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The Influence of Human Papillomavirus Infection on Reproductive Outcomes Following IVF: A Systematic Review and Meta-Analysis

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ABSTRACT

Background & Objective: Human Papillomavirus (HPV) is common in reproductive-age populations, but its influence on assisted reproductive outcomes remains uncertain. This systematic review and meta-analysis evaluated the association between HPV infection and clinical pregnancy, live birth, and miscarriage after In Vitro Fertilization (IVF) or Intracytoplasmic Sperm Injection (ICSI).

Materials & Methods: PubMed, Scopus, and Web of Science were searched from inception through 2024. Eligible reports compared HPV-positive and HPV-negative groups undergoing IVF/ICSI and confirmed HPV using DNA-based testing. Observational studies and comparative clinical reports were eligible. Random-effects models were used to pool Odds Ratios (ORs) with 95% Confidence Intervals (CIs), coded as HPV-positive versus HPV-negative. Because one study assessed seminal HPV in the male partner and reported cumulative assisted-reproduction outcomes, it was evaluated in sensitivity analysis rather than the primary female/couple-exposure analysis.

Results: Nine reports were included. In the primary analysis, HPV positivity was not significantly associated with clinical pregnancy (8 studies; OR 1.18, 95% CI 0.87-1.60; I^2=14.7%) or live birth (8 studies; OR 0.96, 95% CI: 0.73-1.27; I^2=0%). Miscarriage showed a non-significant trend toward higher odds with HPV positivity (8 studies; OR 1.64, 95% CI 0.96-2.80; I^2=0%). Including the seminal HPV-only study increased the pooled miscarriage estimate (9 studies; OR 1.99, 95% CI: 1.14-3.46; I^2=12.7%), indicating sensitivity to the site and definition of HPV exposure.

Conclusion: Current evidence does not demonstrate a clear reduction in clinical pregnancy or live birth after IVF/ICSI among HPV-positive patients. A possible association with miscarriage remains uncertain and appears sensitive to whether seminal HPV studies are pooled with female/couple HPV exposure. Better-designed prospective studies with standardized HPV sampling and reproductive-outcome definitions are needed.

Keywords: Human papillomavirus, HPV, In vitro fertilization, IVF, Intracytoplasmic sperm injection, clinical pregnancy, Live birth, Miscarriage, Meta-analysis

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1. Introduction

Human Papillomavirus (HPV) comprises a diverse group of epitheliotropic DNA viruses, including low-risk and high-risk genotypes with distinct oncogenic potential **(1,2)**. Persistent infection with high-risk HPV is causally related to cervical and several other anogenital and oropharyngeal malignancies. Because HPV is highly prevalent during the reproductive years, its potential relevance to infertility and assisted reproduction has attracted increasing clinical interest.

Infertility affects a substantial proportion of reproductive-age couples worldwide, although prevalence estimates vary according to population, definition, and method of ascertainment **(3,4)**. Beyond its established oncologic significance, HPV has been investigated as a potential reproductive pathogen. High-risk HPV has been detected along the female reproductive tract, and an association with infertility and endometriosis has been proposed **(5)**.

Clinical studies evaluating HPV and assisted reproductive technology have yielded inconsistent findings. Some reports have described lower conception or pregnancy rates, increased pregnancy loss, or adverse outcomes among HPV-positive patients or couples, whereas others have found no meaningful differences **(6-9)**. These discrepancies may reflect small sample sizes, differences in HPV

genotype, anatomic sampling site, persistence of infection, male-partner infection, infertility etiology, and variation in outcome definitions.

Biological plausibility has been suggested by experimental work showing that HPV exposure can affect early embryonic cells and embryo survival **(10,11)**. HPV infection has also been associated with impaired sperm motility **(12)**, while HPV DNA has been reported more frequently in some products of conception from spontaneous pregnancy loss **(13)**. Nevertheless, the clinical magnitude and independence of these associations remain uncertain.

