Published in final edited form as:

Sex Transm Dis. 2014 August ; 41(8): 486–492. doi:10.1097/OLQ.0000000000000159.

Sexual Behaviors and Other Risk Factors for Oral Human Papillomavirus Infections in Young Women

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Abstract

Background—Oral human papillomavirus (HPV) is associated with a rising incidence of certain head and neck cancers, and oral sex has been associated with oral HPV. This study sought to identify more specific patterns of oral sexual activity, including self-inoculation, that are associated with oral HPV infections in young women.

Methods—A total of 1010 women attending a large university completed a computer-based questionnaire and provided oral specimens that were tested for any oral HPV using a Linear Array assay that detects any HPV as well as 37 HPV genotypes. Twenty-seven women provided additional samples up to 12 months after enrollment. Bivariable and multivariable analyses were conducted to identify oral sexual patterns and other risk factors associated with prevalent oral HPV.

Results—Nineteen women had prevalent oral HPV (1.9%), with 10 women (1%) having a type-specific infection. Oral HPV was significantly associated with lifetime coital sex partnership numbers ($P = 0.03$), lifetime and yearly oral sex partnership numbers ($P < 0.01$), and hand and/or sex toy transfer from genitals to mouth ($P < 0.001$). Oral HPV was also associated with greater use of alcohol, cigarettes, marijuana, and sharing of smoking devices, lipstick, or toothbrushes ($P < 0.05$ for each), with an apparent dose-response for alcohol use and smoking behavior, stratified by number of sexual partners. Of 7 women with prevalent HPV who provided follow-up samples, none had evidence of a persistent type-specific infection.

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Conflicts of interest: No other perceived conflicts of interests are declared.

Conclusions—These data provide additional evidence of transmission of oral HPV from oral sexual activity and also suggest possible transmission from self-inoculation or sharing of oral products.

Human papillomavirus (HPV) is the cause of a subset of head and neck squamous cell cancers and oropharyngeal squamous cell carcinomas.¹ It has more than 100 known types; however, nearly 90% of all HPV-associated cancers of the head, neck, and oropharynx are linked to HPV type 16.^{2,3} Although incidence of oropharyngeal cancer has changed little from 1987 to 2007,⁴ the percentage of oropharyngeal tumors associated with HPV has risen from 16.3% in 1984–1989 to 71.7% in 2000–2004 in the United States.^{5,6} This increase of HPV-related oral cancers necessitates a better understanding of the risk factors related to oral HPV acquisition and persistence. The few studies that have examined oral HPV in healthy young women report relatively low prevalence (0.2%–4.6%),^{7–9} making investigation of specific risk factors challenging. Thus, relatively little is known about the specific, independent risk factors associated with oral HPV in this population.

Oral sexual behaviors such as lifetime number of oral sexual partners^{9–11} and open-mouth kissing^{9,10} are the most consistently identified risk factors for oral HPV, although other studies have identified the number of vaginal sexual partners¹⁰ and smoking^{2,8,10,12} to be significantly associated with oral HPV. Because of a significant overlap in drinking, smoking, and sexual risk behavior, it has been difficult to determine the independent association of sexual risk factors,^{8,9} or whether risk increases with a greater number of risk behaviors. Moreover, very little research has focused on specific oral sexual behavior and nontraditional oral sex, such as hand or sex toy transfer from the vagina to the mouth, which may increase the chance self-inoculation of oral HPV. Other factors that we hypothesized might be associated with oral HPV infections included sharing of personal hygiene objects, personal or family health history, history of HPV vaccination, use of hormonal contraceptives, and other oropharyngeal lesions or trauma.

The study objectives were to determine the independent association of various sexual and substance use behaviors as risk factors for oral HPV and to determine the persistence of oral HPV infections within 1 year of follow-up.

