Impact of Male Partner's Multiple Sexual Relationships on HPV and Squamous Intraepithelial Lesion Prevalence among Women in Polygamous Marriages in Senegal – West Africa

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Abstract

Impact of Male Partner's Multiple Sexual Relationships on HPV and Squamous Intraepithelial Lesion Prevalence among Women in Polygamous Marriages in Senegal – West Africa

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Background: While women's sexual behaviors are established risk factors for HPV infection and cervical cancer, the role of male sexual behavior and marital structure remains understudied. Our study examined the association between polygamous versus monogamous marriage, including the number of co-wives, and human papillomavirus (HPV) infection, squamous intraepithelial lesions (SIL), and invasive cervical cancer (ICC) among women in Senegal.
Methods: This cross-sectional study polled secondary data from three prospective studies conducted in Dakar, Senegal, between 1998 and 2010. The analytical sample included 4,891 women aged 15 years and older. The primary exposure was marital structure, categorized as polygamous versus monogamous marriage, with the number of co-wives examined as a secondary exposure. The outcomes included HPV infection types and cervical disease. The HPV

infection types were determined using polymerase-chain-reaction (PCR) and all positive samples with HPV DNA were genotyped for type-specific HPV while the cervical disease status was determined through standardized cytologic screening and histologic evaluation. Generalized linear models (GLMs) were used to estimate odds ratios (ORs) and 95% confidence intervals for the association between marital structure and HPV-related outcomes. All regression models adjusted for study group, clinic, age, parity, and lifetime number of sexual partners.

Results: Of the 4,891 participants, 2,124 (43.4%) were married in monogamous unions, and 2,767 (56.6%) were married in polygamous unions. Overall prevalence of any HPV was higher among women in polygamous unions compared with women in monogamous unions (30.6% vs. 25.6%; AOR = 1.28, 95% CI: 1.09-1.50). Women in polygamous unions also had higher odds of low-risk HPV infection (AOR = 1.28, 95% CI: 1.03-1.59), multiple HPV infections (AOR = 1.39, 95% CI: 1.08-1.79), and non-vaccine (HPV genotypes not covered by 9-valent vaccine) HPV type infection (AOR = 1.30, 95% CI: 1.07-1.59). Although high-risk HPV prevalence was higher among women in polygamous unions, the association was not statistically significant (AOR = 1.18, 95% CI: 0.98-1.42). No meaningful differences were observed for HPV16 or HPV18 infection. Increasing numbers of co-wives were associated with modest increases in overall HPV, low-risk HPV, and LSIL/CIN1 prevalence. As regards cervical disease, LSIL/CIN1 were slightly more frequent among women in polygamous marriages than among those in monogamous marriages (3.8% vs 2.7%), representing 38% higher odds (AOR = 1.38, 95% CI: 0.93-2.07), while no meaningful associations were observed for HSIL/CIN2-3/CIS or ICC.

Conclusion: Polygamous marriage was associated with a higher burden of HPV infection and LSIL/CIN1 among married women in Senegal, particularly LR-HPV, multiple HPV infections, and non-vaccine HPV types. Although associations with advanced cervical disease were less

pronounced, the findings highlight the potential role of male sexual behavior and marital structure in shaping women's HPV risk and support strengthening HPV prevention and cervical cancer screening strategies in settings where polygamous unions are common. This supports greater inclusion of male-centered approaches in HPV prevention efforts.

I. INTRODUCTION

Human Papillomavirus (HPV) is the most common sexually transmitted viral infection worldwide, with most sexually active men and women acquiring an infection during their lifetime.[1,2] These infections disproportionately affect women. The GLOBOCAN 2022 data indicate that cervical cancer, primarily attributed to persistent HPV infections, ranks fourth globally in incidence (660,000 new cases) and mortality (350,000 deaths).[3] Multiple studies show that women are at a higher risk of HPV infection if they have multiple sexual partners or if their male partners have a high lifetime number of sexual partners.[4–6] Prophylactic HPV vaccines are highly effective in preventing infection from vaccine-targeted HPV types[7], yet vaccination coverage remains low in many parts of the world, particularly in Africa, where several countries lack national vaccination programs.[8]

In sub-Saharan Africa (SSA), where the burden of HPV infection remains high, cervical cancer incidence and mortality are among the highest globally, contrasting sharply with the lowest rates observed in regions such as Northern America and Western Asia[2]. Within SSA, high-risk HPV (HR-HPV) types 16 and 18 are the most prevalent, although types 45 and 35 are also frequently reported.[9] A recent systematic review and meta-analysis estimated a pooled HR-HPV prevalence of 55% in the region.[10] Notably, even within SSA, regional variations exist. For instance, Western Africa has one of the highest age-standardized incidence rates, with 29.6 cases per 100,000 women annually.[11] Studies across West African countries report a high prevalence of HR-HPV, with genotype 16 and 18 consistently detected and a substantial proportion of women found to have multiple HR-HPV infections.[12–14] Similar HPV genotype distributions have been reported in Eastern and Southern Africa.[15,16] In Senegal, these trends are evident both in the

general population and among individuals diagnosed with ICC and cervical intraepithelial neoplasia.[17,18] Collectively, these findings underscore the dominance of HR-HPV 16 and 18, which are targeted by current vaccines and are known to be the primary drivers of cervical cancer globally.[19]

HPV transmission and sexual behavior

Human papillomavirus (HPV) is a highly transmissible sexually transmitted infection (STI) that affects both men and women globally.[1,2] Sexual behavior, particularly early sexual debut, unprotected sex, and multiple sexual partners, significantly increases the risk of HPV transmission, with several studies highlighting that a woman's involvement in multiple sexual partnerships further elevates her risk of HPV infection.[10,13,20]

There is growing evidence that underscores the critical role of men in HPV transmission dynamics. A study reports that nearly one in three men globally are infected with at least one genital HPV type, and approximately one in five men carries one or more HR-HPV types.[1] The global pooled prevalence of any HPV type is estimated at 31%, with 21% for high-risk HPV (HR-HPV), which peaks in men between ages 25 and 29 before slightly stabilizing.[1] These findings highlight sexually active men as a persistent reservoir of infection, capable of transmitting HR-HPV to female partners.

The risk posed by male HPV infection to women is evident from studies showing that male genital HPV infection significantly contributes to the presence of HPV infection and increases the risk of cervical cancer in their female partners.[21–23] Further, a multi-center case-control study in Morocco indicated that a husband having multiple sexual partners is associated with HPV infection in women and cervical cancer.[4] More recently, husbands' HPV-positive status has been

directly linked to higher rates of HPV infection and cervical lesion development in their wives (female partners).[24] Together, these findings underscore the importance of including men in HPV prevention and control strategies to protect women's health.

