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Prevalence of and risk factors for anal human papillomavirus in men having sex with women: A cross–national study

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Abstract

Background—While the primary cause of anal cancer is human papillomavirus (HPV) infection in the anal canal, little attention has been paid to the epidemiology of anal HPV in men having sex with women (MSW).

Methods—Anal canal exfoliated cells from 903 MSW in Brazil (São Paulo), Mexico (Cuernavaca) and the United States (Tampa) were tested for HPV DNA.

Results—HPV prevalence in the anal canal (12.0%) was similar in MSW in each city (P=0.77) while 7.0% had oncogenic types. Men in Tampa had a four–fold higher prevalence of HPV 16 than men in São Paulo or Cuernavaca (P<0.001). Duration of relationship with a primary sex partner and ever having oral or anal sex with a man was associated with any HPV type and any oncogenic type while lifetime number of female sex partners was associated with any HPV type.

Conclusions—Anal canal HPV is commonly found in MSW and the prevalence of HPV 16 may differ substantially by geography. Men with a larger number of female sex partners, in a sexual relationship of <1 year duration, and with a history of oral or anal sex with men were most likely to have an anal HPV infection.

Keywords

Human papillomavirus; prevalence; anus; sexual behavior; men; internationality

Human papillomavirus (HPV) is responsible for condylomas and malignancies at a variety of anatomical sites including the cervix, vagina, vulva, penis, anus, and oropharynx. While the incidence of cervical cancer has been decreasing in western countries [1], epidemiologic studies have highlighted anal cancer for its increasing incidence in both men and women [2–4]. The primary cause of anal canal cancer is infection with oncogenic HPV, usually HPV 16 [5–7].

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Anal cancer incidence in United States (US) men has increased several fold since 1973 [8]; however, anal HPV prevalence and risk factors in men having sex with women (MSW) have rarely been reported. To date, only one study has reported prevalence estimates and risk factors for anal HPV in MSW recruited from the general community. In this previously reported study we estimated anal canal HPV prevalence and risk factors in a sample of 222 HIV–negative MSW recruited in the US [9]. The purpose of the present study was to assess anal canal HPV prevalence and risk factors in a larger sample of HIV–negative MSW recruited in Brazil, Mexico, and the US

SUBJECTS, MATERIALS AND METHODS

Study design and questionnaire

Men were recruited in Brazil (São Paulo), Mexico (Cuernavaca), and the US (Tampa) beginning in July, 2005 for the *HPV in Men (HIM) Study*– a cohort study of the natural history of anogenital HPV. Men were enrolled if they were between the ages of 18 and 70 years; resided in the targeted recruitment areas; had no prior anal cancer, penile cancer or genital warts; had no current sexually transmitted disease (STD) diagnosis including HIV; had no history of imprisonment, homelessness, or drug treatment in the prior six months; and were willing to comply with visits every six months for four years. Additional details of the study design and population have been previously described [10,11].

In Brazil, men were recruited from a large clinic in São Paulo that tests for HIV and STDs and from the general population through mass media advertisements. In Mexico, men were recruited in Cuernavaca and the state of Morelos through a large health plan, from factories, and the military. In the US, men were recruited from a large university campus and the general community in Tampa. Participants received a nominal monetary incentive or travel voucher for their participation. All enrolled participants consented to the study protocol which was approved by the human subjects protection committees at each study site.

Procedure

Men who completed their initial *HIM Study* visit between July 2005 and February 2007 were included in the current study (n=1392). At the initial visit men consented to the study before completing an 88–item computer–assisted self–interview (CASI) that was written in the country's primary language (Portuguese, Spanish, or English). The CASI elicited information about participant demographics, substance use, sexual health history, and sexual behaviors implicated in the transmission of HPV. Then a clinician examined the participant's abdominal, genital, anal, and inguinal areas for warts and other signs of STDs before using a saline–wetted Dacron swab to collect exfoliated skin cells from the penis, scrotum and, using a separate swab, the anal canal. The anal canal swab was an optional procedure that collected exfoliated cells from between the anal os and the dentate line. The participant also provided urine and blood to test, in part, for Chlamydia trachomatis, Neisseria gonorrhea, syphilis, and Herpes simplex virus 2. To preserve DNA for HPV analyses, each swab was placed into its own standard transport medium and stored at −70°C until HPV testing was conducted.

HPV testing

All specimens were analyzed in Tampa for HPV DNA and β–globin as described previously [10,11]. Briefly, DNA was extracted using the QIAamp DNA Mini Kit (Qiagen) according to manufacturer's instructions. To identify HPV DNA, laboratory staff used the polymerase chain reaction (PCR) consensus primer system (PGMY 09/11) to amplify a fragment of the HPV L1 gene [12]. HPV genotyping was then conducted on all samples using DNA probes labeled with biotin to detect 37 HPV types: 6, 11, 16, 18, 26, 31, 33, 35, 39, 40, 42, 45, 51–

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56, 58, 59, 61, 62, 64, 66–73, 81–84, IS39, and CP6108 [13]. Accuracy and possible contamination was assessed using non–template negative controls and CaSki DNA positive controls.