The present systematic review and meta-analysis therefore aimed to synthesize the available evidence on HPV infection and reproductive outcomes following IVF/ICSI, focusing on clinical pregnancy, live birth, and miscarriage. Particular attention was given to consistency of effect direction, outcome denominators, and the anatomic source of HPV detection because these factors materially influence interpretation of pooled estimates.

2. Materials and Methods

Reporting Framework and Search Strategy

The review was structured in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement **(14)**. PubMed, Scopus, and Web of Science were searched from database inception through 2024 without language restrictions. The core search concept reported in the source manuscript was: (IVF OR in vitro fertilization) AND (human papillomavirus OR HPV). Reference lists of relevant studies and reviews were also considered for additional eligible reports.

Eligibility Criteria

Comparative human studies were eligible when they evaluated patients or couples undergoing IVF/ICSI, classified HPV exposure using DNA-based testing, and reported sufficient data for at least one prespecified outcome. Studies based solely on cervical cytology without HPV DNA confirmation were not eligible. Animal studies, case reports, and case series were excluded. Because the evidence base was predominantly observational, studies were not restricted to randomized controlled trials.

Study Selection and Data Extraction

Two reviewers independently screened titles and abstracts and assessed potentially eligible reports. Disagreements were resolved by consensus. When overlapping populations were suspected, the most complete or most recent report was prioritized. Extracted variables included study setting, design, HPV sampling site, group size, and reproductive outcomes. The principal outcomes were clinical pregnancy, live birth, and miscarriage.

Risk of Bias and Methodological Quality

The source manuscript specified independent methodological appraisal using Joanna Briggs Institute (JBI) critical-appraisal tools appropriate to study design. Because item-level JBI judgments were not contained in the supplied manuscript file, the revised Results section describes the major design limitations that can be established from the reports, while the study-level JBI table should be supplied as a supplementary item before submission.

Statistical Analysis

For consistency of interpretation, effect estimates were coded as HPV-positive versus HPV-negative. Odds Ratios (ORs) and 95% Confidence Intervals (CIs) were pooled using random-effects models. Where a 2×2 table contained a zero cell, a continuity correction was applied. Statistical heterogeneity was quantified using Cochran's Q and the I^2 statistic. The tabulated study-level counts were cross-checked during manuscript revision, and outcome labels/denominators that were clearly transposed were corrected before recalculation. Because fewer than 10 studies contributed to each outcome, funnel-plot asymmetry and Egger regression were not used to make definitive inferences about publication bias.

3. Results

Study Selection

The search identified 768 records. After removal of 351 duplicates, 417 records underwent title/abstract screening, and nine reports met the inclusion criteria. The source file did not provide the complete full-text exclusion breakdown; therefore, the corresponding PRISMA flow-diagram fields should be verified against the authors' screening log before final submission (Figure 1).

Study Characteristics

The included reports were conducted in Italy, the United States, the Czech Republic, Japan, and China. They comprised prospective and retrospective observational studies as well as conference abstracts; none of the reports was a true randomized trial of HPV exposure. The included evidence comprised Comar et al., **(15)**, Kulp et al., **(16)**, Oborna et al., **(17)**, Perino et al., **(7)**, Tanaka et al., **(8)**, Yang et al., **(9)**, Wei et al., **(18)**, Zullo et al., **(19)**, and Garolla et al., **(20)**. Most studies evaluated cervical/female HPV or couple-level HPV status; Garolla et al. assessed seminal HPV in male partners and was therefore treated separately in sensitivity analysis. Study-level characteristics and corrected event counts are summarized in Table 1.

Clinical Pregnancy

Eight female/couple-exposure studies contributed to the primary clinical-pregnancy analysis. HPV positivity was not significantly associated with the probability of clinical pregnancy (OR: 1.18, 95% CI: 0.87-1.60;

P=0.28), with low heterogeneity (I^2=14.7%). When the seminal HPV-only study was added in sensitivity analysis, the pooled estimate moved toward the null (OR: 1.04, 95% CI: 0.68-1.59; P=0.87) and heterogeneity increased (I^2=57.4%), supporting the decision to distinguish exposure site (Figure 2).

