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Race and prevalence of human papillomavirus infection among men residing in Brazil, Mexico, and the United States

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Abstract

HPV causes anal, penile and oropharyngeal cancers in men. Genital HPV prevalence in men appears to vary by world region with men residing in Asia having among the lowest prevalence. Unfortunately, there is little information on prevalence of HPV infection in men by race. The purpose of this study was to examine HPV prevalence by race across three countries. 3,909 men ages 18–70 years enrolled in an ongoing prospective cohort study of the natural history of HPV in men (The HIM Study) were included in the analysis. Participants completed risk factor questionnaires and samples were taken from the penile epithelium and scrotum for HPV detection. HPV testing of the combined DNA extract was conducted using PCR and genotyping. Asian/Pacific Islanders had the lowest HPV prevalence of 42.2% compared to Blacks (66.2%), and Whites (71.5%). The Asian/Pacific Islander race was strongly protective in univariate analysis (prevalence ratio(PR)= 0.59; 95% confidence interval(CI):0.48 – 0.74) and multivariate analysis for any HPV infection (PR= 0.65; 95% CI:0.52 – 0.8). Stratified analysis by lifetime number of female partners also showed strong inverse associations with the Asian/Pacific Islander race. We consistently observed the lowest prevalence of HPV infection among Asian/Pacific Islanders with moderate inverse associations even after various adjustments for potential confounding factors. Unmeasured behavioral factors, sexual mixing with low risk women, and/or race-specific differences in the frequency of germline variations among immune regulating genes may underlie these associations. Further studies among Asian populations that incorporate measures of immuno-genetics are needed to understand this phenomenon.

Introduction

Human papillomavirus (HPV) is a member of the papillomavirus family of DNA viruses that infect the stratified epithelium of the skin and/or mucous membrane. More than 30 types of HPV are transmitted through sexual contact but most infections do not result in disease as

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Conflicts of interest:
A.R.G interact with companies involved in HPV vaccines, however, these activities are unrelated to the content of the current manuscript. A.R.G is also a consultant to and on the Speakers Bureau of Merck and CO, Inc. L.L.V. is a consultant of Merck, Sharp and Dohme. Other authors of this manuscript have no potential conflict of interest to report.

the infections are mostly transient due to self clearance. HPV is the most common sexually transmitted infection; an estimated 6.2 million persons are newly infected every year in the United States, with mostly asymptomatic or subclinical infections.[1–10] HPV is strongly associated with increased risk of anal, penile and oropharyngeal cancers among men.[1–4] HPV is also linked to infection and subsequent cervical cancer in women. [5–8]

Results from previous studies of HPV prevalence in men have been inconsistent with estimates of prevalence ranging from 0 to 73%.[11] However, recent studies among university students and sexually transmitted disease (STD) clinic attendees in the United States showed a prevalence of 28 to 43% among men, while similar studies in male partners of women with HPV-related cervical abnormalities reported a prevalence of about 70%.[12–16] Prevalence of HPV appears to be lower in regions of Asia. Hai-Rim Shin et al. reported HPV DNA prevalence of 15.2% among Korean female students and 8.7% among Korean male students.[17] A population-based study in China also reported low HPV prevalence. Overall, HPV prevalence was 14.8% and 9.6% among women without cervical abnormalities (14.2 and 8.9%, respectively, age standardized to the world standard population) and HPV prevalence was lower among women younger than 35 years (8.7%) than those older than 35 years (17.8%).[18] Unfortunately, there are limited data on prevalence of HPV infection in men by race and country. In a previous report from our group of 1,160 US men, we observed that men of Asian race appeared to have statistically significantly lower HPV prevalence than men of other races.[19] The purpose of this study was to further examine this association of HPV infection with race by carefully controlling for and stratifying on sexual behavioral risk factors among men ages 18 to 70 years from three international cities.

Materials and Methods

Men enrolled from March 2005 through August 2009 in the ongoing HPV in Men (HIM) Study were included in this analysis. Participants were recruited from Sao Paulo, Brazil; Cuernavaca, Mexico; Tampa, United States. To encourage compliance with follow-up, men received compensation, food or transportation reimbursement for their participation. Prior to study initiation, the Human Subjects Committees of the University of South Florida, the Center for Information and Treatment on STD and AIDS, Brazil, and the National Institute of Public Health of Mexico approved all study procedures. All participants gave written informed consent.

Population

The study population consisted of men who met the following eligibility criteria: (a) ages 18 to 70 years; (b) residents of one of three sites—Sao Paulo, Brazil; the state of Morelos, Mexico; or metropolitan Tampa, Florida, United States; (c) reported no prior diagnosis of penile or anal cancers; (d) had never been diagnosed with genital or anal warts; (e) reported no symptoms of a sexually transmitted infection or treatment for a sexually transmitted infection; (f) were not participating in an HPV vaccine study; (g) had no history of HIV or AIDS; (h) had no history of imprisonment, homelessness, or drug treatment during the past 6 months; and (i) were willing to comply with 10 scheduled visits every 6 months for 4 years with no plans to relocate during this period. To encourage participation by men with a broad range of ages, sexual behaviors and HPV risk factors, men were recruited from three different population sources—the general population, universities, and organized health care systems (Mexico only).