Statistical analyses

Men were categorized as MSW, men having sex with men (MSM), men having sex with men and women (MSMW), and men who denied having any sex based on their answers about recent and lifetime penetrative sexual behavior with women and men. Recent sexual behavior (prior 3 months) was prioritized over lifetime sexual behavior. For example, men who acknowledged any recent sexual behavior with other men were excluded from analysis. Also, men who acknowledged more than two lifetime male anal sex partners were excluded from analysis. A total of 1189 (85.4%) participants were categorized as MSW, 59 (4.2%) as MSM, 82 (5.8%) as MSMW, and 62 (4.5%) as denying any sexual behavior. Only MSW were included in the current study.

A specimen was considered positive for any HPV genotype if it was positive for one or more of 37 HPV types. A specimen was considered positive for oncogenic HPV if any of 13 types were detected (16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, and 66 [14]) regardless of the presence of nononcogenic HPV. Similarly, a specimen was considered positive for any nononcogenic type if one or more nononcogenic types were detected regardless of the presence of oncogenic types. In contrast, specimens with only single or multiple nononcogenic HPV types were classified as having “only nononcogenic types.” By extension, specimens with only single or multiple oncogenic HPV types were classified as “only oncogenic types.” A specimen positive by PCR but without positive genotyping for any of 37 types was labeled as having “unclassified” HPV.

The Fisher exact test was used to assess anal HPV prevalence differences by city. The χ^2 and Kruskal-Wallis tests were used to determine differences in men’s characteristics and sexual behavior by city. The Cochran–Armitage test assessed for linear trends in HPV infection with ordinal participant characteristics.

Odds ratios (OR) and 95% confidence intervals (CI) were calculated by bivariate and multivariate logistic regression for three outcomes: any HPV type, any oncogenic type. and only nononcogenic types. Estimates of effect were calculated for each outcome because factors associated with infection could differ by HPV type [15,16]. Given our prior research that identified an independent association between lifetime number of female sex partners and anal HPV [9], directed acyclic graphs [17,18] were used to identify potential confounders and intermediate variables in the causal pathway between lifetime number of female sex partners and anal HPV. Intermediate variables were excluded from multivariate modeling [19]. Potential confounders were defined as common causes of lifetime number of female sex partners and anal HPV or their surrogates on confounding paths [18]. Variables with a P value of less than 0.20 on a likelihood ratio test were initially included in multivariate modeling. Independent risk factors for anal HPV were identified using a backwards–elimination logistic regression. Confounders were retained during modeling while variables with a $P>0.05$ on a likelihood ratio test were individually removed until a final set of risk factors remained. Previously rejected variables were again assessed for significance ($P<0.05$) in the final model. Data were analyzed using SAS 9.1.3 (SAS Institute Inc., Cary, North Carolina, USA).

RESULTS

Of 1189 MSW, 1010 (84.9%) agreed to anal sampling (86.6% in São Paulo; 92.1% in Cuernavaca; and 75.7% in Tampa). Men who declined were more likely to be recruited in

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Tampa (P<0.001) and aged 18 to 30 years (P<0.001). After adjusting for clinic and age, there were no statistically significant differences in anal swab acceptance by race, ethnicity, marital status, education, or lifetime number of female sex partners. After removing samples that were β–globin negative on both PCR and genotyping tests, 902 (89.3%) men remained for analysis.

Recruitment in each city enrolled participant samples that differed by several characteristics including age and sexual behavior (table 1).

HPV prevalence

Anal HPV prevalence differed little by country (P=0.77). Overall, 12.0% of *HIM Study* participants had anal canal HPV infection (table 2). Seven percent of men had detectable oncogenic HPV and 7% had nononcogenic types. Anal HPV prevalence was significantly different by city for unclassified HPV types (P=0.001), oncogenic types only (P=0.05), and some specific types, most notably HPV 16 (P<0.001). In Tampa, the prevalence of HPV 16 was more than four times higher than in São Paulo or Cuernavaca. The three most common HPV types were 16 (3.2%), CP6108 (1.4%), and 6 and 62 (1.3% for each type). In São Paulo and Cuernavaca 21/37 genotypes were detected. By comparison, in Tampa only 15/37 genotypes were detected (data not shown). Six men had warts at the perianal region or anal os and five of these were positive for anal canal HPV.

Risk factors

Because the prevalence of any HPV type, any oncogenic type, and only nononcogenic types did not differ by city, data from the three cities were combined for the remaining analyses (table 3). In bivariate analyses for any HPV type, a self–reported STD or hepatitis B diagnosis; clinician–diagnosed anogenital warts; and several sexual behavior factors were associated with anal HPV including recent (prior 3 months) and lifetime number of female sex partners, duration of relationship with a primary sex partner, and ever having had oral or anal sex with a man. For any oncogenic HPV type, statistically significant associations were observed with marital status, a self-reported hepatitis B diagnosis, clinician-diagnosed anogenital warts, duration of relationship with a primary sex partner, and ever having had oral or anal sex with a man. In bivariate analysis for only nononcogenic types, recent and lifetime number of female sex partners and age of first sexual intercourse were associated with the outcome.

