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Cervical and Anal Human Papillomavirus Infection in Adult Women in American Samoa

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Abstract

The prevalence of cervical and anal human papillomavirus (HPV) and risk factors associated with infections were evaluated in a cross-sectional study of 211 adult women in American Samoa. Overall, 53% of women reported ever having a Pap smear. Cervical and anal HPV was detected in 10% and 16% of women, respectively; 4% of women had concurrent cervical and anal HPV. The most common cervical genotypes were HPV 6, HPV 16, and HPV 53. Cutaneous HPV types were detected in 40% of anal infections. Cervical HPV infection was associated with anal HPV (age-adjusted odds ratio = 3.32, 1.10–10.00). After age adjustment, cervical HPV was associated with being unmarried, postsecondary education, hot running water at home, multiple sexual partners, nulliparity, condom use, and other contraceptive methods. In multivariate analyses, only age remained associated with cervical HPV and anal HPV. Cervical and anal HPV was more prevalent among younger women; only anal HPV was detected in older women.

Keywords

human papillomavirus; HPV; cervical; anal; American Samoa

Introduction

Human papillomavirus (HPV) is the primary cause of cervical cancer, which remains the second most common malignancy in women worldwide.¹ The prevalence of genital HPV infection varies substantially across different regions of the world.² Geographic variation in HPV prevalence may reflect differences in sexual behavior across populations.³ It is not

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Declaration of Conflicting Interests

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known to what extent the variation in HPV prevalence across populations contributes to differences in cervical cancer occurrence. The majority of cervical malignancies occur in developing countries where cytologic screening is not widely used.⁴ At present, there are no data on the prevalence of HPV infection in the developing nations and territorial entities of Polynesia.

Low levels of cervical cytology screening are evident in American Samoa, a United States territory located in west central Polynesia with a population of approximately 57 000.⁵ In a 2001 population-based survey of American Samoans, only 32% of females aged 18 to 75 years reported having a Pap smear within the prior 3 years.⁶ During the same time period, 87% of adult females in the United States reported having a Pap smear within the prior 3 years.⁷

In addition to cervical malignancies, HPV is also the cause of the majority of anal cancers in both women and men.⁸ Although rare compared with cervical malignancies, the incidence of anal cancers has been increasing in both sexes in the United States and in other countries over the past several decades.^{9,10} Nonetheless, there are scant data on the prevalence of anal HPV in any part of the world.

The present study sought to evaluate the prevalence of both cervical and anal HPV and risk factors associated with these infections in a cross-sectional sample of adult women in American Samoa prior to the introduction of prophylactic HPV vaccination in the community.

Methods

Study Recruitment

The study was approved by the institutional review boards of the University of Hawaii and the Lyndon B. Johnson Tropical Medical Center (LBJ TMC), the only acute care hospital in American Samoa servicing residents from throughout the region. Written informed consent was received from all participants. Study eligibility was limited to females 18 years old, not currently pregnant and at least 6 months postpartum, and having an intact cervix (no prior hysterectomy).

Study participants were recruited from American Samoa's island of Tutuila where 97% of the population resides. Recruitment focused on 6 villages in Tutuila, which collectively had a female population of approximately 7800. Prospective study participants were informed of the study through verbal and written communication from village elders and community leaders within each of the villages. Bilingual health care providers within LBJ TMC also provided study information to prospective participants attending the LBJ TMC obstetrics and gynecology clinic, which provides family planning, prenatal, and gynecologic services to patients throughout American Samoa. The majority of these clinic attendees also came from the 6 villages. The total number of women provided information on the study was not collected. Women interested in participating in the study and meeting the eligibility criteria were scheduled for a study visit. A total of 211 women were enrolled in the study between

November 2008 and March 2009. During this time period, the prophylactic HPV vaccination had not yet been introduced into the community.

Collection of Data and Specimens

Study visits were conducted at the obstetrics and gynecology clinic of LBJ TMC in Pago Pago. A structured questionnaire was used to collect information on demographic characteristics, sexual history, contraceptive use, medical history, and substance use. Specific sexual history questions relevant to HPV exposure but deemed by community members to be overly sensitive were omitted from the questionnaire. The study was available to participants in both Samoan and English languages. The questionnaire was administered by trained female study personnel fluent in both languages.