In Brazil, men were also recruited from the general population at a facility for urogenital care (Centro de Referencia e Tratamento de Doencas Sexualmente Transmissiveis e AIDS, Sao Paulo) and through general media advertising. Men presenting for non–sexually transmitted infection related conditions were enrolled in the present study. In addition, the

spouses and partners of women participating in a large cohort study of the natural history of HPV infection and risk of cervical neoplasia conducted in Sao Paulo since 1993 were also recruited. At the Cuernavaca, Mexico site, the underlying population was comprised of employees and beneficiaries of the Instituto Mexicano de Seguro Social, factory employees, and officials of the Mexican army that were permanently assigned to this geographic area. In the United States, the underlying population came from the University of South Florida and the greater Tampa metropolitan area. Flyers and posters were distributed throughout the campus and community. In addition, men from the broader Tampa community were recruited through the mail and media using brochures and flyers as well as advertisements in local and university papers.

The recruitment sources were similar in the three countries however the number of recruited subjects was slightly different per source. In the United States 40% of subjects were recruited by word of mouth, while 12% and 46% were recruited by the same method in Brazil and Mexico respectively. All three countries had flyers which were posted at universities, health clinics, and local businesses. The percentage recruited at each site by this method was 20%, 40% and 16% in the United States, Brazil, and Mexico respectively.

Study Protocol

The HIM Study protocol includes a pre-enrollment run-in visit, a baseline (enrollment) visit, and eight additional visits after enrollment scheduled 6 months apart. For this analysis, 4074 men who completed both the pre-enrollment and baseline visit formed the study population. Sixty-three men refused response to the race question and one hundred and two men had genital specimens that tested β-globin negative at baseline; therefore 3,909 men who answered the question on race and had genital specimens adequate for HPV determination were included in this analysis.

Risk Factor Questionnaire

An extensive sexual history and health questionnaire given at enrollment assessed sociodemographic characteristics, sexual history, condom use practices, alcohol and tobacco use, and history of abnormal Pap smears in female partners. Based on responses to questions related to any type of sexual contact with another man, number of lifetime male sexual partners was estimated. The questionnaire required approximately 20 minutes to complete and was self-administered using computer assisted self-interviewing. Race and ethnicity were self-reported with one question assessing race and another ethnicity (Hispanic vs. non-Hispanic). In Mexico, US based racial/ethnic categories are not utilized and they self-identify as Mestizo ancestry. Therefore for this analysis we created a new race category, “Mexican”, to denote this mixed ancestry.

HPV Penile and Scrotal Sampling

To maximize sampling and prevent fraying of applicators, three different pre wetted Dacron applicators were used to sample the external genitalia of the participants, and were later combined to form a single sample for the detection of HPV. This method has been previously shown to maximize HPV detection among men and to result in reproducible detection of genital HPV in men.[20,21] The study clinician at each site first swept 360° around the coronal sulcus and then another 360° around the glans penis and placed this swab into a collection vial with standard transport media (STM). A second swab was used to sample the entire skin surface of each of the quadrants of the shaft of the penis (left and right ventral, and left and right dorsal) and placed into a separate collection vial containing STM. A third swab was used for scrotum sampling. All three swabs were placed in separate collection vials containing 500 μl STM. Among uncircumcised men, the foreskin was sampled at the time of collection of the coronal sulcus/glans penis sample. All HPV samples

were stored at −70°C until PCR analyses and genotyping were conducted. Prior to DNA extraction, the three samples were combined to produce one DNA extract per participant clinic visit.

HPV Analysis

HPV testing of the combined DNA extract was conducted using PCR for amplification of a fragment of the HPV L1 gene.[22] DNA extraction was conducted using the QIAamp DNA Mini Kit (Qiagen) according to the instructions of the manufacturer. Briefly, 200 μL aliquots of clinical material were digested with 20 μL of proteinase K solution for 1 h at 65°C, followed by 200 μL of lysis buffer. Specimens were tested for the presence of HPV by amplifying 50 μL of the DNA extracts using the Linear Array HPV genotyping test following the instructions of the manufacturer (Roche Diagnostics). Samples were amplified using Perkin-Elmer GeneAmp PCR System 9700 as directed by the linear array protocol. HPV genotyping was conducted on all samples regardless of HPV PCR result.[23] A participant was considered positive for "any HPV" if he tested HPV positive by PCR or tested positive for at least one genotype. The "Oncogenic" HPV category included men who were positive for at least one of the 13 oncogenic types tested for (HPV types 16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, and 66), including infections with both oncogenic and non-oncogenic types. "Non-oncogenic" HPV infections included single or multiple infections with only non-oncogenic HPV types (6, 11, 26, 40, 42, 53, 54, 55, 61, 62, 64, 67, 68, 69–73, 81–84, IS39, and CP6108). Among the tested samples, 96.8% of specimens obtained were positive for β-Globin. We excluded specimens that were negative for β-Globin and were not considered positive for any HPV type, by either PCR or genotype. Specimens that tested positive for HPV PCR but negative for HPV genotyping were considered HPV unclassified.