In multivariate analyses, three sexual behavior factors were associated with any anal canal HPV type (table 4). Lifetime number of female sex partners (in comparison to 0–2 partners: OR, 3.35 [95% CI 1.37–8.21] for ≥10 partners), duration of relationship with a primary sex partner (in comparison to >10 years: OR, 2.78 [95% CI 1.25–6.17] for a relationship of <1 year and OR, 2.28 [95% CI 1.06–4.92] for a relationship of 5–10 years), and ever having had oral or anal sex with a man (OR, 2.09 [95% CI 1.12–3.91]) were independently associated with anal HPV. In addition, being a former smoker was inversely associated with any HPV type. In multivariate analyses for detection of any oncogenic type, duration of relationship with a primary sex partner (in comparison to >10 years: OR, 6.90 [95% CI 2.22–21.50] for a relationship of <1 year, OR, 3.54 [95% CI 1.04–12.08] for a relationship of 1–4 years, and OR, 3.36 [95% CI 1.07–10.53] for a relationship of 5–10 years) and ever having had sex with a man (OR, 2.45 [95% CI 1.14–5.27]) were independently associated with the outcome. Odds ratio point estimates increased for duration of relationship when compared with the outcome of any HPV type. No factors were independently associated with nononcogenic HPV (data not shown).

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Anal HPV 16 positive MSW were >5 times more likely to have genital HPV 16 detected (OR, 5.30 [95% CI 2.16–13.01]) (table 5).

Anal canal HPV infection prevalence was not associated with age for either any HPV type, any oncogenic type, or only nononcogenic types (figure).

DISCUSSION

This study provides corroborating evidence that HPV is a common infection of the anal canal in men having sex with women. For men recruited in three cities, we found an overall prevalence of 12.0% for any of 37 HPV genotypes while 7.0% of the men had an oncogenic HPV type. These prevalence estimates are similar to results from our previous study with 222 MSW where anal canal HPV was found in 13.1% of men and oncogenic HPV was found in 5.4% of men [9].

Although the male participants recruited in each city differed by age, marital status, and reported sexual behavior, the prevalence of oncogenic and nononcogenic groups was relatively consistent across cities. Most conspicuously, prevalence differed by city for HPV 16.

To our knowledge only three other studies have reported anal HPV prevalence estimates among MSW: our previous study of 222 US MSW where we observed a prevalence estimate of 13.1%, a study conducted among 85 MSW attending an Amsterdam STD clinic where anal HPV was detected in 1.2% of participants [20] and another study that estimated prevalence at 8% in 50 heterosexual partners of women with confirmed HPV infection in São Paulo [21]. The prevalence estimates of the current study and our prior study in the US may differ from these two prior studies because of different populations and sampling techniques; genotyping of samples regardless of PCR–based results; and our use of a newer generation PCR that identified more HPV types. In the current study, we detected 29 of 37 HPV genotypes represented in the Roche Linear Array test kit.

HPV 16, responsible for a large majority of anal malignancies in western countries [5–7,22], was the most common HPV type detected, and its prevalence was more than 4 times higher in Tampa than in the other cities. By comparison, Tampa men did not have an appreciably higher genital HPV 16 prevalence (7.2%) than men in São Paulo or Cuernavaca (8.4% and 4.0%, respectively). There could be a number of reasons for the higher anal canal prevalence in Tampa. First, since global studies indicate that prevalence of cervical HPV 16 can vary widely depending upon geography [23], this may also be the case with HPV 16 in MSW, regardless of the anatomical site. Second, the men in Tampa were younger than those in São Paulo or Cuernavaca. If younger men are more likely than older men to be immunologically naïve to HPV 16, and therefore less likely to have cleared HPV 16 infections, then it follows we may see a higher HPV 16 prevalence; however, adjusting for age did not decrease the association between city and HPV 16. Third, there were differences in the proportion of men at each site who agreed to anal sampling with a higher proportion of Tampa men declining the sampling. If these men also have less risk for HPV 16, then our Tampa sample may have been biased toward a population with higher HPV 16 prevalence. Finally, if men in Tampa, vs those in São Paulo or Cuernavaca, were more likely to conceal sexual behavior with men, then their misclassification as MSW could have led to the observed difference in HPV 16 prevalence.

Epidemiological research with HIV–negative MSM has estimated an anal HPV prevalence of 50% or higher that may be stable across the lifespan [24]. Likewise, in the current study there was no clear age-specific trend in anal canal prevalence among MSW; however, our prevalence estimate for MSW is four–fold lower than the estimated anal canal prevalence in

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HIV–negative MSM [24]. Differences in anal HPV epidemiology between MSW and MSM should be further explored in studies that recruit both groups from the same population.

An increasing lifetime number of female sex partners was strongly associated with any HPV type in these men. Also, men with new relationships (<1 year) with a primary sex partner had increased odds of anal HPV after adjusting for other covariates and confounders. In essence, anal canal HPV appears to be a common infection in MSW except in the context of men in longstanding relationships who have had few female sex partners during the lifespan.

While we are aware of no reports associating duration of relationship and anal HPV, epidemiological studies have found independent associations between the number of sex partners and anal HPV in MSW, MSM, and in women. Similar to the current study, our earlier study found that MSW with anal HPV were significantly more likely to have reported >10 lifetime female sex partners [9]. Chin–Hong, et al. reported that HIV–negative MSM with anal HPV were associated with reporting >5 male sex partners in the preceding 6 months in multivariate regression [24]. Studies of anal HPV with a clinic–based sample of women reported increased acquisition and prevalence of anal HPV in women with reports of >5 lifetime sex partners [25] and >2 lifetime sex partners [26], respectively.

In contrast to genotype–specific HPV, unclassified anal HPV had no association with sexual behavior in men in the current study (data not shown) possibly pointing to the presence of spurious PCR products or HPV types not typically sexually transmitted.