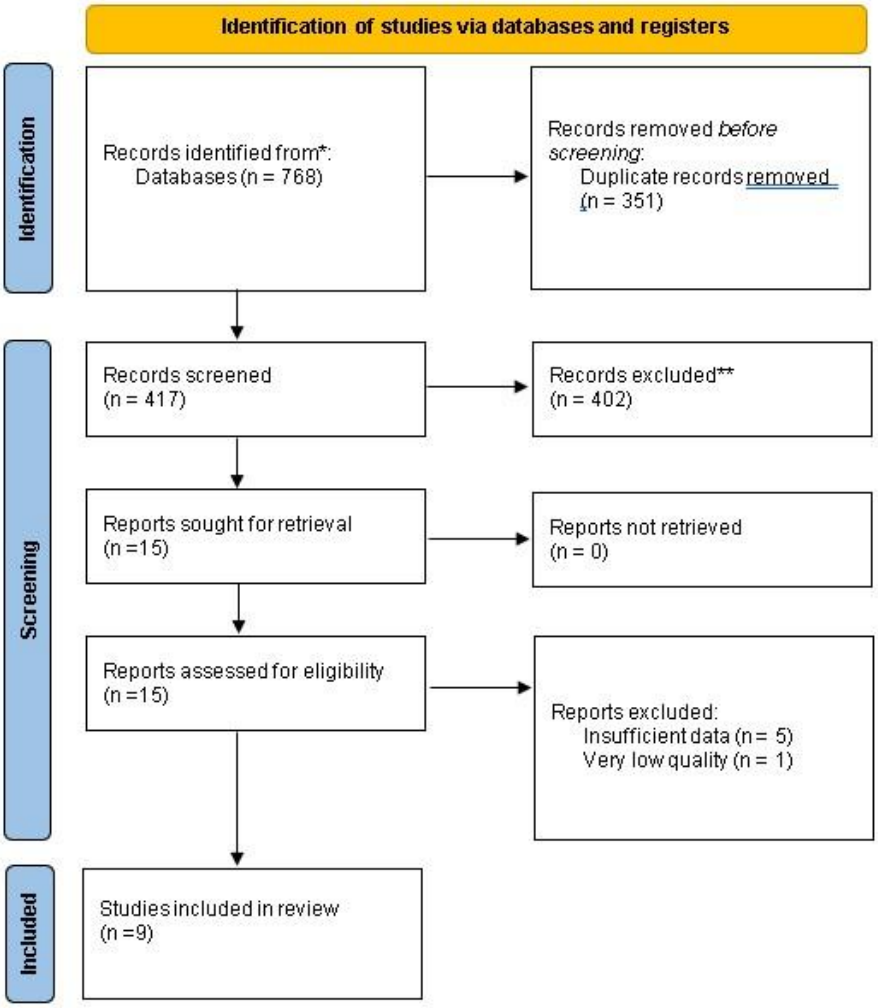


Figure 1. PRISMA flow diagram for study identification, screening, eligibility, and inclusion. The full-text exclusion counts/reasons should be completed from the original screening log

Live Birth

Eight studies reported live-birth data. The pooled estimate showed no significant association between HPV positivity and live birth (OR: 0.96, 95% CI: 0.73-1.27; P=0.79), with no observed heterogeneity (I^2=0%). The seminal HPV-only study did not provide a directly comparable live-birth count for inclusion in this analysis (Figure 3).

Miscarriage

Using miscarriage events relative to clinical pregnancies where the relevant denominator could be reconstructed, eight female/couple-exposure studies showed higher pooled odds of miscarriage among HPV-positive patients, but the association did not reach conventional statistical significance (OR: 1.64, 95% CI: 0.96-2.80; P=0.07; I^2=0%). Inclusion of the seminal HPV-only study increased the pooled estimate to OR: 1.99 (95% CI: 1.14-3.46; P=0.016; I^2=12.7%). The change in statistical significance indicates that the miscarriage result is sensitive to pooling biologically distinct HPV exposure definitions (Figure 4).