Statistical Analysis

Differences in the distribution of sociodemographic and sexual behavior characteristics across race groups were compared using the Monte Carlo estimation of exact Pearson chi-square p-values. Prevalence ratios (PRs) and 95% confidence intervals (CIs) were calculated using univariate and multivariable Poisson regression, with the robust variance.[24,25] Separate models were performed for each of the four HPV outcomes (i.e., any HPV, oncogenic HPV, non-oncogenic only HPV, and unclassified only HPV). White race served as the reference group for the association between race and HPV infection, and men negative for HPV served as the comparison group for the four HPV outcomes. The same sets of variables were included in all multivariable modeling. For variable selection, we first included all candidate variables in a model, and then individually eliminated the variables with a p-value greater than 0.05, starting from the least significant one. This variable selection procedure was performed for the four HPV outcomes. The variables which were not significant in any models, were excluded from the final multivariable models. The candidate variables for the multivariable models included race, age (continuous), education, marital status, current smoking status, circumcision status, lifetime number of female partners, number of female partners in previous 3 or 6 months, lifetime number of male partners, and number of male partners in previous 3 or 6 months. In the sub-group analyses of lifetime number of female partners, PRs and 95% CIs were generated for assessing the association between race and each of the four HPV outcomes. Due to limited sample size in some sub-groups, univariate models were performed only in the sub-groups with more than 20 men.

Results

Table 1 presents the sociodemographic distribution of participants by race. Overall, Asian/Pacific Islanders were more likely to be younger, more educated, single and have fewer

numbers of sexual partners. Whites were less likely to have a new female partner in the previous 3 or 6 months. Blacks were more likely to be older than 45 years, cohabiting, divorced/separated, and have more numbers of female partners within 6 months preceding enrollment. Mexicans were more likely to have less than 12 years of education, be married, be a current smoker, and less likely to be divorced and circumcised (Physician-assessed circumcision) compared to the other races. Mixed race and American Indian were classified as “other” and were more likely to be non-smokers, and have more than 3 new female partners in the previous 3 or 6 months.

Asian/Pacific Islanders had the lowest prevalence of all types of HPV except unclassified HPV and HPV 18 where Asians had the highest prevalence (Table 2). Prevalence of any HPV was statistically significantly lower among Asian/Pacific Islanders (42.3%) compared to Black (66.2%), Mexican (62.3%), Other (67.3%), and White (71.5%) races ($p<0.0001$). The prevalence of HPV infection with oncogenic types was different across race groups ($p<0.0069$). 18.9% of the Asian/Pacific Islanders were positive for oncogenic HPV, while 32.2% of Black, 27.5% of Mexican, 27.1% of other and 31.4% of Whites were positive for any oncogenic type. Prevalence of non-oncogenic HPV type only statistically significantly differed by race ($p=0.0006$), with the highest prevalence observed among Other (24.8%), followed by Black (24.4%), White (23.3%), Mexican (22%) and Asian/Pacific Islanders (6.3%). Prevalence of unclassified HPV differed by race ($p=0.0001$), with Asian/Pacific Islander having the highest (17.1%), followed by White (16.8%), Other (15.4%), Mexican (12.8%), and Black (9.6%) races. We also observed differences in prevalence by race for HPV6, HPV 11, HPV 16, and HPV 18, with significant differences for HPV 11 and HPV 16 ($p=0.02$ and $p=0.0004$ respectively). HPV 6 was most prevalent in the Mexican race group (5.8%). Mexicans also had the highest prevalence of HPV 11 (2.2%), while the prevalence of HPV 16 was highest among Whites (9.2%) and the prevalence of HPV 18 was highest among Asian/Pacific Islanders (3.6%).

Table 3 presents the associations between race and any oncogenic, non-oncogenic and unclassified HPV infection. Compared to Whites, Asian/Pacific Islanders were significantly less likely to have any HPV (PR = 0.59; 95% CI: 0.48, 0.74), oncogenic HPV (PR = 0.47; 95% CI: 0.32, 0.69), or non-oncogenic HPV (PR = 0.22; 95% CI: 0.11, 0.45) in univariate analysis. After adjustment for potential confounders, Asian/Pacific Islanders remained significantly less likely to have any HPV (PR = 0.65; 95% CI: 0.52, 0.80), oncogenic HPV (PR = 0.55; 95% CI: 0.39, 0.79), or non-oncogenic HPV (PR = 0.28; 95% CI: 0.14, 0.58).

Table 4 shows the association between race and HPV infection among the United States population only. Asian/Pacific Islanders showed significantly lower risk in univariate analyses for any HPV (PR = 0.53; 95% CI: 0.4, 0.7), oncogenic HPV (PR = 0.32; 95% CI: 0.18, 0.57) and non-oncogenic HPV (PR = 0.18; 95% CI: 0.07, 0.46) compared to those of white race. The significantly lower risk among Asian Pacific Islanders remained after adjustment for possible confounders; any HPV (PR = 0.61; 95% CI: 0.46, 0.81), oncogenic HPV (PR = 0.43; 95% CI: 0.24, 0.77) and non-oncogenic HPV (PR = 0.26; 95% CI: 0.1, 0.68).