While there are a number of reports of anal canal HPV and sequelae in men and women who acknowledge no history of receptive anal intercourse [7,27–31], the mechanics of how HPV could be transmitted to the anal canal in men who are behaviorally heterosexual is unclear. One recent study provides evidence of hand transmission of HPV to the anus [32] and other studies have isolated the same HPV types on both the fingers and genitals of individuals [33,34]. If hand transmission occurs, some of the men in this study may have been infected during ano–digital sexual behavior with a partner. Alternatively, HPV might be spread by fluids, for example by vaginal secretions that flow to the man's anus during some coital positions; however, we are aware of no evidence that fluids can transmit HPV. Autoinoculation of HPV from the male genitals to the anus, or vice versa, is also possible [32]– a theory supported by the current data where men with HPV 16 at the anal canal were five times more likely to also have HPV 16 at the penis or scrotum compared to men who were anal canal HPV 16 negative.

Of 433 men who acknowledged any anal sex, a large majority (n=353) denied ever having had oral or anal sex with men while the balance (n=78) acknowledged sexual behavior with both women and men (2 were missing); however, these 78 men denied any penetrative sex with men in the prior 3 months and their sexual history and HPV status was reflective more of MSW than MSM. For example, they had a median lifetime number of female sex partners of 12 while 36 of the men acknowledged 1 lifetime male anal sex partner, 22 acknowledged 2, and 18 men reported 0 lifetime male anal sex partners. Also, these men had a prevalence of any HPV genotype of 15.4% while the MSM excluded from this study had a prevalence of 44.9%. Given that lifetime incidence of same-sex sexual behavior is common in US, Mexican, and Brazilian culture [35–37], we believe it is not surprising that men whose dominant pattern of sexual behavior is with women would also acknowledge 1 or 2 sexual experiences with men in their lifetimes; however, it is still possible that some of these 78 men could have had sex with a man as little as 4 months prior to sampling. Such a scenario may have inflated our prevalence estimate for MSW and biased the observed associations.

As with most research studying sexual behavior, our investigation used self–reported data. It is possible that some men may have concealed sexual behaviors, including same–sex sexual

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behavior; however, the CASI is considered an effective method for gathering sensitive sexual behavior data [38–41]. In addition, we have shown this method of interview to be highly reliable with men in each of the three cities [42].

There are other limitations to this study. The current cross–sectional study cannot provide data about the persistence of anal HPV infection in MSW; as such, we cannot comment on the risk for anal cancer in MSW. Also, while the recruitment strategies of the *HIM Study* enrolled a diverse group of men in each city, the voluntary nature of a longitudinal study and the commitment required may attract persons already concerned about HPV due to their own behavior or a partner's behavior. Such a situation could inflate the prevalence estimate. Alternatively, enrollment criteria that excluded men with a diagnosed STD may have attenuated our anal HPV prevalence estimate. Also, while clinicians were trained to deliver the swab directly into the anal canal, it is possible that the swab, at times, may have touched the perianal skin before entry into the anal canal; thus, our estimates of anal canal prevalence may include HPV detected at the perianal region.

This cross–national study commonly found HPV genotypes in the anal canal in MSW in three cities corroborating our prior US study with MSW. Furthermore, the presence of anal canal HPV was associated with an increased lifetime number of female sex partners, new sexual partnerships, and ever having had sex with a man. Future studies are underway that will help clarify the association between prevalent and persistent anal canal HPV infection in MSW.

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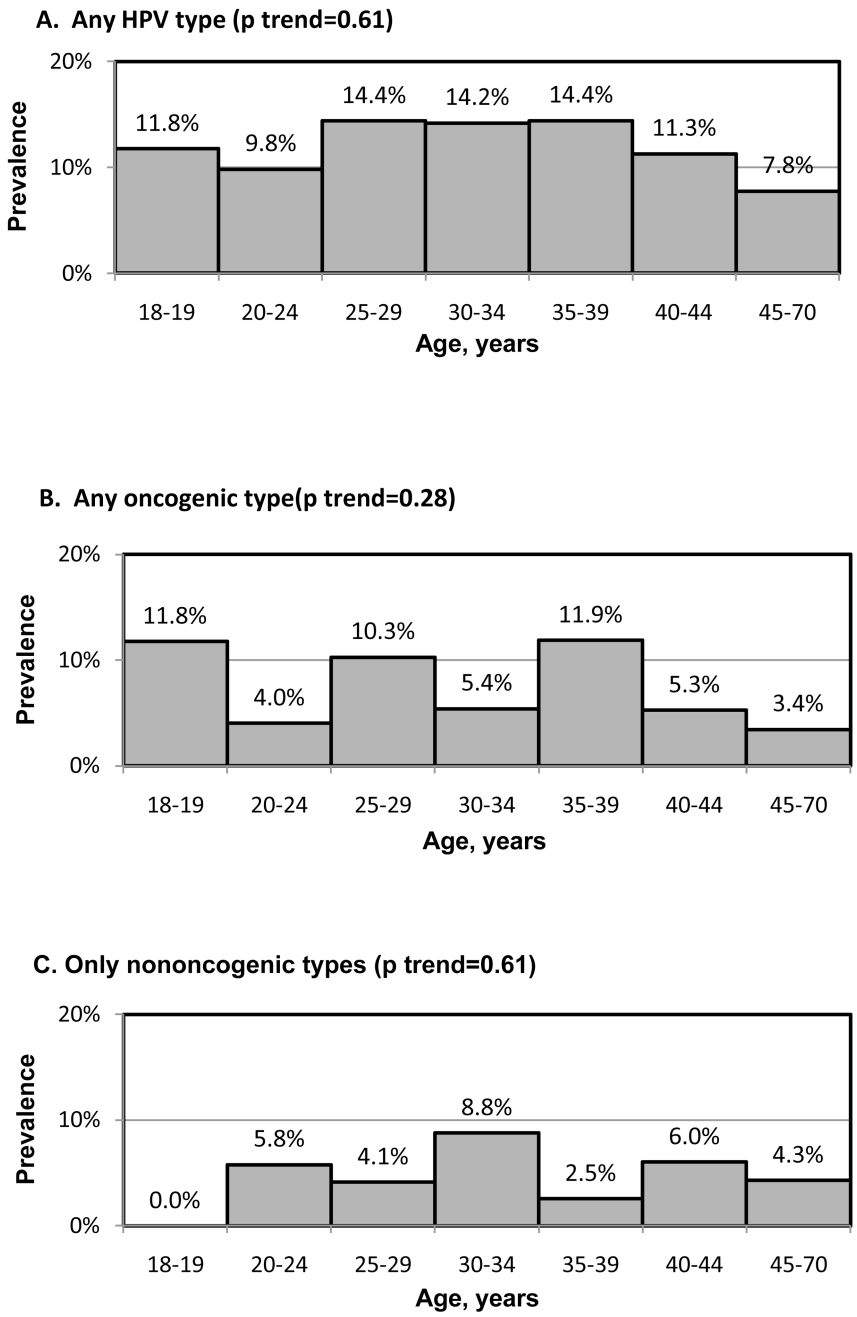

