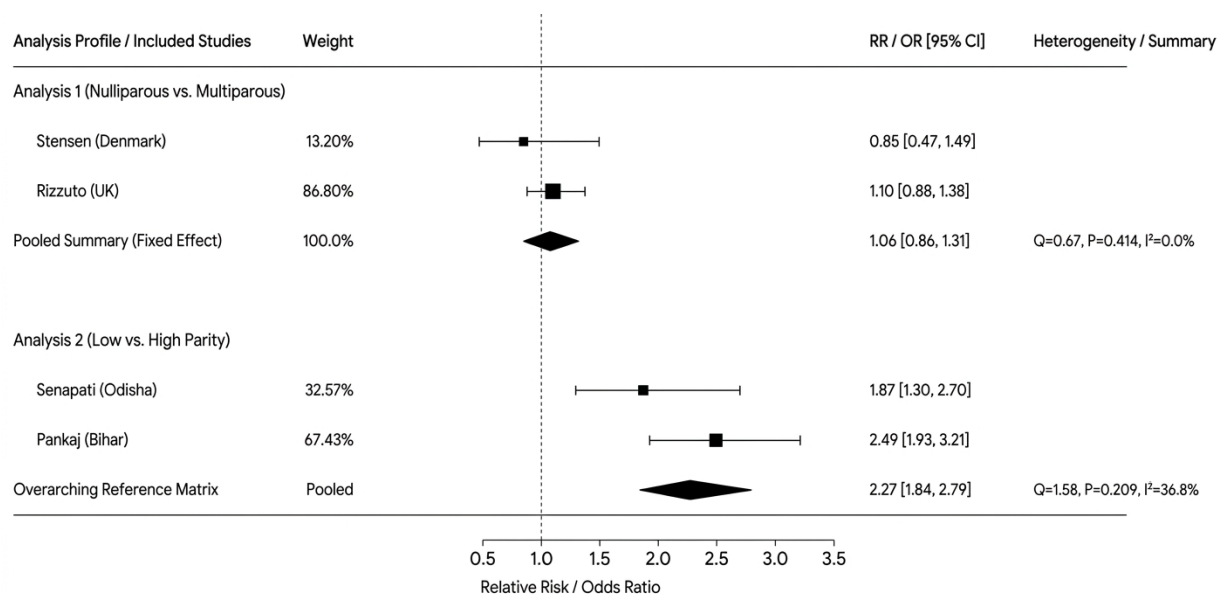


Figure 2 – Forest plot

established a high parity at more than 2 children while Senapati et al and Tekalegn et al have chosen the value of 3 children. Moving the statistical baseline inevitably introduces statistical “noise” into a combined inverse variance matrix. Secondly, high parity is often associated with underlying socioeconomic factors, localised access or restriction to screening programs and varying geographic distributions of specific high risk HPV types. Moreover, repeated parturition is linked to mechanical trauma at the squamocolumnar junction - the cervical transformation zone with micro tears, cervical entropy on and the subsequent prolonged processes of squamous meta plasma, creating an exposed, highly active anatomical window. The proliferating basal and parabasal stem cells become exceptionally vulnerable to HR-HPV capsid entry, accelerating the rate of stable viral integration into the host genome.

Microbiome Dynamics and Pathophysiological Mechanisms

According to Kim (2025), cumulative parity may destabilise the homeostatic architecture of the vaginal micro biome. Prolonged gestational alterations in vaginal pH, localised immunotolerance and hormonal shifts deplete the pool of protective, lactic-acid producing lactobacilli (*Lactobacillus crispatus*, *Lactobacillus jensenii*) which are normally responsible for producing hydrogen peroxide and bacteriocins. The collapse of *Lactobacillus* dominance compromises colonisation resistance, allowing the niche to be overtaken by polymicrobial anaerobic consortia, including *Gardnerella vaginalis*, *Atobium vaginae* and *Prevotella* spp. This anaerobic shift leads to a massive secretion of bacterial mucinases and sialidases that degrade the protective mucous gel layers of the cervix, alongside a heightened release of pro-inflammatory cytokines. Under conditions of localised oxidative stress and epithelial barrier depletion, a permissive environment is generated that allows HR-HPV to successfully evade host immune surveillance, a relationship further corroborated by recent meta-analytic evidence on cervicovaginal microbiota and viral persistence [19].