Table 5 presents the association between race and HPV infection among the 18–30 year old subjects only from all the centers. Asian Pacific Islanders had significantly lower risk for any HPV (PR = 0.61; 95% CI: 0.48, 0.78), oncogenic HPV (PR = 0.49; 95% CI: 0.32, 0.75) and non-oncogenic HPV (PR = 0.18; 95% CI: 0.07, 0.47) compared to Whites. The significantly lower risk was also observed after adjustment for confounders; any HPV (PR = 0.64; 95% CI: 0.5, 0.81), oncogenic HPV (PR = 0.54; 95% CI: 0.36, 0.81) and non-oncogenic HPV (PR = 0.2; 95% CI: 0.08, 0.54).

The lower risk of HPV among Asian/Pacific Islanders remained statistically significant after stratifying by lifetime number of female partners (Table 6). A decreased risk was observed among Asians for any HPV in the strata of 0–1 (PR=0.51; 95% CI: 0.31, 0.84) as well as 2–9 (PR=0.66; 95% CI: 0.41, 0.91) sexual partners. Likewise, Asian race was associated with a decreased risk of non-oncogenic HPV in the strata of 0–1 (PR=0.14; 95% CI: 0.02, 1) as well as 2–9 (PR=0.37; 95% CI: 0.16, 0.85) sexual partners. For oncogenic HPV infection, a decreased risk among Asians was only observed for the 0–1 (PR=0.31; 95% CI: 0.11, 0.93) sexual partner strata.

Discussion

This is one of the few multinational studies of HPV prevalence among White, Black, Mixed races, American Indian, Mexican, and Asian race residing outside of Asia. Results from this study are consistent with our prior findings of racial differences in prevalence of HPV infection.[26] Overall, prevalence of any HPV infection (42.3%) and any oncogenic HPV (18.9%) was lowest among Asian/Pacific Islanders. The low prevalence of HPV among Asian/Pacific Islanders is consistent with previous studies among Asians of Chinese and Korean descent where the reported range of genital HPV prevalence was 28–43%.[12–16]

Absolute differences in the prevalence among our study population (Asian-American and Asian-Brazilian) and reports from Asian countries may be due to the differences in socio-cultural milieu. Asian-Americans are from more than 28 Asian countries. Many Asian cultural traditions place emphasis on strict social conduct with restrained sexuality. This conservative sexual behavior may have been abandoned by Asian-Americans as there is a positive correlation between the level of acculturation to the United States and engagement in sexual activity by Asian-Americans.[27] Over 80% of the Asian/Pacific Islander population in this study were from the United States and none were from Mexico. They comprised 7% of the US study population and <2% of the Brazil study population. In the HIM Study cohort Asian/Pacific Islanders, compared to other races were younger, less likely to be involved in risky sexual behaviors and practices, more likely to be single and attained a higher level of education. It is important to note that the Asian/PI population of the HIM Study reported a low number of total sexual partners in general and in comparison to the other racial groups in the study. However, these characteristics alone do not account for the consistently lower prevalence of HPV infection observed among Asian/Pacific Islanders since lower risk of HPV was observed even after adjustment for potential confounders and stratification on sexual behavior and age. The consistently lower HPV prevalence observed among Asian/Pacific Islanders in the United States population and among 18–30 year-old subjects from all the centers provide additional evidence that the observed differences by race are not solely related to the demographic distribution difference from the three centers. However, it should be noted that while significantly lower risk of HPV was observed among Asian/PI men with fewer sexual partners, the corresponding confidence intervals did overlap with those observed among men of other races.

It is possible that unmeasured behavioral factors and/or sexual mixing with low risk women account for the significantly lower HPV prevalence among Asian men. We do not have information on the race of their sexual partners. If their partners are Asian/PI women with low rates of HPV then this could be another explanation of the significantly lower HPV risk observed among Asian/PI men. Another possible explanation for these findings is that there may be race-specific differences in the frequency of germline variations in various immune regulating genes such as those coding for killer cell immunoglobin-like receptors and human leucocyte antigen (HLA) ligands. Killer cell immunoglobin-like receptors (KIRs) and their human leucocyte antigen ligands identify and destroy aberrant or virally infected cells, are highly polymorphic, and vary in frequency across the human population.[28] In fact, several

variants of HLA are common among Asians and one of the variants, HLA-DRB1*1301, has been shown to possess a possible protective role against HPV 16.[29] HLA-B15 was found to be protective against HPV 16 and HPV 52 infection in southern Chinese women at risk of HPV infection and cervical neoplasia.[30] Although to date there are no published data on KIRs and HPV, KIR2DS4*00101 was the predominant allele found in the Chinese Han population in a recent survey of KIR genes.[31] Thus, elucidating the possible role of germline variations in immune regulating genes could have clinical relevance by identifying susceptible individuals for infection and/or clearance.