Figure.

Age–specific prevalence of human papillomavirus in the anal canal of men having sex with women (n=902): A.– Any HPV type; B.– Any oncogenic type; C. – Only nononcogenic types.

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Table 1

Selected characteristics of men having sex with women in São Paulo, Cuernavaca, and Tampa

Variable	São Paulo n=265	Cuernavaca *n*=359	Tampa *n*=278	*P*[a]
Age				
18–30 years	90 (34.0)	134 (37.3)	186 (66.9)	< 0.001
31–44 years	124 (46.8)	184 (51.3)	68 (24.5)	
45–70 years	51 (19.3)	41 (11.4)	24 (8.6)	
Race[b]				
White	152 (58.5)	5 (1.4)	201 (72.8)	< 0.001
Black	89 (34.2)	0 (0.0)	34 (12.3)	
Mixed/other	19 (7.3)	354 (98.6)	41 (14.9)	
Ethnicity[b]				
Hispanic	32 (12.3)	359(100.0)	35 (12.8)	< 0.001
Non–Hispanic	229 (87.7)	0 (0.0)	238 (87.2)	
Marital status[b]				
Single, never married	85 (32.1)	75 (21.0)	187 (67.5)	< 0.001
Married	93 (35.1)	224 (62.6)	54 (19.5)	
Cohabitating	53 (20.0)	45 (12.6)	8 (2.9)	
Divorced/separated/widowed	34 (12.8)	14 (3.9)	28 (10.1)	
Lifetime no. of female sex partners (median)	15.0	5.0	7.0	< 0.001
Ever had oral or anal sex with a man[b]				
Yes	45 (17.1)	31 (8.7)	18 (6.5)	< 0.001
No	218 (82.9)	325 (91.3)	259 (93.5)	

NOTE. Data are no. (%) of subjects.

[a] *P* values are two-sided and derived from the chi-square test with the exception of the *P* value for lifetime no. of female sex partners which is derived from the Kruskal-Wallis test.

[b] Because some observations were missing, category entries do not sum to 100%.

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Table 2

Prevalence of anal canal human papillomavirus (HPV) in men having sex with women in São Paulo, Cuernavaca, and Tampa

Variable	São Paulo n=265	Cuernavaca *n*=359	Tampa *n*=278	Combined Population *n*=902	*P*[a]
Any HPV type	29 (10.9)	43 (12.0)	36 (13.0)	108 (12.0)	0.77
Any oncogenic type[b]	13 (4.9)	26 (7.2)	24 (8.6)	63 (7.0)	0.22
Only oncogenic types	7 (2.6)	18 (5.0)	20 (7.2)	45 (5.0)	0.05
Any nononcogenic type[c]	22 (8.3)	25 (7.0)	16 (5.8)	63 (7.0)	0.50
Only nononcogenic types	16 (6.0)	17 (4.7)	12 (4.3)	45 (5.0)	0.71
Oncogenic and nononcogenic	6 (2.3)	8 (2.2)	4 (1.4)	18 (2.0)	0.76
Unclassified types only	46 (17.4)	65 (18.1)	24 (8.6)	135 (15.0)	0.001
Multiple types	11 (4.2)	9 (2.5)	7 (2.5)	27 (3.0)	0.43
Types 6 or 11	2 (0.8)	5 (1.4)	6 (2.2)	13 (1.4)	0.39
Types 16 or 18	5 (1.9)	5 (1.4)	20 (7.2)	30 (3.3)	< 0.001
Types 6, 11, 16, or 18	6 (2.3)	10 (2.8)	24 (8.6)	40 (4.4)	< 0.001
Oncogenic types[d]					
16	4 (1.5)	5 (1.4)	20 (7.2)	29 (3.2)	< 0.001
18	1 (0.4)	0 (0.0)	0 (0.0)	1 (0.1)	0.29
51	3 (1.1)	4 (1.1)	2 (0.7)	9 (1.0)	0.84
59	3 (1.1)	6 (1.7)	0 (0.0)	9 (1.0)	0.07
66	0 (0.0)	4 (1.1)	0 (0.0)	4 (0.4)	0.04
Nononcogenic types[e]					
6	2 (0.8)	4 (1.1)	6 (2.2)	12 (1.3)	0.38
11	0 (0.0)	1 (0.3)	0 (0.0)	1 (0.1)	1.00
40	0 (0.0)	0 (0.0)	3 (1.1)	3 (0.3)	0.05
53	5 (1.9)	2 (0.6)	1 (0.4)	8 (0.9)	0.19
61	1 (0.4)	4 (1.1)	0 (0.0)	5 (0.6)	0.16
62	8 (3.0)	3 (0.8)	1 (0.4)	12 (1.3)	0.02
84	2 (0.8)	4 (1.1)	2 (0.7)	8 (0.9)	0.91