Country context: Senegal

Senegal, like many West African countries, continues to struggle with high burdens of cervical cancer, a disease primarily caused by persistent infection with HR-HPV types.[17,18,25] HPV vaccination became available to 9-year-old children in 2018 through a national vaccination program using a cost-effective routine service delivery model in schools, health facilities, and community-based outreach sites.[25,26] Despite the World Health Organization's (WHO) recommendations to scale up cervical cancer prevention through HPV screening to 70% of all women and vaccination of all girls aged 9-14, Senegal's cervical cancer screening coverage remains below target levels (9.5% uptake).[1,25,27–29] Although national efforts toward HPV screening and vaccination are gradually gaining traction[25], the uptake remains limited, particularly in rural and socio-culturally conservative areas where awareness, access, cost, and acceptance remain barriers.[28]

Cervical cancer is currently the most commonly diagnosed cancer among women in the country and ranks second among cancers affecting women aged 15 to 44.[30] Cervical cancer and HPV infections are particularly high among women with HIV in Senegal.[31,32] Nonetheless, among HIV-negative women, 27.1% had any HPV DNA detected, and 11.6% had multiple HPV types.[31] Also, the prevalence of HPV remains high in the general female population.[31,33–35] One key but underexplored factor contributing to Senegal's HPV burden is the practice of polygamous marriage, which is legal and culturally embedded, allowing a man to have multiple wives.[36]

Polygamous unions are prevalent in both urban and rural Senegal and have implications for women's sexual and reproductive health.[37] Several risk factors, including having HIV and other sexually transmitted infections, early sexual debut, early childbearing, high parity, and limited autonomy in sexual partnerships, increase women's risk of HPV acquisition and progression to cervical cancer in Senegal.[28,32,38,39] These create a gap in understanding the distribution of high-risk HPV types, particularly among women whose sexual network dynamics may place them at higher risk.

Rationale for the study

Despite the fact that HPV is a sexually transmitted infection involving two individuals, most existing research has focused on individual-level risk factors, particularly among women. In individual-based studies, the number of sexual partners is consistently the strongest risk factor for infection in women and men.[40,41] Women's estimates of their current male partner's number of partners are also predictive of HPV infection.[42] This highlights a critical gap in understanding the dynamics of HPV transmission within sexual partnerships.

Importantly, even women in monogamous relationships remain at risk of HPV infection if their male partners have multiple sexual partners, particularly given that HPV can be transmitted by asymptomatic individuals.[1] This risk is particularly pronounced in polygamous settings. For instance, a study in Ghana found a high crude prevalence of HPV among a largely polygamous population, which may contribute to the persistently high incidence of cervical cancer in such contexts.[43] While mutual monogamy is often promoted as a preventive strategy, its effectiveness hinges on both partners being truly monogamous and uninfected at the start of the relationship. Additional evidence shows that both a husband's multiple sexual partnerships[28] and polygamy[4,44,45] are significantly associated with HPV infection and cervical cancer in women.

More recently, a study of 251 couples found that wives with HPV-positive husbands had higher infection rates for most HR-HPV genotypes, and husbands' HPV positivity was significantly associated with cervical lesion development in wives.[24] Yet, the role of marital structure, particularly polygamy, in shaping HPV transmission dynamics and related cervical disease burden remains underexplored, despite its cultural and demographic significance in Senegal.[36] To address this gap, the present study sought to generate evidence that can directly inform prevention strategies by examining the prevalence and type distribution of HPV and SIL among women in polygamous versus monogamous unions, including how the number of co-wives is associated with HPV infection and SIL.

Despite the growing evidence, there is limited literature in sub-Saharan Africa, including Senegal, on the impact of male sexual behavior, particularly polygamy, on a woman's HPV infection risk and SIL development. Furthermore, current public health strategies in the region have largely overlooked the role of male partners in HPV prevention, and boys are often excluded from HPV vaccination efforts by parents and stakeholders.[25,46] This underscores the need for research that examines male sexual behavior and its influence on HPV prevalence and SIL among women in Senegal, where polygamy and gendered norms shape sexual health risks. Strengthening HPV screening coverage and tailoring interventions to the realities of women in polygamous unions are essential steps toward equity in cervical cancer prevention.

II. STUDY DESIGN AND METHODOLOGY

Research design

This is a cross-sectional study using secondary data from three prospective studies conducted between 1998 and 2010: 1) Epidemiology and biology of HIV positive and HIV negative cervical neoplasia (1998 - 2002); 2) Developing new approaches for cervical cancer control

(2002 - 2007); and 3) HIV-associated DNA hyper-methylation in cervical cancer (2005 - 2010). Details of the studies are published elsewhere.[31,39,47–49] All three parent studies were approved by the University of Washington and the Senegalese Human Subjects Review Boards, and written informed consent was obtained from each participant at enrollment. Data were de-identified, and shared variables across the studies were pooled for this study.

Research setting

The parent studies were conducted in outpatient clinics in Dakar, Senegal. Most of these studies recruited from multiple sites. The first study enrolled women presenting to community health clinics in Dakar, Senegal or Pikine (a suburb of Dakar). The second study was a cervical screening study conducted at both Dantec Hospital (a referral facility for women identified with presumed cancer in the region) and Fann University Hospital (an outpatient clinic that specializes in infectious diseases). The third study was a prospective cohort of women attending cervical screening services at the infectious disease clinic of Fann University Hospital and at Pikine outpatient clinic, where women presented for general care.

Study Population

The parent studies enrolled women aged 15 years and older who had not previously undergone cervical cancer screening and who were unvaccinated against HPV. Specifically, the first parent study enrolled women 35 years and above.[49] The second study enrolled women 15 years and older[47], while the third study enrolled women 18 years and above.[31,48] This analysis focused on married women of all ages (15 years and above); hence, 4,891 observations will be included in this study.

Inclusion Criteria

1. Currently married, either in a monogamous or polygamous marriage.
2. Have available HPV test results and/or cervical disease evaluation through cytology and/or histology.

Exclusion Criteria

Participants who tested positive for HIV were excluded.