A major strength of this study is the large sample size of the overall cohort and the diversity in behavior and participant race. Location of the study centers in three international cities is also strength of this study. As with every study of this nature, there are limitations that must be considered in interpreting the results. One of the limitations was the sample size for some of the racial groups. The Asian/Pacific Islanders constitute only 2.8% of the study population while Blacks made up 15.9% of the study population. We cannot totally rule out the possibility of selection bias in this study as about 80% of the Asian population was from the United States site alone. The Mexican population does not contribute at all to the Asian group. Residual confounding also cannot be ruled out as the racial groups might be different with the factors related to HPV prevalence and also because of the possibility of unmeasured sexual risk factors for HPV infection. Self- reporting risk factors might either have resulted in either under reporting or exaggeration of the risk factors which may have inadvertently affected the magnitude of the observed associations. The race categories used in this study are based on the United States Office of Management and Budget (OMB) revision to the standards for the classification of federal data on race and ethnicity. In this system Asians are persons with ancestral root to the far east, Southeast Asia, or the Indian subcontinent, and Native Hawaiian or Pacific Islanders are persons with ancestral root to Hawaii, Guam, Samoa, or other Pacific Islands. These racial categories are therefore imperfectly applied to populations residing outside of the US and may have led to some misclassification of race. Finally, although the overall power of the study is robust, generalizability of the findings may be limited as participants were not randomly selected.

Conclusion

We consistently observed the lowest prevalence of HPV infection among Asian/Pacific Islanders with moderate inverse associations after various multi-variable adjustments. Our findings are consistent with previous studies that reported a low prevalence of HPV infection among Asians in continental Asia. Further studies assessing the role of genetics between race and HPV infection are needed to understand this phenomenon.

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Table 1

Distribution of Demographic and Sexual Behavior Characteristics

Characteristic	Overall N (%)	White N (%)	Black N (%)	Asian/PI N (%)	Mexican N (%)	Other N (%)	P-Value[(1)]
Age: Years							
18–30	1909 (48.8)	925 (52.4)	273 (44.4)	90 (81.1)	489 (40.6)	132 (61.7)	<.0001
31–44	1495 (38.2)	609 (34.5)	248 (40.3)	19 (17.1)	553 (46)	66 (30.8)	
45–70	505 (12.9)	232 (13.1)	94 (15.3)	2 (1.8)	161 (13.4)	16 (7.5)	
Total	3909 (100.0)	1766 (45.2)	615 (15.7)	111 (2.8)	1203 (30.8)	214 (5.5)	
Clinic Site							
US	1258 (32.2)	840 (47.6)	221 (35.9)	89 (80.2)	0 (0.0)	108 (50.5)	<.0001
BZ	1373 (35.1)	854 (48.4)	391 (63.6)	22 (19.8)	0 (0.0)	106 (49.5)	
MX	1278 (32.7)	72 (4.1)	3 (0.5)	0 (0.0)	1203 (100.0)	0 (0.0)	
Total	3909 (100.0)	1766 (45.2)	615 (15.7)	111 (2.8)	1203 (30.8)	214 (5.5)	
Ethnicity							
Hispanic	1767 (45.5)	384 (21.9)	75 (12.3)	5 (4.5)	1201 (99.9)	102 (48.8)	<.0001
Non-Hispanic	2114 (54.5)	1367 (78.1)	533 (87.7)	106 (95.5)	1 (0.1)	107 (51.2)	
Total	3881 (100.0)	1751 (45.1)	608 (15.7)	111 (2.9)	1202 (31)	209 (5.4)	
Years of Education							
<12 Years	858 (22.0)	214 (12.1)	130 (21.2)	1 (0.9)	479 (40.1)	34 (15.9)	<.0001
Completed 12 Years	1042 (26.7)	433 (24.6)	226 (36.8)	29 (26.4)	292 (24.4)	62 (29.0)	
13–15 Years	992 (25.5)	625 (35.5)	135 (22.0)	53 (48.2)	112 (9.4)	67 (31.3)	
Completed 16 Years	769 (19.7)	372 (21.1)	100 (16.3)	17 (15.5)	239 (20.0)	41 (19.2)	
>=17 Years	235 (6.0)	118 (6.7)	23 (3.7)	10 (9.1)	74 (6.2)	10 (4.7)	
Total	3896 (100.0)	1762 (45.2)	614 (15.8)	110 (2.8)	1196 (30.7)	214 (5.5)	
Marital Status							
Single	1764 (45.2)	987 (56.0)	292 (47.6)	94 (84.7)	279 (23.3)	112 (52.6)	<.0001
Married	1333 (34.2)	424 (24.1)	156 (25.4)	11 (9.9)	692 (57.7)	50 (23.5)	
Cohabiting	467 (12.0)	166 (9.4)	95 (15.5)	3 (2.7)	175 (14.6)	28 (13.1)	
Divorced/Separated/Widowed	335 (8.6)	185 (10.5)	70 (11.4)	3 (2.7)	54 (4.5)	23 (10.8)	
Total	3899 (100.0)	1762 (45.2)	613 (15.7)	111 (2.8)	1200 (30.8)	213 (5.5)	
Current Smoker							