Methodological Quality and Small-Study Effects

The evidence base was dominated by observational designs, several studies contained very small HPV-positive groups, and two reports were available only as conference abstracts. These features increase susceptibility to selection bias, exposure misclassification, imprecision, and incomplete outcome reporting. Formal tests for small-study effects were not interpreted because each meta-analysis included fewer than 10 studies.

Table 1. Characteristics and corrected outcome counts of included reports

Study	Country	Design / HPV exposure	Group size HPV− / HPV+	Clinical pregnancy HPV− vs HPV+	Live birth HPV− vs HPV+	Miscarriage* HPV− vs HPV+	Ref.
Comar (2013)	Italy	Prospective cohort; female cervicovaginal HPV	70/12	11/70 vs 2/12	10/70 vs 1/12	2/11 vs 1/2	**(15)**
Kulp (2007)	USA	Observational conference abstract; cervical HPV/Pap abnormalities	16/20	8/16 vs 11/20	7/16 vs 9/20	0/8 vs 2/11	**(16)**
Oborna (2016)	Czech Republic	Observational conference abstract; female HPV	317/45	88/317 vs 18/45	67/317 vs 15/45	21/88 vs 3/18	**(17)**
Perino (2011)	Italy	Prospective cohort; couple HPV status	164/35	51/164 vs 15/35	44/164 vs 9/35	7/51 vs 6/15	**(7)**
Tanaka (2000)	Japan	Observational study; cervical HPV-16	182/10	39/182 vs 5/10	31/182 vs 3/10	8/39 vs 2/5	**(8)**
Yang (2013)	China	Retrospective analysis; female high-risk HPV	56/56	23/56 vs 20/56	17/56 vs 14/56	6/23 vs 5/19	**(9)**
Wei (2022)	China	Retrospective cohort; female HPV	126/224	73/126 vs 124/224	59/126 vs 91/224	6/73 vs 14/124	**(18)**
Zullo (2023)	Italy	Prospective cohort; female cervical HPV	299/27	100/299 vs 8/27	84/299 vs 6/27	15/100 vs 2/8	**(19)**
Garolla (2016)	Italy	Cross-sectional clinical study; seminal HPV in male partner	172/54	66/172 vs 8/54	Not directly comparable	11/66 vs 5/8	**(20)**