METHODS

The HPV Oral Prevalence Investigation study sample consisted of women recruited in or around the Student Health Care Center at a large public university located in the southeastern United States between April and September 2011. The sample included women seeking services at the center, as well as women passing by, referred by friends, or responding to brochures or online advertisements. All women 18 years or older and currently enrolled at the university were eligible. All participants provided written informed consent and received a \$20 incentive. The study procedures were approved by institutional review boards at the University of Florida and the Moffitt Cancer Research Center.

Data Collection

At the baseline visit, oral HPV samples were obtained using similar procedures to other recent studies.² Participants were asked to energetically swish 15 mL of Scope mouthwash in their mouth for 15 seconds, gargle for an additional 15 seconds, and then spit the mouthwash into a sterile 50-mL falcon tube. Samples were refrigerated and processed within 24 hours, and then shipped to the Moffitt Cancer Center. Samples were tested for β -globin to confirm oral sample collection, and samples without β -globin were excluded from analysis ($n = 20$). All other specimens received DNA extraction (QIAgen Media Kit), and HPV testing by the Linear Array HPV genotyping assay (Roche Diagnostics). Samples were amplified as directed by the Linear Array protocol, and polymerase chain reaction (PCR) products were analyzed by agarose gel electrophoresis using 2% agarose gels to visualize a 450-base-pair band corresponding to HPV amplification before genotyping. The purpose of this pre-Linear Array step is to identify samples that may harbor HPV infections other than the 37 HPV types included in the genotyping assay. Those who tested positive for any oral HPV, regardless of type, were considered positive for oral HPV. Then, these were tested by Linear Array to identify whether each sample included 1 of 37 specific HPV types included in the assay, including both high-risk (oncogenic) and low-risk types.

Participants also completed a 20-minute questionnaire (available as an online appendix or upon request from the study authors). Responses were entered directly by participants using a netbook computer, with responses transferred via an encrypted wireless signal directly to an online survey database ([surveymonkey.com](https://www.surveymonkey.com) professional version).

Sexual behavior items included the total number of partners (lifetime and past year) for open-mouth kissing, coital sex, and specific oral sexual behaviors (placing the mouth directly on a partner's penis, scrotum, vagina, and anus). After examining the overlap in specific types of oral sexual behavior involving men (penile, scrotal, anal), we combined these as any oral sexual behavior because nearly all who reported oral-scrotal or oral-anal sex also reported oral-penile. We defined overall sexual risk according to the number of lifetime and past-year oral or coital sexual partners (none, 1–2, 3–5, or 6+).

Oral sex behaviors that could increase risk of self-inoculation were assessed by asking “has a hand (yours/someone else's) ever touched your genitals then put the hand or fingers in your mouth?” and “have you ever put your mouth on a sex toy after it touched your or someone else's genitals?” Those who answered “yes” to either item were coded as having ever engaged in potential self-inoculation behaviors.

Alcohol consumption items assessed current (past 3 months) frequency of any drinking, frequency of binge drinking (drinks in 1 occasion), and sharing of cups of alcoholic beverages. The frequency of smoking different products (tobacco, marijuana, hookah, cigars, smokeless tobacco) and sharing of smoking devices were assessed. We created a composite 4-category variable for “any smoking,” based on the highest frequency reported for any type of smoking.

Additional questionnaire measures included demographic characteristics (age, race, ethnicity), reproductive health issues, HPV vaccination status, family history of head and

neck cancers, recent antibiotic use, and oral health history (sharing of personal hygiene products, sores or blisters in the mouth, braces, and history of tonsillectomy, heartburn, or acid reflux).

Follow-Up

All participants with a positive type-specific HPV result ($n = 10$) were invited to complete follow-up assessments at 3, 6, and 12 months after enrollment. For each of these participants, we also invited 2 control subjects, selected at random from other students enrolled on the same day (or close to the same day). At each follow-up, these women were asked to provide an additional oral sample for oral HPV testing.