Data collection

A structured questionnaire was administered by local researchers to collect information on participants' socio-demographic characteristics, sexual behaviors, and reproductive history. Variables included age, age at first sexual intercourse, age at first pregnancy, number of pregnancies, marital status, number of co-wives, number of lifetime sexual partners, contraceptive use, education level, and smoking status, as well as history of sexually transmitted infections.

Furthermore, participants underwent gynecologic examinations during which cervical swab samples were collected for the detection of HPV. Testing for HPV detection followed procedures described by prior literature[47]. Cervical specimens were tested for HPV DNA using a polymerase-chain-reaction (PCR) assay with MY09 and MY11 L1 consensus primers. DNA extraction was performed using the QIAamp DNA Blood Mini Kit (Qiagen, Valencia, CA) according to the manufacturer's protocol. For some samples, a non-kit-based isolation method was used, in which extraction was performed with 20 μg/ml proteinase K at 37°C for 1 hour, and genomic DNA was ethanol precipitated from 200 μl of the processed sample. DNA quality and integrity were verified by amplification of a 268-base pair region of the human β-globin gene.

The presence of HPV DNA was determined by PCR amplification followed by dot blot hybridization, and all positive samples were subsequently genotyped for type-specific HPV. Most specimens were tested using the Roche Linear Array assay (2000-2005) or a liquid bead microarray assay (2005-2010), both of which detected 38 HPV types, including high- and low-risk genotypes (6, 11, 16, 18, 26, 31, 33, 35, 39, 40, 42, 45, 51, 52, 53, 54, 55, 56, 57, 58, 59, 66, 68, 73, 82, 83, 84, as well as 61, 62, 64, 67, 69, 70, 71, 72, 81, IS39 [a subtype of HPV 82], and CP6108 [also known as HPV 89]). In some cases, genotyping was performed using the Roche Line Blot assay, which identified 27 HPV types (6, 11, 16, 18, 26, 31, 33, 35, 39, 40, 42, 45, 51, 52, 53, 54, 55, 56, 57, 58, 59, 66, 68, 73, 82, 83, and 84). Samples that were positive by PCR amplification but negative on type-specific assays were classified as HPV positive, untyped. Detailed procedures for data and sample collection have been previously published.[39,47,49] Aim 1 included data of all individuals with satisfactory HPV DNA test results (positive or negative). Aim 2 included data of all individuals with satisfactory cervical cytology and/or cervical histology results. Therefore, the final dataset included participants with valid results for HPV, cytology, or histology.

Evaluation of cervical disease status

Cervical disease status was determined through standardized cytologic screening and histologic evaluation. Details are reported in the parent studies.[39,47,49] Cervical cytology was performed using the ThinPrep monolayer cell preparation system (Cytyc Corporation, Marlborough, MA).[39] Specimens were interpreted by cytotechnologists and cytopathologists using the Bethesda classification system, which categorizes findings as negative, atypical squamous cells of undetermined significance (ASCUS), low-grade squamous intraepithelial lesion (LSIL), high-grade squamous intraepithelial lesion (HSIL), carcinoma in situ (CIS), or invasive cervical

cancer (ICC).[49] Cytology slides were reviewed locally in Dakar, and for quality assurance, all slides classified as LSIL or worse, as well as a random subset of negative slides, were re-read by cytopathologists at the University of Washington in Seattle.[39]

Women with abnormal cytologic findings or other clinical indications were referred for colposcopy and biopsy. Colposcopically directed biopsy specimens were collected, fixed in formalin, processed using standard hematoxylin and eosin staining, and classified according to WHO criteria as negative, reactive atypical changes, CIN-1, CIN-2, CIN-3/CIS, or ICC.[47] Biopsies were interpreted by experienced pathologists, without knowledge of clinical or other laboratory findings, and a subset was re-reviewed in Seattle to ensure consistency.[39,47]

Cytology was used for initial screening, while histology served as the diagnostic gold standard for confirmation of cervical intraepithelial neoplasia and ICC.[47] When both cytologic and histologic results were available for a participant, the histologic diagnosis was used as the definitive classification of cervical disease status.

Data analysis

All analyses were conducted using RStudio (version 2025.09.1+401; Posit Software, 2025). Statistical significance was assessed at the 0.05 level, and 95% confidence intervals (CIs) were reported for all estimates.

Descriptive analysis was used to characterize the study population. Frequencies and proportions were calculated for categorical variables (e.g., study group, clinic enrolled, number of co-wives, education level, smoking status). At the same time, means and standard deviations were reported for continuous variables (such as age). Specifically, we summarized socio-demographic and reproductive characteristics of study participants overall and stratified by marital structure

(monogamous vs. polygamous unions). Age was grouped into five categories (<30, 30-39, 40-49, 50-59, and ≥60 years) to reflect the distribution of the study population and to align with prior cervical cancer epidemiology literature. Educational level, birthplace, ethnic group, and smoking status were categorized according to the original survey structure. All descriptive analyses were conducted using complete case counts for each variable, and percentages were calculated using non-missing denominators.

The primary exposure variable was marital structure, defined as polygamous marriage (exposure) versus monogamous marriage. To ensure internal consistency, the marital status and number of co-wives variables were harmonized before analysis. The original marital status variable was numerically coded (2 = married monogamous; 3 = married polygamous) and was recoded into descriptive categories. We then cross-validated marital status with the reported number of co-wives to identify and correct logical inconsistencies. Women classified as monogamously married were assigned zero co-wives, whereas participants who reported polygamous marriage but zero co-wives were coded as missing due to incompatible responses. A cleaned co-wives variable was subsequently used to refine marital status: women with one or more co-wives were categorized as polygamously married, and those with zero co-wives as monogamously married. These harmonized variables were used in all descriptive and inferential analyses.

For Aim 1, we used GLMs to evaluate the association between marital structure and HPV genotype detection, and to estimate odds ratios with 95% CIs. To minimize potential selection bias and ensure an unbiased screening population, women with ICC were excluded from this analysis, as HPV infection is expected among all ICC cases. Analyses considered three HPV-related outcomes: (1) overall HPV prevalence, (2) 38 individual HPV types classified according to oncogenic potential, with types 16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, and 59 classified as

HR-HPV[50], and (3) multiple HPV infections. We conducted a sub-analysis to determine the prevalence and association of HPV 16 and 18 - the widely reported types associated with persistent HPV infection and ICC. Modeling these HPV types allowed assessment of whether the association between marital status and the number of co-wives and infection risk varies by any HPV infection, HR-HPV, LR-HPV, multiple HPV infections, or HPV 16 and 18. Also, we assessed HPV subtypes based on vaccine-preventable characteristics with the 9-valent vaccine (6, 11, 16, 18, 31, 33, 45, 52, 58). Additionally, we assessed whether the risk associated with polygamous marriage increases with an increasing number of co-wives.