Study clinicians collected cervical cytology (Pap) specimens. Following the Pap smear, exfoliated cells from the cervix and anus were sequentially collected for HPV testing. Cervical specimens were collected with a cytobrush from the endocervix and ectocervix, including the transformation zone. Anal specimens were collected from the anal canal using a Dacron swab moistened with sterile saline. Specimens were placed into 1-mL collection media (Qiagen, Valencia, CA) and immediately stored at 4°C prior to transport. Pap smear slides and cell specimens (on ice) were sent via air to Honolulu, Hawaii. Pap smears were read by experienced cytotechnologists and abnormal results were confirmed by a pathologist. Cytology results were classified according to the updated Bethesda Classification System.¹¹

HPV DNA Testing and Genotyping

DNA was extracted from cervical and anal cell specimens using commercial reagents (Qiagen). The polymerase chain reaction (PCR) used PGMY09/PGMY11 primers to amplify a 450-bp consensus region of the L1 HPV genome. These primers are capable of detecting a broad range of HPV types.¹² HPV-positive specimens were subsequently genotyped using a reverse line blot detection method for 37 different mucosal HPV genotypes (Roche Molecular Systems, Basel, Switzerland). This commercial genotyping assay detects the major genital HPV types.¹³ HPV-positive specimens that were subsequently found to be negative in the genotyping assay were reevaluated using an assay detecting a broad range of cutaneous HPV types that are not included in the genotyping assay. This nested PCR assay uses degenerate primer pairs (FAP59/64 and FAP 6085F/6319R) targeting a 235-bp region of L1.¹⁴ HPV-positive specimens that were negative by both the Roche assay and the nested PCR assay were considered to be untyped. Cutaneous HPV types were not individually genotyped. All specimens were also PCR tested using GH20 and PC04 primers to amplify a 268-bp region of the human β -globin gene as an internal control for sample sufficiency. Specimens testing negative for β -globin were considered to be insufficient and were excluded from analyses.

Statistical Analysis

SAS software version 9.1 (SAS Institute, Inc, Cary, NC) was used for analyses of data. HPV genotypes were grouped as any HPV, oncogenic HPV, and other HPV. Oncogenic types included HPV 16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, and 68.¹⁵ Other HPV included

nononcogenic types and HPV types of undetermined risk status (HPV 6, 11, 26, 34, 40, 42, 44, 53, 54, 55, 61, 62, 64, 66, 67, 70, 71, 72, 73, 81, 82, 83, 84, and 89). Individual genotypes were not available for cutaneous HPV types, which include papillomavirus types HPV 5, 8, 9, 12, 14, 15, 17, 19, 20, 21, 22, 23, 24, 25, 36, 37, 38, 47, 49, 75, 76, 80, 92, 93, and 96.^{14,16} The association of possible risk factors with HPV status was evaluated using unconditional logistic regression with adjustment for participants' age at the time of study visit. Significant variables were included in multivariate logistic regression models. Odds ratios and 95% confidence intervals were used as measures of association. All tests were 2-sided, and $P < .05$ was considered statistically significant.

Results

Characteristics of the study population and HPV DNA and cytology results are detailed in Table 1. Data were missing from a substantial number of variables because of nonresponse to specific questions. A total of 211 adult women aged 18 to 82 years were enrolled. The majority of women were of Samoan ancestry and 45% were born in American Samoa. The majority of women were married and more than half were not educated beyond high school. More than half of women had no income or an annual income of less than US\$10 000. Most participants spoke Samoan exclusively in their households and the majority reported adhering to traditional and communal lifestyles. A total of 13% of women were currently tobacco smokers. Overall, 53% percent reported ever having a Pap smear and 11% of these women reported a history of abnormal Pap smear. The majority of women were parous and most reported having had vaginal intercourse. Birth control pills (33%) and condoms (20%) were the most commonly used contraceptives.

Cervical HPV infection was detected in 10% of females. Anal HPV infection was detected in 16%. Concurrent cervical and anal HPV infections were observed in 4% of women. Four women had cytologic evidence of cervical dysplasia and all were positive for cervical HPV DNA. Among cytologically normal women, cervical and anal HPV prevalence was 8% and 14%, respectively. Cytologic evidence of bacterial vaginosis was found in 21 (10%) women.

In total, 19 different HPV genotypes were detected in the cervix (Figure 1). The most common cervical types were HPV 6, 16, and 53, each of which was detected in 24% of infections. Ten of the 21 cervical HPV infections involved multiple HPV types. A total of 16 different genotypes were detected in the anus. HPV 53, found in 17% of infections, was the most common type. Seven anal infections involved a single HPV type and 9 were infection with multiple HPV types. In addition, 12 anal infections were of cutaneous HPV types and 2 were untyped. All 7 concurrent cervical–anal infections involved one or more of the same genotype present in both cervix and anus.