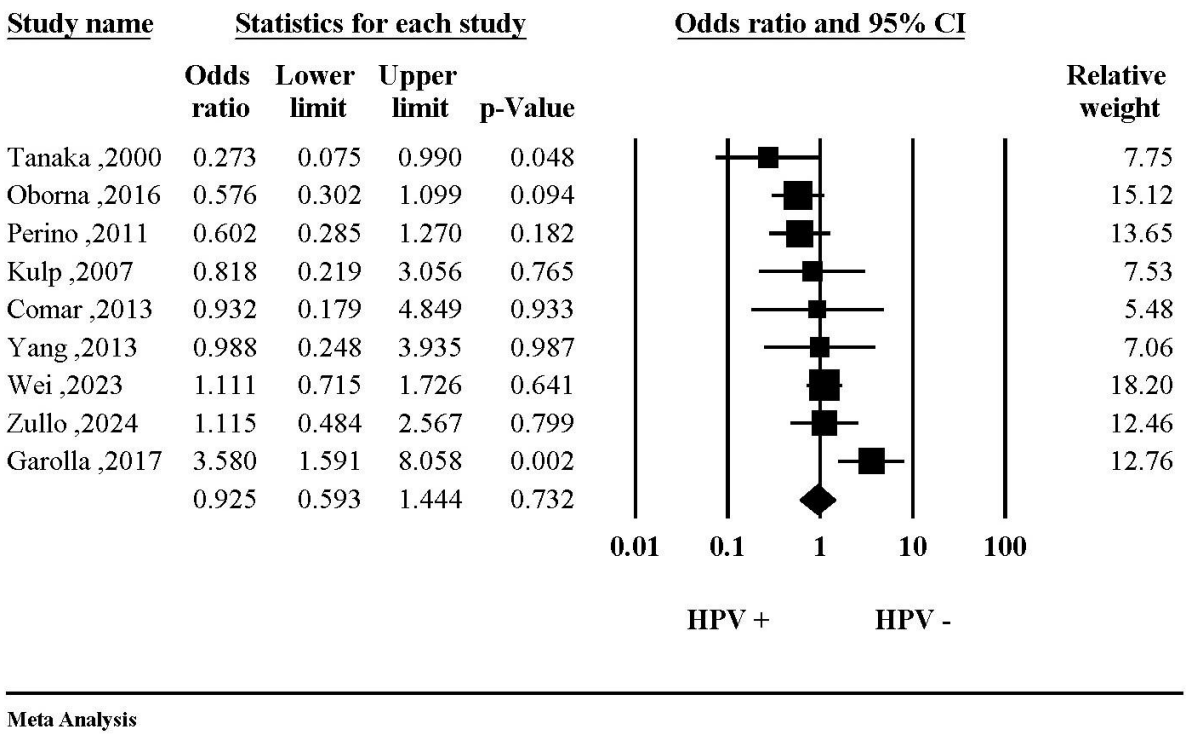


Figure 2. **Forest plot of the association between HPV positivity and clinical pregnancy after IVF/ICSI (HPV-positive versus HPV-negative).**