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Variable	São Paulo n=265	Cuernavaca *n*=359	Tampa *n*=278	Combined Population *n*=902	*P*[a]
CP6108	8 (3.0)	5 (1.4)	0 (0.0)	13 (1.4)	< 0.01

NOTE. Data are no. (%) of subjects.

[a]Fisher exact test

[b]Prevalence of at least 1 of 13 oncogenic types, regardless of the presence of any other HPV type.

[c]Prevalence of at least 1 nononcogenic type, regardless of the presence of any other HPV type.

[d]Oncogenic types 35, 39, 45, 52, 56, and 58 are not shown; each was detected in the anal canal, but the prevalence was < 1.0%. Types 31 and 33 were not detected.

[e]Nononcogenic types 42, 55, 67, 68, 70, 71, 72, 81, 82, and 83 are not shown; each was detected in the anal canal, but the prevalence was < 1.0%. Types 26, 54, 64, 69, 73 and IS39 were not detected.

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Table 3

Factors associated with anal canal human papillomavirus (HPV) infection in men having sex with women in São Paulo, Cuernavaca, and Tampa: bivariate analyses (***n***=902).

		Any HPV type		Any oncogenic type		Only nononcogenic types	
Factor	***n***	***n* (%)**	**OR (95% CI)**	***n* (%)**	**OR (95% CI)**	***n* (%)**	**OR (95% CI)**
Age							
18–30 years	410	49 (12.0)	Reference	32 (7.8)	Reference	17 (4.2)	Reference
31–44 years	376	50 (13.3)	1.13 (0.74– 1.72)	27 (7.2)	0.91 (0.54– 1.56)	23 (6.1)	1.51 (0.79– 2.87)
45–70 years	116	9 (7.8)	0.62 (0.30– 1.30)	4 (3.5)	0.42 (0.15– 1.22)	5 (4.3)	1.04 (0.38– 2.89)
Race[a]							
White	358	44 (12.3)	Reference	25 (7.0)	Reference	19 (5.3)	Reference
Black	123	10 (8.1)	0.63 (0.31– 1.30)	5 (4.1)	0.56 (0.21– 1.51)	5 (4.1)	0.76 (0.28– 2.07)
Mixed/other	414	53 (12.8)	1.05 (0.68– 1.61)	33 (8.0)	1.15 (0.67– 1.98)	20 (4.8)	0.91 (0.48– 1.73)
Ethnicity[a]							
Hispanic	426	52 (12.2)	Reference	32 (7.5)	Reference	20 (4.7)	Reference
Non–Hispanic	467	53 (11.4)	0.92 (0.61– 1.38)	29 (6.2)	0.82 (0.48– 1.37)	24 (5.1)	1.10 (0.60– 2.02)
Marital status[a]							
Single, never married	347	46 (13.3)	Reference	32 (9.2)	Reference	14 (4.0)	Reference
Married	371	34 (9.2)	0.66 (0.41– 1.06)	19 (5.1)	0.53 (0.30– 0.96)	15 (4.0)	1.00 (0.48– 2.11)
Cohabitating	106	14 (13.2)	1.00 (0.52– 1.89)	5 (4.7)	0.49 (0.19– 1.28)	9 (8.5)	2.21 (0.93– 5.25)
Divorced/separated/widowed	76	14 (18.4)	1.48 (0.77– 2.85)	7 (9.2)	1.0 (0.42– 2.36)	7 (9.2)	2.41 (0.94– 6.20)
Prepuce present (clinician record)[a]							
Yes	553	67 (12.1)	1.04 (0.68– 1.60)	36 (6.5)	0.85 (0.50– 1.45)	31 (5.6)	1.39 (0.72– 2.69)
Partially	28	4 (14.3)	1.26 (0.42– 3.84)	3 (10.7)	1.47 (0.41– 5.21)	1 (3.6)	0.87 (0.11– 6.88)
No	317	37 (11.7)	Reference	24 (7.6)	Reference	13 (4.1)	Reference
Ever STD diagnosis[a]							
Yes	136	25 (18.4)	1.84 (1.13– 3.01)	14 (10.3)	1.66 (0.89– 3.11)	11 (8.1)	1.90 (0.93– 3.85)
No	744	81 (10.9)	Reference	48 (6.5)	Reference	33 (4.4)	Reference