Characteristic	Overall N (%)	White N (%)	Black N (%)	Asian/PI N (%)	Mexican N (%)	Other N (%)	P-Value(1)
No	2985 (76.4)	1399 (79.3)	488 (79.5)	98 (89.1)	823 (68.4)	177 (82.7)	<.0001
Yes	920 (23.6)	365 (20.7)	126 (20.5)	12 (10.9)	380 (31.6)	37 (17.3)	
Total	3905 (100.0)	1764 (45.2)	614 (15.7)	110 (2.8)	1203 (30.8)	214 (5.5)	
Circumcised							
Not Circumcised	2500 (64.0)	877 (49.7)	397 (64.6)	65 (58.6)	1020 (84.8)	141 (65.9)	<.0001
Circumcised	1409 (36.0)	889 (50.3)	218 (35.4)	46 (41.4)	183 (15.2)	73 (34.1)	
Total	3909 (100.0)	1766 (45.2)	615 (15.7)	111 (2.8)	1203 (30.8)	214 (5.5)	
Lifetime Number of Female Partners							
0–1	683 (17.5)	307 (17.4)	104 (16.9)	36 (32.4)	200 (16.6)	36 (16.8)	<.0001
2–9	1574 (40.3)	638 (36.1)	163 (26.5)	53 (47.7)	641 (53.3)	79 (36.9)	
10–29	967 (24.7)	474 (26.8)	184 (29.9)	6 (5.4)	253 (21.0)	50 (23.4)	
30+	476 (12.2)	267 (15.1)	114 (18.5)	9 (8.1)	48 (4.0)	38 (17.8)	
Refused	209 (5.3)	80 (4.5)	50 (8.1)	7 (6.3)	61 (5.1)	11 (5.1)	
Total	3909 (100.0)	1766 (45.2)	615 (15.7)	111 (2.8)	1203 (30.8)	214 (5.5)	
Number of Female Partners in Past 3 or 6 Months							
None	806 (20.6)	315 (17.8)	99 (16.1)	17 (15.3)	338 (28.1)	37 (17.3)	<.0001
1	1584 (40.5)	788 (44.6)	210 (34.1)	54 (48.6)	463 (38.5)	69 (32.2)	
2	507 (13.0)	211 (11.9)	94 (15.3)	10 (9.0)	163 (13.5)	29 (13.6)	
3+	514 (13.1)	256 (14.5)	116 (18.9)	11 (9.9)	84 (7.0)	47 (22.0)	
Refused	498 (12.7)	196 (11.1)	96 (15.6)	19 (17.1)	155 (12.9)	32 (15.0)	
Total	3909 (100.0)	1766 (45.2)	615 (15.7)	111 (2.8)	1203 (30.8)	214 (5.5)	
Lifetime Number of Male Partners							
0–1	3498 (89.5)	1521 (86.1)	528 (85.9)	108 (97.3)	1147 (95.3)	194 (90.7)	<.0001
2–9	228 (5.8)	127 (7.2)	49 (8.0)	3 (2.7)	42 (3.5)	7 (3.3)	
10–29	85 (2.2)	50 (2.8)	18 (2.9)	0 (0.0)	11 (0.9)	6 (2.8)	
30+	69 (1.8)	48 (2.7)	14 (2.3)	0 (0.0)	2 (0.2)	5 (2.3)	
Refused	29 (0.7)	20 (1.1)	6 (1.0)	0 (0.0)	1 (0.1)	2 (0.9)	
Total	3909 (100.0)	1766 (45.2)	615 (15.7)	111 (2.8)	1203 (30.8)	214 (5.5)	
Number of Male Partners in Past 3 or 6 Months							
None	3645 (93.8)	1601 (91.3)	567 (92.5)	110 (99.1)	1170 (97.9)	197 (92.9)	<.0001
1	100 (2.6)	60 (3.4)	16 (2.6)	1 (0.9)	18 (1.5)	5 (2.4)	

Characteristic	Overall N (%)	White N (%)	Black N (%)	Asian/PI N (%)	Mexican N (%)	Other N (%)	P-Value(1)
2	46 (1.2)	30 (1.7)	9 (1.5)	0 (0.0)	3 (0.3)	4 (1.9)	
3+	93 (2.4)	62 (3.5)	21 (3.4)	0 (0.0)	4 (0.3)	6 (2.8)	
Total	3884 (100.0)	1753 (45.1)	613 (15.8)	111 (2.9)	1195 (30.8)	212 (5.5)	
HPV Grouping							
Pure Negative	1299 (33.2)	504 (28.5)	208 (33.8)	64 (57.7)	453 (37.7)	70 (32.7)	<.0001
Oncogenic	1162 (29.7)	554 (31.4)	198 (32.2)	21 (18.9)	331 (27.5)	58 (27.1)	
Non-Oncogenic Only	886 (22.7)	411 (23.3)	150 (24.4)	7 (6.3)	265 (22.0)	53 (24.8)	
Unclassified Only	562 (14.4)	297 (16.8)	59 (9.6)	19 (17.1)	154 (12.8)	33 (15.4)	
Total	3909 (100.0)	1766 (45.2)	615 (15.7)	111 (2.8)	1203 (30.8)	214 (5.5)	