Data Analysis

The primary outcome was any positive result for oral HPV by the PCR assay ($n = 19$). Bivariate analyses were conducted to compare characteristics of persons with and without oral HPV, and statistical significance was assessed using a 2-tailed t test, χ^2 test, or Fisher exact test. Alcohol use and smoking behavior were evaluated for a dose-response relationship with oral HPV, stratified by number of past-year penile/vaginal sex partners. For sexual partner numbers and frequency of alcohol and smoking, we evaluated potential dose-response relationships using the Cochran-Armitage test for trend.

We conducted multivariable logistic regression to assess the independent association of specific types of oral sexual behavior with oral HPV. Because of multicollinearity between specific oral sexual behaviors, analyses assessed risk for oral HPV separately for each oral sexual behavior, while controlling for smoking and alcohol use. Dichotomous measures of lifetime kissing partners (< 5 partners, ≥ 5), oral-penile partners (< 5 partners, ≥ 5), oral-vaginal sex (ever, never), self-inoculation behavior (ever, never), weekly alcohol consumption (yes, no), and weekly smoking (yes, no) were used.

Finally, we created a risk index variable that added the number of risks for each individual woman (weekly alcohol, weekly smoking, and > 2 past-year sex partners), to create a score of 0 to 3, to further assess whether having multiple risk factors was associated with corresponding increased risk of oral HPV. All data analyses were conducted using SAS version 9.3 (Cary, NC).

RESULTS

Of 1030 women who enrolled, 20 provided an invalid oral sample (β -globin negative) and were excluded, leaving a final sample of 1010 women. Nineteen had any oral HPV detected by PCR (1.9%), of whom 10 had a specific HPV type identified: HPV-16 (2), HPV-59 (3), HPV-84 (2), and 1 each with HPV-51, HPV-62, and HPV-73. The mean age of the sample was 21.9 years (range, 18–54 years), with 73% being 22 years or younger. This sample was racially diverse: 51% of participants were non-Hispanic white; 15%, non-Hispanic black; 14%, Hispanic; 10%, Asian; and 10%, other race. Neither age nor race/ethnicity was significantly associated with oral HPV in this sample.

Table 1 presents the bivariate relationships of various sexual risk behaviors and oral HPV. Most participants reported ever kissing a partner (93%), performing oral sex (77%), or having coital sex (75%). Past-year oral sexual behavior included oral penile (67%) and oral vaginal (2.6%). There was evidence of a dose-response relationship for lifetime and past-year number of open-mouth kissing, oral sex, and coital sex partners (Fig. 1). Self-inoculation behavior was also associated with increased risk of oral HPV, whereas condom use was not.

The bivariate associations of oral HPV to other health conditions and behaviors are shown in Table 2. Most participants (58%) reported drinking any alcohol in the last month, with 17% having 5 or more drinks on at least 1 occasion. Of those consuming any alcohol, 60% reported sharing a drinking cup during the last month. The frequency of any drinking and binge drinking was significantly associated with oral HPV (Table 2), with evidence of a dose-response relationship (Fig. 2), regardless of the number of past-year sex partners. Smoking was less common; with past-month smoking reported by 4% for cigarettes, 2% for hookah, and 7% for marijuana. Almost all (95%) who reported any smoking in the past year shared a smoking device at least sometimes. Oral HPV infections were significantly associated with smoking cigarettes, smoking marijuana, and sharing of smoking devices (Table 2), with evidence of a dose-response relationship between oral HPV and any smoking, stratified by number of past-year penile/vaginal sex partners (Fig. 2). Overall, oral HPV risk increased according to the number of major risk factors (sexual partners, alcohol, smoking), with oral HPV prevalence of 1.0%, 1.3%, 7.6%, and 8.3%, in women with 0 to 3 risk factors, respectively (Cochran-Armitage exact trend test, $P < 0.001$).