For Aim 2, we examined the relationship between marital type and cervical disease outcomes using GLMs. Outcomes were categorized into four levels: Normal/Atypical Squamous Cells of Undetermined Significance (ASCUS), LSIL, HSIL, and ICC. To estimate the association between marital structure and each outcome, we fit separate GLMs using logistic regression. Each model compared one lesion category to the Normal/ASCUS reference group, producing category-specific odds ratios. Separate GLMs were preferred over multinomial logistic regression to allow category-specific interpretation and to account for differences in prevalence and clinical significance among lesion categories. The results are presented as odds ratios with corresponding 95% CIs. Since both outcomes (SIL and ICC) were derived from both cytology and histology, we prioritized histology results when available, as histology represents the diagnostic gold standard. For women without histology, cytology findings were used.

All regression models adjusted for study group, clinic, age, parity, and lifetime number of sexual partners (LSP). These variables were selected based on biological plausibility and prior literature.[1,4,39,47,49] Variables with more than 5% missing data were not included in the main models to avoid loss of sample size, power, and potential selection bias. Because LSP, an

important behavioral risk factor for HPV infection and cervical cancer, had substantial missing data (16.6%), sensitivity analyses were conducted to assess the impact of excluding it from the models. The estimates were materially unchanged, suggesting minimal residual confounding; therefore, LSP was retained in the final adjusted models because of its established relevance to HPV-related outcomes.

III. RESULTS

Socio-demographic and reproductive characteristics of study participants by marital structure (monogamous vs polygamous unions)

The analysis included 4,891 participants, of whom 2,124 (43.4%) were married in monogamous unions and 2,767 (56.6%) in polygamous unions (Table 1). Participants were enrolled in three primary studies: Hypermethylation 1,730 (35.4%), New Approaches 454 (9.3%), and Epi/Bio 2,707 (55.3%). The distribution of marital type differed across studies: the Hypermethylation group had a higher proportion of women in monogamous marriages 857 (40.3%), while the Epi/Bio group had a higher proportion of women in polygamous marriages 1,624 (58.7%). The majority (76.5%) of the participants were recruited from the Pikine clinic. Among women in polygamous marriages only, 992 (58.5%) reported having one co-wife, 452 (26.7%) had 2 co-wives, and 252 (14.9%) had three or more. However, out of participants, 1071 (21.9%) of the total sample and 1071 (38.7%) of those in polygamous unions had missing values for the number of co-wives.

The overall mean age was 43.6 (SD = 8.3), with women in polygamous marriages being somewhat older than women in monogamous marriages. The majority of participants were born in Senegal (97.8%) and of the Wolof ethnic group (61.0%). In addition, the majority (99.6%) of

the participants had never smoked. Of the participants, 1,995 (40.8%) had no formal education, and this proportion was higher among women in polygamous marriages, 1,224 (44.2%), than among women in monogamous marriages, 771 (36.3%). Parity was high across both groups, with 1,354 (27.7%) having had five or six children, with a similar distribution across both groups. With regard to contraceptive use, most participants, 3,632 (74.5%), reported using no method, particularly among women in polygamous marriages, 2,176 (78.9%). Most 2,765 (67.6%) women reported having had only one sexual partner in their lifetime.

Prevalence and association of HPV status among women in polygamous unions compared with those in monogamous unions

Women in polygamous unions were more likely to have detectable HPV infection compared to consistently monogamously married women (Table 2). Overall HPV positivity was 30.6% in polygamous unions versus 25.6% in monogamous unions, corresponding to a 28% odds of prevalent infection (AOR = 1.28, 95% CI: 1.09-1.50). Similarly, HR-HPV infection was also more common among polygamously married women (20.0% vs 17.3%), with 18% higher odds of HR-HPV infection among polygamously married women (AOR = 1.18, 95% CI: 0.98-1.42). LR-HPV infections followed a comparable pattern, occurring in 11.4% of polygamous women versus 9.1% of monogamous women. Women in polygamous unions had 28% higher odds of LR-HPV infection in the fully adjusted model (AOR = 1.28, 95% CI: 1.03-1.59). Further, multiple HPV infections were infrequent but still more common among women in polygamous unions compared to those in monogamous unions (8.7% vs 6.7%), with 39% higher odds among women in polygamous unions (AOR = 1.39, 95% CI: 1.08-1.79).

HPV16 and HPV18 infection patterns among women in polygamous unions compared with those in monogamous unions

The prevalence of HPV16 and HPV18 was low overall and showed minimal differences between women in monogamous and polygamous unions (Table 3). HPV16 prevalence was 5.7% among women in monogamous unions and 6.3% among women in polygamous unions, while HPV18 prevalence was 2.9% and 2.7%, respectively. There were no statistically significant differences in HPV16 or HPV18 infection by marital structure in either univariable or multivariable analyses.

Distribution of vaccine-preventable and non-vaccine preventable HPV types among women in monogamous compared to those in polygamous marriages

Vaccine-preventable HPV types, those included in the 9-valent HPV vaccine, were detected in 14.5% of monogamously married women and 16.7% of polygamously married women. This difference corresponded to 12% increased odds of vaccine-preventable HPV infection among women in polygamous marriages (AOR = 1.12, 95% CI: 0.92-1.37). Vaccine non-preventable HPV types were also more common among women in polygamous unions (14.0%) compared with those in monogamous unions (11.2%). Women in polygamous marriages had 30% higher odds of infection with non-vaccine HPV types (AOR = 1.30, 95% CI: 1.07-1.59) (Table 4).

Prevalence and association of HPV status by number of co-wives in polygamous unions

When examining the number of co-wives among women in polygamous unions, HPV prevalence was slightly more common as the number of co-wives increased (38.7% for one co-wife, 42.5% for two co-wives, and 43.9% for three or more co-wives) (Table 5). HR-HPV prevalence, however, was comparable across co-wife categories, 28.0%, 29.0%, and 28.5%, respectively, with crude and adjusted odds ratios consistently close to the null. Further, LR-HPV infections increased slightly with the number of co-wives (15.4%, 16.6%, and 18.7% for one, two, and three or more co-wives, respectively). For multiple HPV infections, prevalence was similar

across the groups (12.3% for one co-wife, 13.3% for two co-wives, and 12.6% for three or more co-wives). However, no statistically significant differences in HPV outcomes were observed across co-wife categories in either crude or adjusted analyses.