(1)Exact Pearson Chi-squared p value using Monte Carlo estimation

Table 2

HPV Infection distribution by race

		Race					
Factors n(%)	**Overall (n=3909)**	**White (n=1766)**	**Black (n=615)**	**Asian/PI (n=111)**	**Mexican (n=1203)**	**Other (n=214)**	**P Value(1)**
Positive for Any HPV	2610 (66.8)	1262 (71.5)	407 (66.2)	47 (42.3)	750 (62.3)	144 (67.3)	<.0001
Positive for Oncogenic HPV	1162 (29.7)	554 (31.4)	198 (32.2)	21 (18.9)	331 (27.5)	58 (27.1)	0.0069
Positive for Non-Oncogenic HPV Only	886 (22.7)	411 (23.3)	150 (24.4)	7 (6.3)	265 (22.0)	53 (24.8)	0.0006
Positive for Unclassified HPV Only	562 (14.4)	297 (16.8)	59 (9.6)	19 (17.1)	154 (12.8)	33 (15.4)	0.0001
Positive for HPV 6	238 (6.1)	118 (6.7)	35 (5.7)	5 (4.5)	70 (5.8)	10 (4.7)	0.6194
Positive for HPV 11	52 (1.3)	17 (1.0)	5 (0.8)	0 (0.0)	27 (2.2)	3 (1.4)	0.0199
Positive for HPV 16	299 (7.6)	162 (9.2)	55 (8.9)	4 (3.6)	62 (5.2)	16 (7.5)	0.0004
Positive for HPV 18	90 (2.3)	52 (2.9)	13 (2.1)	4 (3.6)	17 (1.4)	4 (1.9)	0.0708

Table 3

Association between race and HPV infection

	Any HPV		Oncogenic HPV		Non-Oncogenic Only HPV		Unclassified Only HPV	
Race	**Univariate PR (95% CI) n=3909**	**Multivariable PR (95% CI) n=3874**	**Univariate PR (95% CI) n=2461**	**Multivariable PR (95% CI) n=2440**	**Univariate PR (95% CI) n=2185**	**Multivariable PR (95% CI) n=2171**	**Univariate PR (95% CI) n=1861**	**Multivariable PR (95% CI) n=1849**
White	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
Black	0.93 (0.87–0.99)	0.9 (0.84–0.96)	0.93 (0.83–1.04)	0.88 (0.79–0.99)	0.93 (0.81–1.07)	0.86 (0.74–0.99)	0.60 (0.47–0.76)	0.63 (0.49–0.81)
Asian/PI	0.59 (0.48–0.74)	0.65 (0.52–0.80)	0.47 (0.32–0.69)	0.55 (0.39–0.79)	0.22 (0.11–0.45)	0.28 (0.14–0.58)	0.62 (0.41–0.93)	0.64 (0.42–0.95)
Mexican	0.87 (0.83–0.92)	0.92 (0.86–0.97)	0.81 (0.73–0.89)	0.95 (0.85–1.06)	0.82 (0.73–0.93)	0.87 (0.77–1.00)	0.68 (0.58–0.81)	0.69 (0.57–0.83)
Other	0.94 (0.85–1.04)	0.93 (0.84–1.02)	0.87 (0.71–1.06)	0.82 (0.67–1.00)	0.96 (0.77–1.19)	0.94 (0.76–1.16)	0.86 (0.64–1.16)	0.91 (0.68–1.22)

Table 4

Association Between Race and HPV Infection (Prevalence Ratios) for US Only

	Any HPV		Oncogenic HPV		Non-Oncogenic Only HPV		Unclassified Only HPV	
Race	**Univariate PR (95% CI)**	**Multivariable PR (95% CI)**	**Univariate PR (95% CI)**	**Multivariable PR (95% CI)**	**Univariate PR (95% CI)**	**Multivariable PR (95% CI)**	**Univariate PR (95% CI)**	**Multivariable PR (95% CI)**
White	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
Black	0.94 (0.85–1.05)	0.91 (0.81–1.01)	1.02 (0.84–1.23)	0.94 (0.77–1.14)	1.05 (0.82–1.35)	0.94 (0.73–1.20)	0.46 (0.29–0.72)	0.46 (0.28–0.73)
Asian/PI	0.53 (0.40–0.70)	0.61 (0.46–0.81)	0.32 (0.18–0.57)	0.43 (0.24–0.77)	0.18 (0.07–0.46)	0.26 (0.10–0.68)	0.63 (0.41–0.95)	0.63 (0.41–0.98)
Other	0.90 (0.77–1.06)	0.92 (0.79–1.08)	0.84 (0.61–1.14)	0.87 (0.65–1.18)	0.87 (0.59–1.27)	0.95 (0.66–1.37)	0.81 (0.55–1.20)	0.82 (0.55–1.22)

PR, Prevalence Ratio; CI, Confidence Interval.

Prevalence Ratios using PROC GENMOD in SAS

Multivariable models are adjusted for age (continuous), circumcision status, marital status, lifetime number of female partners, number of female partners in previous 3/6 months.

Candidate variables education and current smoking status were not adjusted for because they would not significant in any models.

Candidate variables lifetime number of male partners and number of male partners in previous 3/6 months were not adjusted for because of small sample sizes.