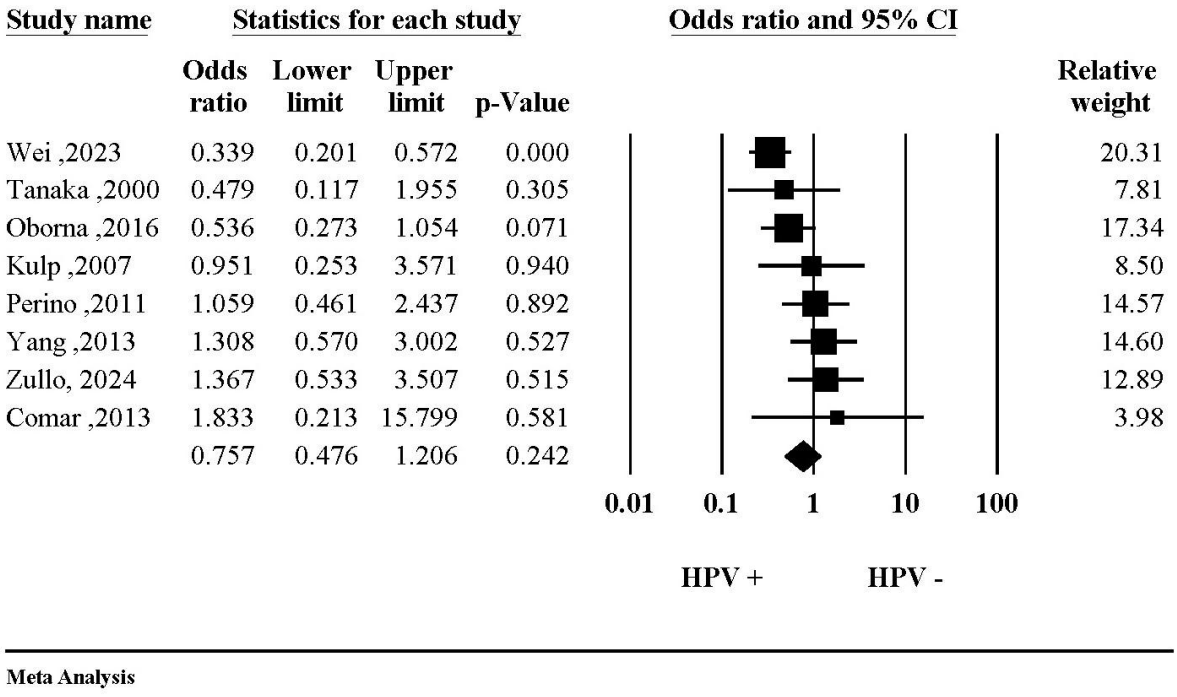


Figure 3. **Forest plot of the association between HPV positivity and live birth after IVF/ICSI (HPV-positive versus HPV-negative).**

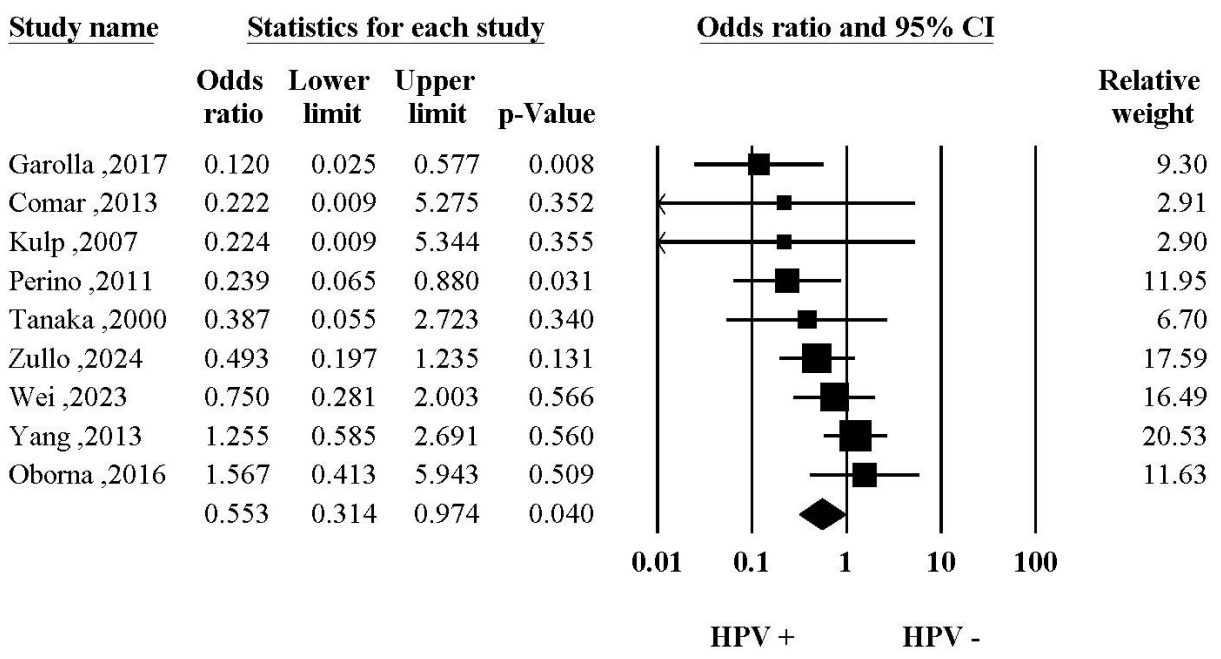


Figure 4. Forest plot of the association between HPV positivity and miscarriage after IVF/ICSI, with sensitivity analysis for inclusion of the seminal HPV-only study.

4. Discussion

Principal Findings

In this revised synthesis, HPV positivity was not associated with a statistically significant difference in clinical pregnancy or live birth after IVF/ICSI. The miscarriage signal was less stable: the primary analysis of female/couple HPV exposure showed a non-significant increase, whereas inclusion of a seminal HPV-only study produced a statistically significant pooled association. The central implication is therefore not that HPV has a uniform detrimental effect on IVF outcomes, but that reproductive associations may differ according to the infected partner, anatomic sampling site, and definition of the outcome.