Prevalence and association of HPV16 and HPV18 status by number of co-wives in polygamous unions

Table 6 presents the prevalence of HPV16 and HPV18 across categories of co-wives among women in polygamous unions. HPV16 prevalence was highest among women with two co-wives (12.6%), compared with 8.5% among those with one co-wife and 8.1% among those with three or more co-wives. In the adjusted models, women with two co-wives had 49% higher odds of HPV16 infection compared with those with one co-wife (AOR = 1.49, 95% CI: 0.99-2.24), although the association was not statistically significant. HPV18 prevalence was low across all co-wife groups, ranging from 3.4% to 4.5%. The adjusted model indicated no meaningful association between the number of co-wives and HPV18 infection.

Prevalence and association of vaccine-preventable and vaccine-non preventable HPV types by number of co-wives in polygamous unions

Vaccine-preventable HPV types were detected in 23.7% of women with one co-wife, 24.6% of those with two co-wives, and 21.8% of those with three or more co-wives, with no meaningful differences observed across groups (Table 7). Vaccine non-preventable HPV types were slightly more common with increasing number of co-wives, with prevalence rising from 18.3% among women with one co-wife to 20.6% among those with two co-wives and 21.4% among those with three or more co-wives. Adjusted models produced similar estimates as the crude, with the fully adjusted model showing 17% increased odds for women with two co-wives (AOR = 1.17, 95%

CI: 0.87-1.57) and 30% increased odds for women with three or more co-wives (AOR = 1.30, 95% CI: 0.89-1.87).

Association of SIL or cervical cancer among women in polygamous unions compared to those in monogamous unions

Overall, women in polygamous unions had 38% higher odds of LSIL/CIN1 compared with women in monogamous unions (AOR = 1.38, 95% CI: 0.93-2.07). For HSIL/CIN2-3/CIS, women in polygamous unions had 19% higher crude odds compared with women in monogamous unions (OR = 1.19, 95% CI: 0.83-1.73) with the adjusted model showing a 3% lower odds (AOR = 0.97, 95% CI: 0.63-1.53) (Table 8). Also, women in polygamous unions had 21% lower odds of ICC compared to the reference group (AOR = 0.79, 95% CI: 0.53-1.18).

Association of SIL or cervical cancer by number of co-wives in polygamous unions

Among women in polygamous unions, the distribution of cervical findings varied with an increasing number of co-wives. Compared with women with one co-wive, women with two co-wives had had 24% higher odds of LSIL/CIN1 (AOR = 1.24, 95% CI: 0.66-2.24), while those with three or more co-wives showed 66% higher odds (AOR = 1.66, 95% CI: 0.84-3.17). Also, for HSIL/CIN2-3/CIS adjusted odds ratios suggested slightly higher odds among women with two co-wives (AOR = 1.03, 95% CI: 0.51-2.02) and 0.85 lower odds for three or more co-wives (AOR = 0.85, 95% CI: 0.34-1.90). With regards to ICC, adjusted odds ratios indicated lower odds of ICC for women with two co-wives (AOR = 0.92, 95% CI: 0.47-1.82) and three or more co-wives (AOR = 0.49, 95% CI: 0.20-1.20) compared to those with one co-wife (Table 9).

IV. DISCUSSION

This cross-sectional study examined the association between marital structure and HPV-related outcomes among married women aged 15 and above in Senegal, West Africa. Women in polygamous unions had a consistently higher burden of HPV infection and early cervical abnormalities compared with women in monogamous unions. Higher odds were observed for overall HPV infection, LR-HPV, multiple HPV infections, non-vaccine preventable HPV genotypes and LSIL/CIN1. Although the association between polygamous marriage and HR-HPV infection was not statistically significant, HR-HPV prevalence remained higher among women in polygamous unions. Further, increasing numbers of co-wives were associated with increases in overall HPV prevalence, LR-HPV and LSIL/CIN1 prevalence, suggesting a possible gradient related to increasing sexual network concurrency.

Our findings support the hypothesis that male sexual behavior and marital structure may play an important role in shaping women's HPV exposure and infection dynamics in Senegal. HPV transmission occurs within sexual networks, and sexually active men can serve as reservoirs for HPV infection, particularly in the context of having multiple sexual partners.[1] In polygamous unions, repeated exposure to HPV through shared male partners may increase the opportunities for HPV infection, reinfection, and viral persistence among female partners. Persistent oncogenic HPV infection is a necessary precursor to cervical cancer, while LSIL/CIN1 is often a transient manifestation of active HPV infection that may regress, persist, or less commonly progress to higher-grade lesions depending on factors such as HPV genotype and persistence of infection. Therefore, the higher prevalence of LSIL/CIN1 observed among women in polygamous unions may primarily reflect increased HPV exposure and transmission dynamics within concurrent sexual networks.[29,51] This finding is important in Senegal, where cervical cancer screening remains critically low. Recent national estimates indicate that only 9.5% of women aged 15-49

years have ever undergone cervical cancer screening, which is substantially below the WHO cervical cancer elimination target of screening 70% of women by age 35 and again by age 45.[27,29]

Our findings are consistent with prior literature linking male sexual behavior, multiple sexual partnerships and polygamy to women's HPV infection risk and cervical disease. Studies conducted in Morocco, Algeria, Nigeria and Ghana have reported higher HPV prevalence and increased cervical cancer among women in polygamous unions or among women whose husbands had multiple sexual partners.[4,43–45] In Morocco specifically, husbands' multiple sexual partnerships were significantly associated with ICC among women.[4] A study in China reported that women whose husbands tested positive for HPV had a substantially higher prevalence of HPV infection and cervical lesions compared with women whose husbands tested negative for HPV.[24] Together, these studies reinforce the importance of considering male sexual behavior and couple-level sexual dynamics in HPV prevention strategies.

The higher prevalence of multiple HPV infections among women in polygamous unions observed in this study is consistent with evidence from SSA indicating that concurrent HPV infections are common in high-burden settings and may increase the likelihood of persistent infection and progression to cervical disease.[9,10] In addition, women in polygamous unions demonstrated a higher burden of HPV genotypes not currently covered by the 9-valent vaccine. This finding is notable, given the growing evidence of the circulation of diverse HPV genotypes in West Africa, in addition to the common oncogenic types HPV 16 and 18.[17,34] Although this study did not specifically differentiate high-risk non-vaccine HPV types, these findings highlight the importance of continued surveillance of circulating HPV genotypes to inform future vaccination and cervical cancer screening strategies.