The distribution of HPV infection by age group differed for cervical and anal infections (Figure 2). Cervical and anal HPV infections were most frequently observed in the youngest women, peaking at ages 24 to 29 years. Compared with younger women, cervical HPV prevalence was substantially lower among women aged 30 to 49 years, and no infection was observed among those 50 years and older. Anal HPV prevalence also declined with age but was observed in all age groups.

The relationship of cervical and anal HPV infection to characteristics of the study population was evaluated (Table 2). Cervical HPV was associated with concurrent anal HPV infection. Women younger than 30 years were at increased risk of cervical HPV. Single women were at increased risk of cervical infection compared to their married counterparts. Elevated risks of cervical HPV were also observed among women with postsecondary education and among those with hot, running water in their home. Women with a history of pregnancy were at decreased risk of cervical HPV. All women with cervical HPV reported a history of vaginal intercourse. Women reporting 5 or more male sexual partners in their lifetime were at increased risk of cervical HPV. Cervical HPV was also associated with ever use of specific contraceptive practices including condoms, natural family planning, birth control pill, withdrawal prior to ejaculation, and the morning after pill. In multivariate analyses, only age <30 years remained a significant predictor of cervical HPV.

Few risk factors were observed for anal HPV infection. All women with anal HPV reported a history of vaginal intercourse. In multivariate analyses, women younger than 30 years had an increased risk of anal HPV.

Discussion

We are the first to report on the prevalence of HPV infection among adult women in American Samoa. Our prevalence estimates were not influenced by HPV prophylactic vaccination as the study was conducted prior to introduction of vaccination into the community. The overall 10% prevalence of cervical HPV (8% in cytologically normal women) is consistent with estimates in other parts of the world. In a pooled analysis of women aged 15 to 74 years from 11 countries in Africa, Asia, South America, and Europe, the overall age-adjusted prevalence of cervical HPV among cytologically normal women ranged from 1% to 26% (mean 9%).¹⁷ In a population-based US sample not limited to cytologically normal women, the overall prevalence of genital HPV among females aged 15 to 59 years was 27%.¹⁸

Interestingly, the prevalence of anal HPV infection was higher than that of cervical HPV in American Samoa. Little is known regarding the epidemiology of anal HPV in immune competent women and there are no global estimates of anal HPV infection. In a multiethnic cohort of healthy women in Hawaii, the prevalence of anal HPV was comparable to that of cervical HPV¹⁹ and both were higher than that observed in American Samoa. The extent to which anal HPV infection is responsible for the increase in anal cancers in different parts of the world is not known.

Cervical cancer has been observed to be one of the most common malignancies in other Pacific Island populations.²⁰ Although an excess incidence of invasive cervical cancer in American Samoa has not been definitively documented, this population is likely at a high risk for developing invasive cervical cancer given the lack of screening. A little more than half of the women reported ever having a Pap smear, confirming the results of a previous population-based study in American Samoa, which observed low levels of Pap screening.⁶ Although 11% of previously screened women reported an abnormal Pap history, this may be

an underestimate given that 34% of these women reported having diagnostic or treatment procedures related to cervical abnormalities.

Our results are consistent with the evidence that cervical HPV infection is largely tied to sexual behavior.³ Global variation in the prevalence of cervical HPV have been, to some extent, attributed to differences in sexual practices in different parts of the world.^{3,21} We observed that risk of cervical HPV was elevated among younger women—an observation consistently seen in other parts of the world and commonly attributed to the high rate of HPV acquisition with initial sexual activity.^{2,3} Age of initial sexual activity, a risk factor inconsistently observed across populations,³ was not associated with risk of cervical infection in this population. Age at sexual debut in this population was older than that reported in the United States²² and may reflect the influence of traditional cultural norms. An increased risk of cervical HPV infection was observed among women who had never been married. Marital status is likely a proxy for sexual activity in this population whereby single adult women are more likely to have multiple partners. Conversely, a positive pregnancy history may be a proxy for marriage and fewer sexual partners, which may explain the inverse association of pregnancy and cervical HPV. Higher education and hot-running water in the home, which were both associated with increased cervical HPV risk, may also be indicators of a less traditional lifestyle, including sexual behavior. The positive association of condom use and cervical HPV has been inconsistently observed in other populations.³ Nonbarrier contraceptive practices—natural family planning, withdrawal, birth control pills, and the morning after pill—were also associated with increased risk of cervical infection. Contraceptive use may also be a proxy for greater sexual activity.