Furthermore, the biological mechanisms of viral persistence may be further influenced by systemic immune modulators such as Vitamin D. The named vitamin plays a critical role in maintaining the physical barrier in the genitourinary system

by remodeling proteins in tight and gap junctions, facilitating the transition of monocytes into macrophages to enhance phagocytosis. However, clinical data from Zonguldak, Turkey suggests that endemic deficiencies may confound these protective effects: a study of 143 patients with a mean Vitamin D levels were nearly identical between HR-HPV positive (17.63 ± 7.83 ng/mL) and negative (17.54 ± 9.20 ng/mL) groups [17]. Notably, 10.2% of the infected cohort maintained sufficient vitamin levels of more than 30 ng/mL with 28.6% suffering from severe deficiency. This widespread deficiency suggests that for women with high parity, the risk of progression may be compounded: mechanical trauma to the cervical transformation zone during multiple births may be paired with an impaired natural immune response and a weakened mucosal barrier, for both of which are exacerbated by suboptimal Vitamin D levels. Consequently, the persistence of HR-HPV in multiparous women should be viewed not just as a result of reproductive trauma but as a multi-factorial failure of local and systemic host defenses.

Regional Context and Clinical Implications

When positioned within the regional context of Central Asia, this meta-analysis offers insight into shifting public health figures in Kazakhstan. The international analysis confirms that cumulative childbirth history may significantly accentuate the cervical oncogenesis risk with the $I^2=69.12\%$ while Kazakhstan has a relatively high fertility rate of 2.57 children per woman, intersecting with the current evolution in cervical cancer screening dynamics. These findings suggest that regional screening algorithms should integrate reproductive history as a crucial stratification metric for high-risk cohorts.

Limitations

This study has several limitations. First, the number of included studies is relatively small, which may affect statistical power. Second, there was a lack of standardized consensus on parity thresholds across studies (e.g., >2 vs. >3 births), contributing to heterogeneity. Finally, while we attempted to control for parity, socioeconomic status and access to regional screening programs remain potential confounding factors that were not consistently reported across all primary datasets. In addition, the small number of included studies limited the formal

assessment of publication bias, and sensitivity analysis was not performed for the same reason. The small number of included studies also precluded meaningful subgroup analyses or meta-regression. Future studies should utilize standardized parity definitions and large-scale longitudinal data to further refine these associations.

Conclusion

This systematic review and meta-analysis suggest that while the initial physiological transition from a nulliparous to a multiparous state does not significantly alter baseline HR-HPV persistence, high parity is associated with an increased risk of cytological progression. Due to the observational nature of the included studies and observed methodological heterogeneity, these findings should be interpreted with caution. They indicate an association rather than definitive causality, likely mediated by cumulative local mechanical trauma and microenvironmental shifts in the cervical transformation zone. Additional high-quality, large-scale prospective cohort studies are urgently needed to validate these dose-response thresholds and to inform tailored clinical screening protocols in high-fertility settings.

Author Contributions: Conceptualization, N. K.; methodology, N. K. and T. U.; validation, N. K., T. U., and

A. S.; formal analysis, A. S. and S. K.; investigation, N. K., T. U., and A. S.; resources, N. K.; data curation, A. S. and A. K.; writing original draft preparation, N. K., T. U., and K. A.; writing – review and editing, A. K. and A. S.; project administration, N. K. The authors have read and agreed to the published version of the manuscript.

Disclosures: The authors have no conflicts of interest.

Acknowledgments: Authors are thankful to the University Medical Center for providing support to carry out the work.

Funding: This research was funded by the Science Committee of the Ministry of Science and Higher Education of the Republic of Kazakhstan (Grant №BR24992853, Title: National program for study of HPV with development of integrated approach to the effective).

Data availability statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Artificial Intelligence (AI) Disclosure Statement: The authors declare no AI Tools used for preparation of this work.

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