Table 5

Association Between Race and HPV Infection (Prevalence Ratios) 18–30 Year Old Subjects Only

	Any HPV		Oncogenic HPV		Non-Oncogenic Only HPV		Unclassified Only HPV	
Race	**Univariate PR (95% CI)**	**Multivariable PR (95% CI)**	**Univariate PR (95% CI)**	**Multivariable PR (95% CI)**	**Univariate PR (95% CI)**	**Multivariable PR (95% CI)**	**Univariate PR (95% CI)**	**Multivariable PR (95% CI)**
White	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
Black	0.95 (0.87–1.05)	0.90 (0.82–0.99)	1.08 (0.93–1.26)	0.98 (0.84–1.13)	0.83 (0.64–1.08)	0.76 (0.59–0.99)	0.60 (0.42–0.85)	0.63 (0.44–0.91)
Asian/PI	0.61 (0.48–0.78)	0.64 (0.50–0.81)	0.49 (0.32–0.75)	0.54 (0.36–0.81)	0.18 (0.07–0.47)	0.20 (0.08–0.54)	0.65 (0.43–1.01)	0.65 (0.42–1.00)
Other	0.99 (0.87–1.12)	0.94 (0.83–1.06)	0.98 (0.77–1.24)	0.89 (0.71–1.12)	1.08 (0.81–1.44)	0.95 (0.72–1.27)	0.83 (0.56–1.22)	0.81 (0.55–1.20)
Mexican	0.88 (0.81–0.96)	0.88 (0.81–0.96)	0.82 (0.71–0.96)	0.91 (0.78–1.07)	0.89 (0.73–1.08)	0.80 (0.65–0.98)	0.65 (0.51–0.83)	0.66 (0.50–0.87)

PR, Prevalence Ratio; CI, Confidence Interval.

Prevalence Ratios using PROC GENMOD in SAS

Multivariable models are adjusted for circumcision status, marital status, lifetime number of female partners, number of female partners in previous 3/6 months.

Candidate variables education and current smoking status were not adjusted for because they would not significant in any models.

Candidate variables lifetime number of male partners and number of male partners in previous 3/6 months were not adjusted for because of small sample sizes.

Table 6

Association Between Race and HPV Infection by Lifetime Number of Partners

	Lifetime Number Of Female Partners				
	0–1 PR (95% CI)	**2–9 PR (95% CI)**	**10–29 PR (95% CI)**	**30+ PR (95% CI)**	**Refused PR (95% CI)**
Any HPV					
Race, N	683	1574	961	467	191
White	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
Black	0.85 (0.69–1.05)	0.99 (0.88–1.13)	0.86 (0.77–0.95)	0.9 (0.79–1.02)	0.99 (0.80–1.21)
Asian/PI	0.51 (0.31–0.84)	0.66 (0.49–0.91)	NE	NE	NE
Mexican	0.85 (0.72–1.00)	0.93 (0.86–1.01)	0.90 (0.83–0.98)	1.00 (0.87–1.16)	0.72 (0.55–0.94)
Other	0.74 (0.51–1.08)	1.03 (0.87–1.21)	0.81 (0.66–1.00)	1.04 (0.90–1.21)	NE
Oncogenic HPV					
Race, N	452	956	609	293	127
White	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
Black	0.94 (0.64–1.37)	1.14 (0.88–1.47)	0.77 (0.64–0.92)	0.88 (0.72–1.08)	0.89 (0.60–1.31)
Asian/PI	0.31 (0.11–0.93)	0.60 (0.33–1.07)	NE	NE	NE
Mexican	0.83 (0.6–1.16)	1.01 (0.84–1.20)	0.85 (0.74–0.99)	0.92 (0.68–1.26)	0.61 (0.39–0.93)
Other	0.58 (0.26–1.31)	1.05 (0.72–1.54)	0.65 (0.44–0.97)	1.05 (0.80–1.38)	NE
Non-Oncogenic HPV Only					
Race, N	425	912	495	232	109
White	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
Black	0.93 (0.58–1.51)	1.04 (0.79–1.38)	0.8 (0.64–1.01)	0.77 (0.57–1.05)	1.11 (0.72–1.71)
Asian/PI	0.14 (0.02–1.00)	0.37 (0.16–0.85)	NE	NE	NE
Mexican	1.04 (0.72–1.51)	0.89 (0.74–1.08)	0.85 (0.70–1.04)	1.09 (0.79–1.49)	0.51 (0.28–0.95)
Other	0.97 (0.49–1.92)	0.99 (0.66–1.49)	0.79 (0.54–1.16)	1.16 (0.85–1.60)	NE
Unclassified HPV Only					
Race, N	440	874	317	113	60
White	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
Black	0.5 (0.29–0.86)	0.74 (0.51–1.08)	0.50 (0.30–0.86)	0.58 (0.27–1.21)	NE
Asian/PI	0.54 (0.36–0.79)	0.74 (0.60–0.91)	0.54 (0.33–0.86)	NE	0.76 (0.28–2.10)
Mexican	0.58 (0.29–1.14)	0.60 (0.33–1.08)	NE	NE	NE

	Lifetime Number Of Female Partners				
	0–1 PR (95% CI)	**2–9 PR (95% CI)**	**10–29 PR (95% CI)**	**30+ PR (95% CI)**	**Refused PR (95% CI)**
Other	0.44 (0.18–1.10)	1.09 (0.76–1.58)	0.51 (0.21–1.26)	NE	NE

PR, Prevalence Ratio; CI, Confidence Interval

Men pure negative for HPV served as the comparison group for the four HPV outcomes

Models were unadjusted for any other variables due to small sample sizes

NE: Not estimated due to race sub-groups with <20 samples