Few risk factors were observed for anal HPV infection. Anal intercourse has been identified as a risk factor for anal HPV.²³ Because of concerns over cultural acceptability; however, we did not include questions regarding anal intercourse in the questionnaire. Consequently, we were not able to examine the association of anal intercourse with anal HPV.

The lack of risk factors for anal HPV infection is consistent with random, passive transmission from proximate genital sites. Women with concurrent cervical–anal infections were infected with one or more of the same genotypes at both sites. Genotype concordance between paired cervical–anal infections was also observed in our Hawaii cohort¹⁹ and may indicate a common source of infection or passive transmission from one site to the other. Nonetheless, most women with anal HPV infection did not have concurrent cervical HPV infection; only 4% of women were infected at both sites.

Substantially more women had an anal HPV infection alone compared with those with cervical HPV alone. Overall, 40% of anal HPV infections were cutaneous HPV types. Cutaneous HPV types are commonly found on human skin and are considered to be nononcogenic, although certain types have been associated with a rare squamous cell carcinoma.¹⁶ Cutaneous HPV types are distinct from the mucosal HPV types of the α -genus, which are predominant in the genitals and comprised all genotypes detected in cervical HPV in this population. As we did not test samples with identified α genus types for cutaneous HPV, so we cannot overlook the possibility that specimens positive for α -genus types appear together with cutaneous HPV types.

When all variables were controlled for, only age remained a significant predictor of cervical and anal HPV. The age distributions of cervical and anal HPV infection were very different. Cervical HPV was most prevalent in women younger than 30 years; prevalence was highest in the 24- to 29-year age group. Cervical HPV prevalence dropped dramatically after age 30 and was not observed among women 50 years and older. This pattern of peak viral prevalence among the youngest women is a general pattern observed in other regions of the world, including the United States, Europe, and parts of Asia.^{2,21,24} Anal HPV infection, by contrast, showed a wider age distribution in our Samoan population.

Our results provide evidence that anal HPV infection is epidemiologically distinct from cervical HPV. The lack of sexual and other risk factors, broader age distribution, and predominance of cutaneous HPV types suggests that anal HPV infection in this population may be acquired primarily through nonsexual modes involving passive contact and/or auto-inoculation.

Nonetheless, our findings must be interpreted with caution as given our limited sample size, which resulted in wide confidence intervals for our measures of association. The small sample size may also have limited the representativeness of our study population, which accounted for approximately 3% of the female population in the 6 villages targeted for recruitment. The large majority of the study population was of Samoan ancestry and most appeared to adhere to traditional Samoan lifestyle. It has been anecdotally reported that clan and matriarchal systems strongly influence sexual behavior and as well as health care utilization in American Samoa and adherence to these traditional systems varies widely across villages. Our ability to assess sexual history was limited as potentially offensive questions were excluded in the development of the questionnaire. Furthermore, nonresponse to questions, particularly those regarding sexual history, was substantial. Consequently, we were unable to fully account for potential confounders in the evaluation of factors associated with HPV. In addition, some study participants may have been acquainted with study staff who were all from the community. To some extent, this may have influenced both nonresponse as well as the candidness of responses

The present cross-sectional study only provides limited information on the natural history of HPV in this population. Prevalence measures of HPV DNA provide a limited measure of the epidemiology of this sexually transmitted infection given the highly transient nature of this infection. Persistent infections with oncogenic HPV types are most clinically relevant to cervical cancer risk.²⁵ Ideally, HPV should be measured through repeated follow-up over extended periods of time to evaluate the duration of infection. In our cohort of women in Hawaii, we observed that the rates of acquisition of cervical and anal HPV were comparable, but HPV cleared more rapidly in the anus than the cervix.^{26–28} Greater persistence of oncogenic HPV types in the cervix than the anus could explain the higher incidence of cervical compared with anal malignancies observed globally.

Conclusion

The burden of cervical cancer in America Samoa is not fully known. Nonetheless, given the low rates of cytologic screening, this population would likely benefit from comprehensive cervical cancer control strategies.