Further, although women in polygamous unions had a higher prevalence of LSIL/CIN1, no statistically significant associations were observed for HSIL/CIN2-3/CIS or ICC. Progression from HPV infection to ICC typically occurs over many years and depends on several additional factors, including the host immune response, persistence of infection, and treatment availability, which may or may not be present in this population[52]. In this study, the duration of exposure to polygamous marriage was unknown, and some unions may not have existed long enough for differences in advanced cervical disease outcomes to be apparent. Nonetheless, the increased burden of HPV infection and LSIL/CIN1 among women in polygamous unions represents an earlier stage of the continuum of ICC, particularly in settings where access to timely cervical cancer screening and treatment remains limited.

Strengths and limitations of the study

Our study has several strengths. First, we utilized a large pooled dataset of 4,891 women derived from three prospective studies conducted over more than a decade in Senegal. Second, the study incorporated detailed HPV genotyping data and cervical cytology and histology assessments, allowing for the examination of both HPV subtype distribution and cervical disease outcomes. Third, by examining both marital structure and number of co-wives, this study provides a more comprehensive understanding of sexual network dynamics and women's HPV risk in a culturally relevant context that remains understudied in SSA. Finally, the study population was largely unscreened, providing a unique opportunity to examine naturally occurring HPV infection and cervical disease patterns in the absence of widespread cervical cancer screening and treatment interventions.

However, several limitations should be considered when interpreting the results. First, the cross-sectional design limits causal inference and prevents assessment of temporal relationships

between marital structure, HPV persistence, and progression to cervical disease. Second, information on the duration and timing of marriages for women and their husbands was unavailable, limiting assessment of the length of exposure to concurrent sexual partnerships. Third, the data were collected between 1998 and 2010, prior to the introduction of the HPV vaccination program in Senegal in 2018, and therefore may not fully reflect the current epidemiology of HPV infection in the post-vaccine era. Fourth, participants were recruited from outpatient and referral clinics, which may limit generalizability to the broader Senegal population. Fifth, although women known to be HIV-positive were excluded, not all participants underwent HIV testing, and some women living with HIV may have been inadvertently included. Sixth, the number of co-wives for women in polygamous unions was missing for almost 40%, which resulted in underpowered analyses related to this factor and might have introduced bias due to differential missing data. Seventh, there were small sample sizes for specific HPV genotypes, HSIL/CIN2-3/CIS and ICC which reduced analytic power for women with specific HPV infections. Finally, residual confounding is possible for sexual behavior variables with missing data exceeding 5% that were not controlled for.

Policy implications

The findings from this study have implications for HPV prevention and cervical cancer control and elimination in Senegal and across SSA.

1. The results support expanding access to secondary prevention services, including HPV screening, to facilitate early detection and treatment of pre-cancerous lesions.
2. The findings underscore the importance of incorporating men more explicitly into HPV prevention efforts through culturally appropriate education, community engagement, and consideration of including young boys in the HPV vaccination strategies. Given that HPV

transmission occurs within sexual partnerships, prevention strategies that focus exclusively on women may overlook important drivers of infection transmission.

3. Although current HPV vaccines protect against the HPV types responsible for the majority of cervical cancers, the higher burden of non-vaccine HPV types observed in this study highlights the importance of continued cervical cancer screening and ongoing surveillance of circulating HPV genotypes as HPV vaccination programs expand across Africa.
4. These findings support the development of culturally tailored, couple-based and community-based HPV prevention strategies and interventions that address sexual network dynamics and barriers to screening and vaccination uptake. Strengthening HPV prevention programs will be essential in achieving the WHO cervical cancer elimination targets and reducing cervical cancer disparities among women in high-burdened settings.

V. CONCLUSION

This study highlights the potential role of marital structure and sexual network dynamics in shaping women's HPV exposure and early cervical disease outcomes in Senegal. Women in polygamous unions demonstrated a higher burden of HPV infection and LSIL/CIN1, suggesting increased opportunities for HPV transmission within concurrent sexual partnerships. These findings underscore the importance of strengthening culturally responsive HPV prevention, cervical cancer screening, and public health interventions in settings where polygamous unions are common. Future longitudinal research incorporating partner-level HPV data and duration of marital exposure is needed to better understand the relationship between polygamous marriage, HPV persistence, and progression to cervical cancer.

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Table 1: Socio-demographic and reproductive characteristics of study participants by marital structure (monogamous vs polygamous unions)

Characteristic	Married-Monogamous (N = 2124)	Married-Polygamous (N = 2767)	Overall Total (N = 4891)
Study group			
Hypermethylation	857 (40.3%)	873 (31.6%)	1730 (35.4%)
New Approaches	184 (8.7%)	270 (9.8%)	454 (9.3%)
Epi/Bio	1083 (51.0%)	1624 (58.7%)	2707 (55.3%)
Clinic			
Dantec	376 (17.7%)	495 (17.9%)	871 (17.8%)
Fann	157 (7.4%)	120 (4.3%)	277 (5.7%)
Pikine	1591 (74.9%)	2152 (77.8%)	3743 (76.5%)
Number of co-wives*			
None	2124 (100.0%)	0 (0.0%)	2124 (55.6%)
1	0 (0.0%)	992 (58.5%)	992 (26.0%)
2	0 (0.0%)	452 (26.7%)	452 (11.8%)
3+	0 (0.0%)	252 (14.9%)	252 (6.6%)
Age group			
Less than 30	105 (4.9%)	27 (1.0%)	132 (2.7%)
30-39	780 (36.7%)	658 (23.8%)	1438 (29.4%)
40-49	913 (43.0%)	1335 (48.3%)	2248 (46.0%)
50-59	266 (12.5%)	575 (20.8%)	841 (17.2%)
60+	59 (2.8%)	171 (6.2%)	230 (4.7%)
Birthplace*			
Senegal	1266 (97.4%)	1572 (98.1%)	2838 (97.8%)
Other West Africa	34 (2.6%)	29 (1.8%)	63 (2.2%)
Outside West Africa	0 (0.0%)	1 (0.1%)	1 (0.0%)
Ethnic group*			
Wolof	968 (54.6%)	1542 (65.9%)	2510 (61.0%)
Pulaar	406 (22.9%)	430 (18.4%)	836 (20.3%)
Serere	218 (12.3%)	209 (8.9%)	427 (10.4%)