Sharing lipstick or a toothbrush in the past month (reported by 20%) was significantly associated with oral HPV infections. Recent antibiotic use, personal or family-history of HPV-related disease, sores in mouth, or having braces were not significantly associated with oral HPV. None of the 101 women with a history of tonsillectomy and none of the 72 with a history of acid reflux had prevalent oral HPV, although these results were not statistically significant. Of note, none of the 50% of women who had received any HPV vaccination had prevalent oral HPV types covered by HPV vaccines (type 16, 18, 6, or 11), although there were only 2 HPV-16 infections detected in the sample (Table 2).

Multivariable Analyses

When controlling for smoking and alcohol use, having 6+ oral-penile partners (odds ratio [OR], 2.79; 95% confidence interval [CI], 1.01–7.73; $P < 0.05$) and engaging in self-inoculation behavior (OR, 3.7; 95% CI, 1.4–9.7; $P < 0.01$) were significantly associated with increased risk for prevalent oral HPV (Table 3). Having 6+ kissing partners (OR, 2.24; 95% CI, 0.75–6.73; $P = 0.15$) and ever-performing oral-vaginal sex (OR, 2.0; 95% CI, 0.34–7.85; $P = 0.49$) also were associated with increased odds of oral HPV but were not statistically significantly associated with oral HPV (Table 3). Self-inoculation behavior continued to be associated with a statistically significant increased risk of oral HPV, even when also controlling for any one of the other oral-sexual behaviors.

Longitudinal Follow-Up

Twenty-three of 30 participants invited for follow-up had at least 1 follow-up assessment for oral HPV at 3 months (n = 10), 6 months (n = 21), and 12 months (n = 11). None of the 7 women with baseline oral HPV had type-specific persistence at any follow-up: 5 were never positive again and the remaining 2 were positive at month 6 with HPV types different from those present at enrollment. Of the 16 control subjects, 13 (81%) had no positive results and 3 (19%) tested positive for oral HPV at a subsequent visit.

DISCUSSION

The overall prevalence of oral HPV was 1.9% in this sample of 1010 college-enrolled women. This prevalence is consistent with, or slightly lower than, the overall prevalence of oral HPV in a recent national sample.⁸ Although the prevalence is low, we found several health behaviors to be significantly associated with prevalent HPV infections, which collectively suggest several possible methods of direct transfer of the virus from one person to another or from one anatomic site to another. Alcohol, smoking, and sexual behavior each seemed to be independently associated with risk, as shown by evidence of dose-response relationships, persistent associations within strata of the other risk factors, and the fact that having 2 or more of these risk factors was associated with a substantial increase in risk compared with having only 1 (or no) risk factor.

In our study, greater numbers of lifetime and past-year sexual partners were associated with an increased risk of oral HPV infections, with similar relationships for deep kissing, oral-genital, and coital sexual exposures. Therefore, we were not able to determine whether one specific type of sexual activity was more strongly associated with risk than others. Other studies have reported associations with either vaginal sexual behavior,¹⁰ oral sexual behavior^{9,10} or both,¹⁰ and deep kissing has been associated with oral HPV infections in young men^{9,10} and young women.⁹ Therefore, the data collectively suggest that oral HPV is transmitted sexually, although it should be noted that a few women in our sample had oral HPV but reported no history of oral or vaginal sexual behavior.

Our data also suggest that self-inoculation (hand and/or sex toy transfer from genitals to mouth) is a possible mechanism by which genital HPV could be transferred to the oral cavity of the same individual. One recent study indicated that women with genital HPV had 8.5 times the odds of oral HPV.¹³ Furthermore, another study found a concordance rate between female genital HPV and her male partner's hand to be as high as 60%.¹⁴ Our study is the first to report a significant association between self-inoculation behaviors and oral HPV infection, although we did not have access to genital samples from participants to confirm type-specific concordance in the oral and genital tract.