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Figure 1.
Distribution of human papillomavirus (HPV) genotypes in cervical and anal infections,
American Samoa, 2008–2009

Figure 2.

Cervical and anal human papillomavirus (HPV) prevalence by age group, American Samoa, 2008–2009

Table 1

Study Population, Human Papillomavirus (HPV) DNA, and Cervical Cytology: American Samoa, 2008–2009
(N = 211)

	n ^a	Percentage ^b
Age in years; mean (SD)	40.2 (13.4)	
18–23	33	16
24–29	29	14
30–39	45	21
40–49	53	25
50–59	36	17
60+	15	7
Ethnicity/race		
Samoan	167	84
Part Samoan	9	4
Other	23	12
Birthplace		
American Samoa	92	45
Western Samoa	70	34
United States	23	11
Other	20	10
Marital status		
Single/never married	34	17
Married	130	64
Living as married	12	6
Separated/divorced	13	6
Widowed	15	7
Education level completed		
Less than high school	14	7
Some high school	35	17
High school graduate	59	28
Vocational/technical school	18	9
Some college	50	24
College degree	31	15
Employment		
Full time	107	55
Part time	14	7
Student (part or full time)	14	7
Not employed/other	59	30
Annual income (\$)		
None	11	6
<10 000	91	49
10 000	77	41

	n ^a	Percentage ^b
Don't know	8	4
Primary language		
Samoan	135	67
Samoan and English	21	10
English	35	17
Other	10	5
Traditional lifestyle indicators ^c		
Practice fa'alavelave	175	86
Participate in the to'onai	118	58
Participate in village activities	103	51
Live on communal land	130	64
Hot, running water in home	44	22
Air conditioning in home	72	35
Cigarette smoking (daily for 6 months or more)		
Ever	31	17
Currently	25	13
Alcohol consumption (≥ 1 drink/week for ≥ 6 months)		
Ever	22	13
Cancer history		
Cervical cancer	1	<1
Other cancer	2	1
Pap smear history		
Ever	99	53
Within past 5 years	54	29
Abnormal Pap ^d	11	11
Cervical abnormality-related procedure ^{d,e}	30	34
Ever pregnant	138	82
Ever vaginal sex	161	90
Age in years at first vaginal sex ^f ; mean (SD)	20.5 (3.8)	
<17	19	12
17–19	47	30
20–24	63	41
25	26	17
Ever oral sex	72	47
Contraceptives (ever used) ^f		
Condom	28	20
Intrauterine device	16	11
Morning after pill	8	6
Natural family planning	19	14
Birth control pill	50	33
Birth control patch	15	10

	n ^a	Percentage ^b
Tubal ligation	10	7
Withdrawal	21	15
HPV DNA		
Cervical HPV+		
Any type ^g	21	10
Oncogenic type(s) ^h	11	5
Anal HPV+1		
Any type ⁱ	30	16
Oncogenic type(s) ^h	8	4
Cervical and anal HPV DNA ^j		
Cervical+ anal–	11	6
Cervical– anal+	23	12
Cervical+ anal+	7	4
Cervical– anal–	146	78
Cervical cytology ^k		
Normal	194	96
Atypia	3	2
Low-grade squamous intraepithelial lesion (SIL)	3	2
SIL (grade unspecified)	1	<1
Normal cervical cytology		
Cervical HPV DNA-positive ^l	15	8
Anal HPV DNA-positive ^m	25	14
Cervical cytologic microflora ^k		
Bacterial vaginosis	21	10
Trichomonas vaginalis	5	2
Candida	5	2

^aDenominators vary by variable because of missing data resulting from nonresponse. Missing data: ethnicity/race (n = 12); birthplace (n = 6); marital status (n = 7); education level (n = 4); employment (n = 17); income (n = 24); primary language (n = 10); practice fa'alavelave (n = 7); participate in village activities (n = 13); participate in the to'onai (n = 18); live on communal land (n = 16); hot, running water in home (n = 29); air conditioning in home (n = 19); cigarette smoking (n = 24); regular alcohol consumption (n = 39); history of cancer (n = 32); Pap smear history (n = 24); cervical abnormality-related procedure (n = 48); ever pregnant (n = 42); ever vaginal sex (n = 33); age at first vaginal sex (n = 39); oral sex (n = 57); condoms (n = 53); intrauterine device (n = 52); morning after pill (n = 58); natural family planning (n = 56); birth control pills (n = 42); birth control patch (n = 49); tubal ligation (n = 58); withdrawal (n = 58).