Other	180 (10.2%)	160 (6.8%)	340 (8.3%)
Educational Level*			
None	771 (53.8%)	1224 (69.0%)	1995 (62.2%)
Primary	444 (31.0%)	379 (21.4%)	823 (25.7%)
Secondary	198 (13.8%)	160 (9.0%)	358 (11.2%)
University	19 (1.3%)	11 (0.6%)	30 (0.9%)
Parity			
0	91 (4.3%)	110 (4.0%)	201 (4.1%)
1-2	303 (14.4%)	338 (12.3%)	641 (13.2%)
3-4	510 (24.2%)	491 (17.8%)	1001 (20.6%)
5-6	585 (27.7%)	769 (28.0%)	1354 (27.8%)
7-8	418 (19.8%)	695 (25.3%)	1113 (22.9%)
9+	204 (9.7%)	348 (12.7%)	552 (11.4%)
Contraception method			
No contraception	1456 (68.8%)	2176 (78.9%)	3632 (74.5%)
Pill	7 (0.3%)	0 (0.0%)	7 (0.1%)
Injectable	226 (10.7%)	154 (5.6%)	380 (7.8%)
Condoms	199 (9.4%)	186 (6.7%)	385 (7.9%)
Other contraception	204 (9.6%)	214 (7.8%)	418 (8.6%)
Pregnant	24 (1.1%)	27 (1.0%)	51 (1.0%)
Lifetime number of sex partners*			
1	1349 (76.2%)	1416 (61.3%)	2765 (67.8%)
2	376 (21.2%)	758 (32.8%)	1134 (27.8%)
3+	46 (2.6%)	136 (5.9%)	182 (4.5%)
Smoking status*			
Never	1294 (99.5%)	1593 (99.6%)	2887 (99.6%)
Current	5 (0.4%)	7 (0.4%)	12 (0.4%)
Past	1 (0.1%)	0 (0.0%)	1 (0.0%)

*Indicates variables with total missing data >5%: Number of co-wives (21.9%), birthplace (40.7%), ethnic group (15.4%), educational level (34.5%), lifetime number of sexual partners (16.6%), and smoking status (40.7%)

Table 2: Prevalence and association of HPV status among women in polygamous unions compared with those in monogamous unions.

Variable	Married-Monogamous (N = 2124)	Married-Polygamous (N = 2767)	Crude OR (95% CI)	Fully adjusted model (95% CI)
Any HPV			1.28 (1.13 -1.46)	1.28 (1.09-1.50)
No	1559 (74.4%)	1890 (69.4)		
Yes	536 (25.6%)	834 (30.6%)		
Any HR-HPV			1.19 (1.03 -1.38)	1.18 (0.98-1.42)
No	1732 (82.7%)	2179 (80.0%)		
Yes	363 (17.3%)	545 (20.0%)		
Any LR-HPV			1.29 (1.06 -1.56)	1.28 (1.03-1.59)
No	1904 (90.9%)	2413 (88.6%)		
Yes	191 (9.1%)	311 (11.4%)		
Multiple HPV			1.33 (1.07 -1.66)	1.39 (1.08-1.79)
No	1955 (93.3%)	2487 (91.3%)		
Yes	140 (6.7%)	237 (8.7%)		

Any HR-HPV: Any of 16, 18, 31, 33, 35, 39, is39, 45, 51, 52, 56, 58, 59, 66, 68
Any LR-HPV: Any of 6, 11, 26, 40, 42, 53, 54, 55, 57, 61, 6108, 62, 64, 67, 69, 70, 71, 72, 73, 81, 82, 83, 84
Variables adjusted for in all regression models: study group, clinic, age, parity, and lifetime number of sexual partners

Table 3: Prevalence of HPV 16 and 18 among women in polygamous unions compared with those in monogamous unions

Variable	Married-Monogamous (N = 2124)	Married-Polygamous (N = 2767)	Crude OR (95% CI)	Fully adjusted model (95% CI)
HPV 16			1.11 (0.87-1.41)	0.95 (0.71-1.28)
No	1995 (94.3%)	2552 (93.7%)		
Yes	120 (5.7%)	172 (6.3%)		
HPV 18			0.95 (0.67-1.34)	0.89 (0.59-1.34)
No	2035 (97.1%)	2650 (97.3%)		
Yes	60 (2.9%)	74 (2.7%)		

Variables adjusted for in all regression models: study group, clinic, age, parity, and lifetime number of sexual partners

Table 4: Prevalence of vaccine-preventable and vaccine non-preventable HPV types among women in polygamous unions compared with those in monogamous unions

Variable	Married-Monogamous (N = 2124)	Married-Polygamous (N = 2767)	Crude OR (95% CI)	Fully adjusted model (95% CI)
Vaccine preventable			1.18 (1.01-1.39)	1.12 (0.92-1.37)
No	1816 (85.5%)	2304 (83.3%)		
Yes	308 (14.5%)	463 (16.7%)		
Vaccine non-preventable			1.29 (1.09-1.53)	1.30 (1.07-1.59)
No	1886 (88.8%)	2380 (86.0%)		
Yes	238 (11.2%)	387 (14.0%)		

Vaccine-preventable HPV types: 6, 11, 16, 18, 31, 33, 45, 52, 58
Vaccine non-preventable HPV types: 35, 39, IS39, 45, 51, 56, 59, 66, 68, 26, 40, 42, 53, 54, 55, 57, 61, 6108, 62, 64, 67, 69, 70, 71, 72, 73, 81, 82, 83, 84
Variables adjusted for in all regression models: study group, clinic, age, parity, and lifetime number of sexual partners

Table 5: Prevalence and association of HPV status by number of co-wives in polygamous unions