Our study also provides evidence that alcohol consumption and smoking are each independent risk factors for oral HPV, which is significant because both alcohol consumption and smoking are associated with higher rates of oropharyngeal and other head and neck cancers.^{15–19} Although previous studies have not reported a significant association of alcohol consumption on oral HPV, several previous studies have found an association between smoking and oral HPV.^{2,8,10,12} Alcohol and smoking could be associated with oral

HPV due to sharing of cups or smoking devices, to a local effect that affects host susceptibility, or to associations with risky sexual behavior or risky sexual partners. We found that the patterns of alcohol and tobacco-related risks were similar in women with low numbers and with high numbers of sexual partners, which is consistent with an association of alcohol and smoking behavior that is independent from sexual activity.

We also found that women who reported sharing of toothbrushes, lipsticks, or other similar items were significantly more likely to have oral HPV infection, which suggests that oral HPV could be transmitted by the sharing of these types of items. Also of interest, none of the women with a history of tonsillectomy or who reported current acid reflux symptoms had oral HPV infections, although these results were not statistically significant. Also, none of the women who had received at least 1 previous HPV vaccination had oral HPV types that are included in currently available HPV vaccinations (HPV-16 and HPV-18).²⁰ It is of note that only 2 oral HPV infections in our sample were either type 16 or 18, and thus, this association requires further examination.

The study has several strengths. The sample was racially and demographically diverse, which enhances generalizability to women in this age range. In addition, the study used the validated methods of HPV testing as used in other studies.^{2,8} The questionnaire was conducted on netbook computers to encourage participants to reduce the possibility of social desirability bias.

Several potential limitations should be mentioned. Because those with any oral HPV detection were considered positive for HPV, we could not identify risk factors for specific types of HPV infection, particularly the alpha types that have been linked to oropharyngeal cancers. However, it is likely that the risk factors of any HPV identified in the current study are significant risk factors for the HPV alpha types, as transmission risk by HPV type is not known to be differential. Our sample attended a single university, although the proportion of women ever-engaging in oral sex (78%) was similar to another self-report study among women aged 18 to 25 years.²¹ The low number of HPV positives in the study limited the ability to conduct multivariable analyses using more than 3 variables, and the cross-sectional nature of the study limited our ability to demonstrate temporal associations. Likewise, with limited numbers of HPV infections, a significance test for the dose-response for each alcohol use and smoking behavior, stratified by number of past-year sex partners, was not possible.

Comparison of oral HPV prevalence across different populations and publications can be challenging because the definition of oral HPV can vary according to the specific diagnostic test used. It is also possible that testing could miss some oral HPV if the oral HPV is latent,²² is present in low viral copy number, or resolves spontaneously.²³ To address this issue, we need additional research that includes more frequent sampling, or studies that look for oral HPV in the deep oral mucosa or tonsils.

In conclusion, we found that oral HPV are relatively uncommon in college-enrolled women, despite the high prevalence of risk behaviors associated with oral HPV. Although the information presented in this article can help us understand how HPV viruses may be transferred, the overall risk of infection was low. Human papillomavirus type 16, which is

the most significant type in terms of head and neck cancer, was present in only 2 of the 1010 women. We found that alcohol, smoking, and sexual behaviors were each independently associated with increased risk, and that the combination of 2 or more of these risk behaviors was associated with especially high risk. It remains unproven whether prevalent oral HPV infections in young women will result in the development of cancer many years in the future, or whether screening and intervention strategies can prevent the development of HPV-related oropharyngeal cancers.

Acknowledgments

The HPV Oral Prevalence Investigation study would like to recognize the support of the staff at the University of Florida Student Health Services (Toni Ratliff, Terri Wilkinson, Zenon, Bob, Catherine Seemann), public health students who participated in recruitment and study planning (Shandrika Gordon, Angela DeQuesada, Jason Poling), and Dan'elle C. Smith and the laboratory team at Moffitt.

Funding: This work was supported in part by a research grant from the Investigator-Initiated Studies Program of Merck Sharp & Dohme Corp, a subsidiary of Merck & Co, Inc. Funding was provided to the University of Florida with no specific restrictions. The opinions expressed are those of the authors and do not necessarily represent those of Merck Sharp & Dohme Corp. No recommendations for or against any product provided by Merck are made. Dr Barkley is currently a Merck Employee, but his work on this study and manuscript was completed before being hired by Merck.