^bTotal percentages slightly less than or greater than 100 because of rounding.

^cFa'alavelave is a practice involving family and extended family members pooling their resources to help family members in need; to'onai is the practice of family and extended families coming together for a meal, attendance at church, or other village events.

^dExcludes women reporting never having a Pap smear.

^eIncludes repeat Pap smears, biopsy, colposcopy, cryotherapy, laser therapy, and surgery.

^fExcludes individuals with no history of vaginal sex.

^gExcludes 3 women from whom cervical cell specimens were not obtained.

^h Includes infections with both oncogenic and nononcogenic/undetermined risk types.

ⁱ Excludes 2 women from whom anal cell specimens were not obtained and 21 with insufficient cell specimens.

^j Excludes 3 women from whom cervical cell specimens were not obtained, 2 women from whom anal cell specimens were not obtained, and 21 with insufficient anal cell specimens.

^k Excludes 2 women from whom cervical cytology specimens were not obtained and 8 with insufficient Pap specimens.

^l Excludes women with uncollected or insufficient cervical and/or Pap specimens

^m Excludes women with uncollected or insufficient anal specimens.

Table 2
Relationship of Cervical and Anal HPV in American Samoa With Selected Characteristics

Characteristic ^a	Cervical HPV DNA ^b			Anal HPV DNA ^b		
	+	-	Age-Adjusted OR (95% CI) ^c	+	-	Age-Adjusted OR (95% CI) ^c
Cervical HPV DNA						
Negative	—	—	—	23	146	1.00
Positive				7	11	3.32 (1.10–10.00)
Demographic factors						
Age (years)						
30	6	142	1.00	17	120	1.00
<30	15	45	7.87 (2.89–21.74)	13	38	2.42 (1.08–5.44)
Marital status						
Ever married	8	148	1.00	20	125	1.00
Single/never married	13	32	3.82 (1.26–11.56)	8	31	0.96 (0.34–2.72)
Education level						
High school graduate	5	102	1.00	11	86	1.00
>High school	16	81	4.58 (1.52–13.74)	18	70	1.95 (0.86–4.43)
Traditional lifestyle indicators						
Hot, running water in home						
No	10	126	1.00	16	109	1.00
Yes	10	33	3.75 (1.36–10.39)	8	30	1.80 (0.69–4.69)
Sexual and reproductive factors						
Pregnant ^d						
Never	9	21	1.00	4	22	1.00
Ever	10	127	0.27 (0.09–0.80)	22	102	1.43 (0.43–4.72)
Lifetime no. of male partners ^e						
<5	14	177	1.00	27	147	1.00
5	7	10	9.29 (2.78–31.00)	3	11	1.34 (0.35–5.19)
Condoms ^{d,f}						
Never	11	84	1.00	17	70	1.00

Characteristic ^a	Cervical HPV DNA ^b			Anal HPV DNA ^b		
	+	-	Age-Adjusted OR (95% CI) ^c	+	-	Age-Adjusted OR (95% CI) ^c
Ever	8	19	3.24 (1.06–9.86)	8	16	1.96 (0.72–5.38)
Natural family planning ^{d,f}						
Never	11	90	1.00	20	71	1.00
Ever	6	12	8.27 (1.98–34.66)	4	13	1.14 (0.33–3.93)
Birth control pill ^{d,f}						
Never	10	76	1.00	16	62	1.00
Ever	9	33	3.03 (1.01–9.10)	8	30	1.14 (0.33–3.93)
Withdrawal prior to ejaculation ^{d,f}						
Never	10	86	1.00	20	69	1.00
Ever	6	15	3.50 (1.01–12.08)	3	14	0.70 (0.18–2.70)
Morning after pill ^{d,f}						
Never	12	97	1.00	22	77	1.00
Ever	5	3	7.37 (1.51–35.99)	1	6	0.45 (0.05–4.14)

Abbreviations: HPV, human papillomavirus; OR, odds ratio; CI, confidence interval.

^aFrequencies exclude those with missing responses.

^bCervical HPV results exclude 3 individuals with no cervical specimen; anal HPV results exclude 2 individuals with missing anal specimens and 21 β -globin negative samples.

^cORs and 95% CIs derived through unconditional logistic regression.

^dMissing data from 20% or more of total subjects because of incomplete interviews or nonresponse.

^eNumber of male vaginal intercourse partners.

^fExcludes individuals with no history of vaginal sex.