Variable	1 co-wife (N = 992)	2 co-wives (N = 452)	3+ co-wives (N = 252)	Crude OR (95% CI)	Fully adjusted model (95% CI)
Any HPV				2 vs 1: 1.17 (0.93-1.47) 3+ vs 1: 1.24 (0.93-1.64)	2 vs 1: 1.10 (0.85-1.44) 3+ vs 1: 1.32 (0.95-1.83)
No	600 (61.3%)	256 (57.5%)	138 (56.1%)		
Yes	379 (38.7%)	189 (42.5%)	108 (43.9%)		
Any HR-HPV				2 vs 1: 1.05 (0.82-1.34) 3+ vs 1: 1.02 (0.75-1.39)	2 vs 1: 0.95 (0.70-1.28) 3+ vs 1: 1.05 (0.71-1.53)
No	705 (72.0%)	316 (71.0%)	176 (71.5%)		
Yes	274 (28.0%)	129 (29.0%)	70 (28.5%)		
Any LR-HPV				2 vs 1: 1.09 (0.80-1.48) 3+ vs 1: 1.26 (0.87-1.80)	2 vs 1: 1.09 (0.79-1.50) 3+ vs 1: 1.36 (0.91-2.00)
No	828 (84.6%)	371 (83.4%)	200 (81.3%)		
Yes	151 (15.4%)	74 (16.6%)	46 (18.7%)		
Multiple HPV				2 vs 1: 1.09 (0.78-1.52) 3+ vs 1: 1.03 (0.67-1.56)	2 vs 1: 1.07 (0.74-1.52) 3+ vs 1: 1.15 (0.71-1.80)
No	859 (87.7%)	386 (86.7%)	215 (87.4%)		
Yes	120 (12.3%)	59 (13.3%)	31 (12.6%)		

Vaccine-preventable HPV types: 6, 11, 16, 18, 31, 33, 45, 52, 58
Vaccine non-preventable HPV types: 35, 39, IS39, 45, 51, 56, 59, 66, 68, 26, 40, 42, 53, 54, 55, 57, 61, 6108, 62, 64, 67, 69, 70, 71, 72, 73, 81, 82, 83, 84
Variables adjusted for in all regression models: study group, clinic, age, parity, and lifetime number of sexual partners

Table 6: Prevalence and association of HPV 16 and 18 by number of co-wives in polygamous unions

Variable	1 co-wife (N = 992)	2 co-wives (N = 452)	3+ co-wives (N = 252)	Crude OR (95% CI)	Fully adjusted model (95% CI)
HPV 16				2 vs 1: 1.55 (1.08-1.22) 3+ vs 1: 0.96 (0.56-1.56)	2 vs 1: 1.51 (0.98-2.32) 3+ vs 1: 0.90 (0.45-1.73)
No	896 (91.5%)	389 (87.4%)	226 (91.9%)		
Yes	83 (8.5%)	56 (12.6%)	20 (8.1%)		
HPV 18				2 vs 1: 0.74 (0.40-1.32) 3+ vs 1: 0.81 (0.36-1.60)	2 vs 1: 0.69 (0.35-1.26) 3+ vs 1: 0.60 (0.22-1.42)
No	935 (95.5%)	430 (96.6%)	237 (96.3%)		
Yes	44 (4.5%)	15 (3.4%)	9 (3.7%)		

Variables adjusted for in all regression models: study group, clinic, age, parity, and lifetime number of sexual partners

Table 7: Prevalence and association of vaccine-preventable and vaccine non-preventable HPV types by number of co-wives in polygamous unions

Variable	1 co-wife (N = 992)	2 co-wives (N = 452)	3+ co-wives (N = 252)	Crude OR (95% CI)	Fully adjusted model (95% CI)
Vaccine preventable				2 vs 1: 1.05 (0.81-1.36) 3+ vs 1: 0.90 (0.64-1.25)	2 vs 1: 0.93 (0.68-1.27) 3+ vs 1: 0.86 (0.57-1.30)
No	757 (76.3%)	341 (75.4%)	197 (78.2%)		
Yes	235 (23.7%)	111 (24.6%)	55 (21.8%)		
Vaccine non-preventable				2 vs 1: 1.15 (0.87-1.52) 3+ vs 1: 1.21 (0.86-1.70)	2 vs 1: 1.17 (0.87-1.57) 3+ vs 1: 1.30 (0.89-1.87)
No	810 (81.7%)	359 (79.4%)	198 (78.6%)		
Yes	182 (18.3%)	93 (20.6%)	54 (21.4%)		

Vaccine-preventable HPV types: 6, 11, 16, 18, 31, 33, 45, 52, 58

Vaccine non-preventable HPV types: 35, 39, IS39, 45, 51, 56, 59, 66, 68, 26, 40, 42, 53, 54, 55, 57, 61, 6108, 62, 64, 67, 69, 70, 71, 72, 73, 81, 82, 83, 84

Variables adjusted for in all regression models: study group, clinic, age, parity, and lifetime number of sexual partners

Table 8: Association of cervical screening outcomes among women in polygamous unions compared to those in monogamous unions

Cervical screening outcomes	Married-Monogamous (N = 2124)	Married-Polygamous (N = 2767)	Crude OR (95% CI)	Fully adjusted model (95% CI)
Normal/ASCUS	1640 (83.1%)	2075 (81.7%)	Ref	Ref
LSIL/CIN1	53 (2.7%)	96 (3.8%)	1.43 (1.02 - 2.03)	1.38 (0.93-2.07)
HSIL/CIN2-3/CIS	49 (2.5%)	74 (2.9%)	1.19 (0.83 - 1.73)	0.97 (0.63-1.53)
Invasive Cancer	232 (11.8%)	296 (11.6%)	1.01 (0.84 - 1.21)	0.79 (0.53-1.18)

Variables adjusted for in all regression models: study group, clinic, age, parity, and lifetime number of sexual partners

Table 9: Association of cervical screening outcomes by number of co-wives in polygamous unions

Variable	1 co-wife (N = 992)	2 co-wives (N = 452)	3+ co-wives (N = 252)	Crude OR (95% CI)	Fully adjusted model (95% CI)
Normal/ASCUS	712 (74.7%)	305 (70.1%)	183 (74.4%)	Ref	Ref
LSIL/CIN1	41 (4.3%)	23 (5.3%)	17 (6.9%)	2 vs 1: 1.31 (0.76-0.08) 3+ vs 1: 1.61 (0.87-2.86)	2 vs 1: 1.24 (0.66-2.24) 3+ vs 1: 1.66 (0.84-3.17)
HSIL/CIN2-3/CIS	33 (3.5%)	20 (4.6%)	14 (5.7%)	2 vs 1: 1.41 (0.79-2.48) 3+ vs 1: 1.65 (0.84-3.09)	2 vs 1: 1.03 (0.51-2.02) 3+ vs 1: 0.85 (0.34-1.90)
Invasive Cancer	167 (17.5%)	87 (20.0%)	32 (13.0%)	2 vs 1: 1.21 (0.91-1.62) 3+ vs 1: 0.75 (0.49-1.11)	2 vs 1: 0.92 (0.47-1.82) 3+ vs 1: 0.49 (0.20-1.20)

Variables adjusted for in all regression models: study group, clinic, age, parity, and lifetime number of sexual partners

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