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Figure 1.
Number of sexual partners (oral or vaginal) in the past year and prevalence of oral HPV infections in 1010 college women.

Figure 2.
Prevalence of oral HPV infections according to frequency of alcohol and smoking, stratified by the number of past-year penile/vaginal sexual partners in 1010 college women.

TABLE 1

Associations of Sexual Behaviors and Oral HPV Infection in Young Women (n = 1010)

Characteristic	Total, n (%) [*]	HPV Positive, n (%) [†]	P
Sexual activities			
Kissing (open mouth) partners—lifetime			
(0)	71 (7.1)	1 (1.4)	0.2
(1–2)	147 (14.7)	1 (0.7)	
(3–5)	214 (21.4)	3 (1.4)	
(6+)	569 (56.8)	14 (2.5)	
Kissing (open mouth) partners—past year			
(0)	127 (12.6)	2 (1.6)	0.02
(1–2)	560 (55.6)	7 (1.2)	
(3–5)	190 (18.9)	3 (1.6)	
(6+)	130 (12.9)	7 (5.4)	
Oral sex partners—lifetime			
(0)	230 (22.8)	2 (0.9)	0.004
(1–2)	371 (36.8)	5 (1.3)	
(3–5)	267 (26.5)	4 (1.5)	
(6+)	140 (13.9)	8 (5.7)	
Oral sex partners—past year			
(0)	335 (33.2)	3 (0.9)	0.005
(1–2)	565 (56.1)	10 (1.8)	
(3–5)	97 (9.6)	5 (5.1)	
(6+)	11 (1.1)	1 (9.0)	
Penile/Vaginal sex partners—lifetime			
(0)	246 (24.7)	3 (1.2)	0.03
(1–2)	315 (31.7)	3 (1.0)	
(3–5)	221 (22.2)	5 (2.3)	
(6+)	212 (21.4)	8 (3.8)	
Penile/Vaginal sex partners—past year			
(0)	284 (28.6)	4 (1.4)	0.09
(1–2)	571 (57.4)	10 (1.8)	
(3–5)	116 (11.7)	3 (2.6)	
(6+)	23 (2.3)	2 (8.7)	
Hand from genitals to mouth			
Never/Less than monthly	860 (89.3)	10 (1.2)	<0.0001
Monthly	103 (10.7)	9 (8.7)	
Sex toy from genitals to own mouth			
Never	919 (92.5)	15 (1.6)	0.049
Ever	75 (7.5)	4 (5.3)	
Condom use (past year)			
N/A (not sex active in past year)	247 (24.5)	3 (1.2)	0.8

Characteristic	Total, n (%) [*]	HPV Positive, n (%) [†]	<i>P</i>
No	395 (39.1)	9 (2.3)	
Yes	367 (36.4)	7 (1.9)	

^{*} Total for each category may not add up to 1010 due to missing data.

[†] Percent HPV positive within predictor strata.

TABLE 2

Association of Substance Use Behavior, Health Conditions, and Oral Health on Oral HPV Infections Oral Health Issues in Young Women (n = 1010)