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		Any HPV type		Any oncogenic type		Only nononcogenic types	
Factor	***n***	***n* (%)**	**OR (95% CI)**	***n* (%)**	**OR (95% CI)**	***n* (%)**	**OR (95% CI)**
Ever hepatitis B diagnosis[a]							
Yes	9	4 (44.4)	6.05 (1.60–22.91)	3 (33.3)	6.71 (1.64– 27.49)	1 (11.1)	2.51 (0.31–20.56)
No	865	101 (11.7)	Reference	60 (6.9)	Reference	41 (4.7)	Reference
Lifetime no. of female sex partners[a]							
0–2 partners	129	7 (5.4)	Reference	5 (3.9)	Reference	2 (1.6)	Reference
3–9 partners	365	39 (10.7)	2.08 (0.91– 4.79)	25 (6.9)	1.82 (0.68– 4.87)	14 (3.8)	2.53 (0.57–11.30)
≥ 10	359	56 (15.6)	3.22 (1.43– 7.26)	31 (8.6)	2.34 (0.89– 6.16)	25 (7.0)	4.75 (1.11–20.36)
trend[b]		*P*=0.001		*P*=0.07		*P*=0.07	
Female sex partners during preceding 3 months[a]							
0 women	248	27 (10.9)	1.16 (0.69– 1.95)	18 (7.3)	1.31 (0.69– 2.49)	9 (3.6)	0.93 (0.40– 2.13)
1 woman	409	39 (9.5)	Reference	23 (5.6)	Reference	16 (3.9)	Reference
≥ 2 women	220	39 (17.7)	2.04 (1.27– 3.30)	20 (9.1)	1.68 (0.90– 3.13)	19 (8.6)	2.32 (1.17– 4.61)
trend[b]		*P*=0.03		*P*=0.47		*P*=0.02	
New female sex partners during preceding 3 months[a]							
0 women	593	65 (11.0)	0.83 (0.51– 1.35)	40 (6.8)	1.00 (0.52– 1.92)	25 (4.2)	0.66 (0.33– 1.35)
1 woman	193	25 (13.0)	Reference	13 (6.7)	Reference	12 (6.2)	Reference
≥ 2 women	77	11 (14.3)	1.12 (0.52– 2.41)	6 (7.8)	1.17 (0.43– 3.20)	5 (6.5)	1.05 (0.36– 3.08)
trend[b]		*P*=0.30		*P*=0.79		*P*=0.21	
Ever had anal sex with a female or male[a]							
Yes	433	48 (11.1)	0.97 (0.63– 1.50)	25 (5.8)	0.77 (0.44– 1.33)	23 (5.3)	1.36 (0.71– 2.62)
No	405	46 (11.4)	Reference	30 (7.4)	Reference	16 (4.0)	Reference
Sex with someone other than a ‘steady’ partner during preceding 3 months[a]							
Yes	177	28 (15.8)	1.67 (1.02– 2.73)	14 (7.9)	1.35 (0.70– 2.59)	14 (7.9)	1.39 (0.65– 3.00)

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		Any HPV type		Any oncogenic type		Only nononcogenic types	
Factor	***n***	***n* (%)**	**OR (95% CI)**	***n* (%)**	**OR (95% CI)**	***n* (%)**	**OR (95% CI)**
No	534	54 (10.1)	Reference	32 (6.0)	Reference	22 (4.1)	Reference
No steady partner	182	26 (14.3)	1.48 (0.90– 2.45)	17 (9.3)	1.62 (0.88– 2.99)	9 (5.0)	1.79 (0.88– 3.63)
Frequency of sex with females during preceding month[a]							
0–1 times	209	26 (12.4)	Reference	14 (6.7)	Reference	12 (5.7)	Reference
2–4 times	206	23 (11.2)	0.89 (0.49– 1.61)	18 (8.7)	1.33 (0.65– 2.76)	5 (2.4)	0.41 (0.14– 1.18)
5–10 times	243	30 (12.4)	0.99 (0.57– 1.74)	16 (6.6)	0.98 (0.47– 2.06)	14 (5.8)	1.00 (0.45– 2.22)
≥ 11 times	210	26 (12.4)	1.00 (0.56– 1.78)	14 (6.7)	1.00 (0.46– 2.14)	12 (5.7)	1.00 (0.44– 2.27)
Duration of relationship with primary sex partner[a]							
No primary sex partner	183	26 (14.2)	2.27 (1.17– 4.44)	17 (9.3)	4.42 (1.60– 12.24)	9 (4.9)	1.09 (0.43– 2.75)
< 1 year	197	30 (15.2)	2.47 (1.29– 4.74)	21 (10.7)	5.16 (1.91– 13.95)	9 (4.6)	1.01 (0.40– 2.54)
1–4 years	132	15 (11.4)	1.76 (0.83– 3.73)	8 (6.1)	2.79 (0.89– 8.71)	7 (5.3)	1.18 (0.44– 3.18)
5–10 years	158	21 (13.3)	2.11 (1.05– 4.23)	11 (7.0)	3.23 (1.10– 9.50)	10 (6.3)	1.43 (0.58– 3.51)
> 10 years	221	15 (6.8)	Reference	5 (2.3)	Reference	10 (4.5)	Reference
trend[b]		*P*=0.01		*P*=0.001		*P*=0.91	
Age at first sexual intercourse with a woman, years[a]							
< 15	154	24 (15.6)	Reference	10 (6.5)	Reference	14 (9.1)	Reference
≥ 15 years	738	83 (11.3)	0.69 (0.42– 1.12)	53 (7.2)	1.11 (0.55– 2.24)	30 (4.1)	0.42 (0.22– 0.82)
Ever had oral or anal sex with a man[a]							
Yes	94	19 (20.2)	2.03 (1.17– 3.52)	12 (12.8)	2.16 (1.10– 4.21)	7 (7.5)	1.62 (0.70– 3.73)
No	802	89 (11.1)	Reference	51 (6.4)	Reference	38 (4.7)	Reference
Anogenital warts (reported by clinician)							
Yes	56	14 (25.0)	2.67 (1.40– 5.07)	9 (16.1)	2.81 (1.31– 6.03)	5 (8.9)	1.98 (0.75– 5.22)
No	846	94 (11.1)	Reference	54 (6.4)	Reference	40 (4.7)	Reference
Smoking status							