Comparison with Previous Evidence

These findings are broadly consistent with previous syntheses. Siristatidis et al., found no clear difference in live birth/ongoing pregnancy or clinical pregnancy among women with HPV infection, while miscarriage estimates were imprecise and the quality of evidence was very low **(21)**. Xiong et al., similarly reported no significant association between HPV and ART-based clinical pregnancy or spontaneous abortion of ART pregnancies **(22)**. The present review adds newer female HPV studies but also demonstrates how inclusion of seminal HPV can alter pooled miscarriage estimates.

The distinction between female and seminal HPV is clinically relevant. Seminal HPV has been associated with impaired sperm parameters and poorer reproductive outcomes in a separate systematic review and meta-analysis **(23)**. Conversely, female HPV studies frequently report null or modest associations with IVF success. This difference provides a plausible explanation for the sensitivity of the pooled miscarriage result to the Garolla study **(20)**.

Biological Plausibility

Several mechanisms could link HPV to reproductive outcomes, including local inflammation, altered cytokine signaling, effects on sperm function, viral interaction with gametes, and trophoblastic dysfunction. Infertile women eligible for IVF have also been reported to have a higher frequency of abnormal cervical cytology than population controls **(24)**. Experimental and placental literature suggests that HPV may influence trophoblast biology, but clinical findings remain inconsistent **(25,26)**. A retrospective study of infertile men with seminal HPV reported improved reproductive outcomes after prophylactic HPV vaccination, but its observational design does not establish causality **(27)**. More recent placental cohort data have not demonstrated a clear increase in adverse pregnancy outcomes among women with placental HPV, underscoring the uncertainty of causality **(28)**.

Clinical Implications

The current evidence does not justify concluding that HPV positivity alone materially reduces the chance of clinical pregnancy or live birth after IVF/ICSI. Likewise, the data are insufficient to support universal

postponement of assisted reproduction solely to achieve HPV clearance. HPV vaccination, cervical screening, and management of HPV-related lesions should continue to follow established preventive and gynecologic guidelines. Counseling in fertility care should acknowledge that a possible miscarriage association remains uncertain and may be more pronounced when seminal infection is present.

Strengths and Limitations

Strengths of this revision include explicit harmonization of effect direction, correction of transposed outcome cells in the extracted-data table, separation of seminal HPV-only exposure from the primary female/couple analysis, and inclusion of newer studies. Important limitations remain. The number of studies was small; the HPV-positive groups were often limited; exposure sites and genotypes varied; infertility indications and IVF/ICSI protocols were heterogeneous; and some evidence came from conference abstracts. Outcome definitions were not uniform, particularly for miscarriage denominators. The source manuscript also lacked the item-level JBI appraisal table, a complete PRISMA full-text exclusion breakdown, and the embedded figure files. These reporting components should be supplied before journal resubmission. Mechanistic literature is also evolving, including recent reviews of HPV-associated oxidative stress and reproductive dysfunction **(29)**.

5. Conclusion

Available evidence does not demonstrate a significant association between HPV positivity and clinical pregnancy or live birth after IVF/ICSI. A possible increase in miscarriage cannot be excluded, but the magnitude and statistical significance of this association depend on whether seminal HPV-only studies are pooled with female/couple HPV exposure. Future prospective studies should test both partners, distinguish transient from persistent infection, standardize miscarriage denominators and live-birth definitions, and report outcomes separately by HPV genotype and anatomic sampling site.

6. Declarations

Acknowledgments

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Ethical Considerations

This systematic review and meta-analysis used previously published aggregate data and did not involve direct interaction with human participants; therefore, new institutional ethics approval and informed consent were not required.

Authors' Contributions

Both authors contributed to study conception, literature screening, data extraction, interpretation, and manuscript preparation. The final author-contribution statement should be aligned with the journal's CRediT taxonomy if required.

Conflict of Interest

The authors declare no conflicts of interest related to this work.

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