Characteristic	Total, n (%) [*]	HPV Positive, n (%) [†]	P
Alcohol use in the past 3 mo			
None	240 (23.8)	3 (1.3)	<0.0001
Weekly or less	528 (52.4)	6 (1.1)	
2–3 times a week	211 (21.0)	6 (2.8)	
4+ times a week	28 (2.8)	4 (14.3)	
Drink 5 drinks per occasion at least weekly			
No	939 (95.1)	15 (1.6)	0.01
Yes	48 (4.9)	4 (8.3)	
Share drinking cups—at least weekly			
No	587 (76.7)	11 (1.9)	0.5
Yes	178 (23.3)	5 (2.8)	
Smoking behavior in the past 3 mo			
None	792 (78.4)	9 (1.1)	<0.0004
Weekly or less	151 (15.0)	5 (3.3)	
2–3 times a week	25 (2.5)	1 (4.0)	
4+ times a week	42 (4.2)	4 (9.5)	
Smoking cigarettes—at least weekly			
No	954 (95.2)	15 (1.6)	0.01
Yes	48 (4.8)	4 (8.3)	
Smoking marijuana—at least weekly			
No	963 (96.1)	16 (1.7)	0.03
Yes	39 (3.9)	3 (7.7)	
Share smoking device (ever)			
No	802 (79.4)	10 (1.2)	0.008
Yes	208 (20.6)	9 (4.3)	
History of HPV-related disease [‡]			
No	830 (86.2)	17 (2.0)	0.5
Yes	133 (13.8)	1 (0.8)	
HPV vaccination (ever)			
None	475 (50.9)	11 (2.3)	0.3
1	459 (49.1)	6 (1.3)	
Family history head/neck cancer			
No	795 (93.4)	16 (2.0)	1.0
Yes	56 (6.6)	1 (1.8)	
Recent antibiotic use (past month)			
No	852 (84.6)	14 (1.6)	0.2
Yes	155 (15.4)	5 (3.2)	
Share lipstick or toothbrush (past month)			

Characteristic	Total, n (%) [*]	HPV Positive, n (%) [†]	<i>P</i>
No	808 (80.1)	11 (1.4)	0.04
Yes	201 (19.9)	8 (4.0)	
Sores or blisters in the mouth (past month)			1.0
No	916 (91.1)	18 (2.0)	
Yes	90 (8.9)	1 (1.1)	
Braces—current			0.8
No	833 (82.7)	17 (2.0)	
Yes	174 (17.3)	2 (1.1)	
Past tonsillectomy			0.2
No	908 (90)	19 (2.1)	
Yes	101 (10)	0 (0.0)	
Heartburn or acid reflux (past month)			0.4
No	935 (92.9)	19 (2.0)	
Yes	72 (7.1)	0 (0.0)	

^{*} Total for each category may not add up to 1010 due to missing data.

[†] Percent HPV positive within predictor strata.

[‡] HPV-related disease included history of abnormal Papanicolaou test result, colposcopy, or genital warts.

TABLE 3

Multivariable Association of Different Types of Sexual Behavior on Oral HPV Infections in Young Women (n = 1010), Controlling for Alcohol Use and Smoking Behavior

Oral Sexual Behavior	OR (95% CIs)*					
Weekly alcohol use (reference < weekly use)	2.37 (0.86–6.53)	2.22 (0.81–6.05)	2.92 (1.0–8.58) [†]	2.17 (0.79–5.99)	2.11 (0.77–5.74)	2.49 (0.82–8.91)
Weekly smoking behavior (reference < weekly use)	3.17 (1.02–9.82) [†]	3.07 (1.01–9.32) [†]	3.21 (0.80–10.9)	2.44 (0.76–7.77)	2.51 (0.81–7.79)	2.49 (0.59–8.91)
6+ Lifetime kissing partners (reference < 6 partners)	2.24 (0.75–6.73)			1.90 (0.63–5.71)		
6+ Lifetime oral-penile partners (reference < 6 partners)		2.79 (1.01–7.73) [†]			2.17 (0.78–6.07)	
Lifetime oral-vaginal (reference = never)			2.00 (0.34–7.84)			1.38 (0.22–5.89)
Self-inoculation behavior (reference = never)				3.43 (1.30–9.04) [†]	3.09 (1.16–8.26) [†]	3.46 (1.12–10.4) [†]

* The first 4 models assess the association of different sexual behaviors, controlling for alcohol use and smoking behavior; The last 3 models assess the association of self-inoculation behavior and oral HPV, controlling for alcohol use, smoking behavior, and any one of the traditional sexual behaviors.

[†] $P < 0.05$.

[‡] $P < 0.01$.