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		Any HPV type		Any oncogenic type		Only nononcogenic types	
Factor	***n***	***n* (%)**	**OR (95% CI)**	***n* (%)**	**OR (95% CI)**	***n* (%)**	**OR (95% CI)**
Never	481	64 (13.3)	Reference	36 (7.5)	Reference	28 (5.8)	Reference
Former	187	15 (8.0)	0.57 (0.32– 1.03)	8 (4.3)	0.55 (0.25– 1.21)	7 (3.7)	0.63 (0.27– 1.47)
Current	234	29 (12.4)	0.92 (0.58– 1.47)	19 (8.1)	1.09 (0.61– 1.95)	10 (4.3)	0.72 (0.35– 1.51)

NOTE. ‘Any HPV type’ cases are positive for at least one of 37 HPV types. ‘Any oncogenic type’ cases are positive for at least one of 13 oncogenic HPV types regardless of the presence of nononcogenic types. ‘Only nononcogenic types’ cases are positive only for single or multiple nononcogenic types. CI, confidence interval; OR, odds ratio; STD, sexually transmitted disease.

[a] Because some observations were missing and/or included a ‘Don’t know’ response, category entries do not sum to 100%.

[b] Cochran–Armitage test for trend.

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Table 4

Factors associated with anal human papillomavirus (HPV) infection in men having sex with women in São Paulo, Cuernavaca, and Tampa: multivariate analyses.

	Any HPV type[a]	Any Oncogenic type[b]
Factor	**OR (95% CI)**	**OR (95% CI)**
Lifetime no. of female sex partners		
0–2 partners	Reference	Reference
3–9 partners	1.89 (0.80– 4.48)	1.60 (0.58– 4.44)
≥ 10 partners	3.35 (1.37– 8.21)	2.34 (0.81– 6.75)
Duration of relationship with primary sex partner		
No primary sex partner	2.24 (0.99– 5.07)	4.53 (1.43– 14.29)
< 1 year	2.78 (1.25– 6.17)	6.90 (2.22– 21.50)
1–4 years	2.01 (0.85– 4.78)	3.54 (1.04– 12.08)
5–10 years	2.28 (1.06– 4.92)	3.36 (1.07– 10.53)
> 10 years	Reference	Reference
Ever had oral or anal sex with a man		
Yes	2.09 (1.12– 3.91)	2.45 (1.14– 5.27)
No	Reference	Reference
Smoking status		
Never	Reference	Reference[c]
Former	0.50 (0.26– 0.96)	0.66 (0.27– 1.57)
Current	0.71 (0.42– 1.21)	0.73 (0.34– 1.56)

NOTE. ‘Any HPV type’ cases are positive for at least one of 37 HPV types. ‘Any oncogenic type’ cases are positive for at least one of 13 oncogenic HPV types regardless of the presence of nononcogenic types. ‘Only nononcogenic types’ cases are positive only for single or multiple nononcogenic types. Odds ratios in each model are adjusted for confounders: no. of female sex partners during the preceding 3 months, age, and study site. CI, confidence interval; OR, odds ratio.

[a]Odds ratios adjusted by confounders and other variables in model.

[b]Odds ratios adjusted by confounders and other variables in model with the exception of smoking status which is shown only for comparison purposes.

[c]Smoking status is adjusted only by confounders.

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Table 5

Association between anal canal human papillomavirus (HPV) types and penile/scrotal HPV types in men having sex with women in the HPV in Men (HIM) Study, São Paulo, Cuernavaca, and Tampa, 2005 – 2009

		anal canal		
Penile/scrotal		**HPV+**		
HPV types	***n*[a]**	***n* (%)**	**OR**	**(95% CI)**
Any[a]	Any anal canal HPV type[a]			
yes	457	79 (17.3)	3.17	(2.01–5.02)
no	437	27 (6.2)	Reference	
Any oncogenic[b]	Any oncogenic anal canal HPV type[b]			
yes	270	29 (10.7)	2.23	(1.32- 3.76)
no	624	32 (5.1)	Reference	
Only nononcogenic[c]	Only nononcogenic anal canal HPV types[c]			
yes	187	13 (7.0)	1.58	(0.81- 3.07)
no	866	32 (4.5)	Reference	
16	Anal canal HPV type 16			
yes	56	7 (12.5)	5.30	(2.16- 13.01)
no	838	22 (2.6)	Reference	

Note. Table includes observations with a valid anal canal sample and a valid genital sample.

[a]Any of 37 HPV types

[b]Any of 13 oncogenic HPV types regardless of the presence of nononcogenic types.

[c]Detection of only single or multiple nononcogenic types.