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# Racial/Ethnic Differences in HPV 16/18 Genotypes and Integration Status among Women with a History of Cytological Abnormalities

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## Abstract

**Objective—**HPV genotype distribution varies by race/ethnicity, but is unclear whether there are racial/ethnic variations in HPV 16/18 integration in the host genome. We describe HPV16/18 infection and integration status in a racially/ethnically diverse sample of women with a recent abnormal Pap test.

**Methods—**Patients (n=640) represent a subset of women participating in a clinical trial. Cervical swabs were tested for HPV16/18 DNA using type-specific polymerase chain reaction assays. Viral integration status was assessed using type-specific integration assays and categorized as fully integrated, fully non-integrated, or mixed. Unconditional logistic regression was used to generate unadjusted (OR) and adjusted odds ratios (aOR) to assess the association between self-reported race/ethnicity and risk of these outcomes.

**Results—**Hispanic and non-Hispanic black women had half the odds of prevalent HPV16 compared to non-Hispanic white women (aORs: 0.43 and 0.45, respectively). The prevalence odds

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of HPV18 was less than half among Hispanic women (aOR: 0.48), but not significantly different between black and white women (aOR: 0.72). Among women with prevalent HPV16, the odds of fully integrated viral DNA were significantly higher among black women (aORs: 2.78) and marginally higher among Hispanic women (aOR: 1.93). No racial/ethnic differences were observed for HPV18 DNA integration.

**Conclusions—**While HPV16 and 18 infections were less prevalent among Hispanic and black women compared to whites, their HPV16 DNA was more likely to be present in a fully integrated state. This could potentially contribute to the higher rates of abnormal cytology and cervical dysplasia observed among Hispanic and black women.

### Keywords

Human papillomavirus; HPV16; HPV18; viral integration; race/ethnicity

## Introduction

Cervical cancer is the second most common malignancy among women worldwide (1) Although widespread screening has drastically declined its incidence in the United States (U.S.) and other developed countries, an estimated 12,820 women will be diagnosed with cervical cancer in the U.S. in 2017 and 4,210 will experience a cervical-cancer related death (2, 3). Significant racial/ethnic disparities persist in the incidence of invasive cervical cancer in the U.S., with incidence rates 19 to 29% higher among non-Hispanic black and Hispanic women, respectively, compared to non-Hispanic whites (3). While socioeconomic and healthcare factors are postulated to drive this disparity, biological and genetic factors may affect the genomic behavior of the causative agent, human papillomavirus (HPV)(4).

Persistent infection with carcinogenic HPV is a causal and necessary factor for the development of cervical cancer (5). While there are 15 high-risk types of anogenital HPV (6), high-risk HPV types 16 and 18 are causally associated with over 70% of cervical cancers (7) and approximately 50% of cervical pre-cancerous lesions (8). These genotypes are the primary targets of the three currently-licensed HPV vaccines (Cervarix®, Gardasil®, and Gardasil9®). High-risk HPV types 31, 33, 45, 52, and 58 are associated with an additional 20% of cervical cancers and are additional targets of the most recently licensed nonavalent vaccine (Gardasil9®).

Persistent infection with high-risk HPV drives carcinogenesis by creating an environment of local immune suppression and genomic instability that leads to genomic alterations to the host cell as well as the integration of the viral and host genomes (9). Such viral integration is believed to be a key factor for the carcinogenic transformation of HPV. During its natural life cycle and in low-grade lesions, the HPV genome persists in a circular (episomal) form. With the progression from high-grade lesions to carcinomas, an increasing number of HPV genomes become integrated into the host genome (10–12). In addition to severity of disease, the prevalence of episomal versus integrated HPV genomes varies by infecting HPV type. For example, HPV 16 is more likely to be present in mixed form, expressing both episomal and integrated viral genomes (13).

There is evidence that the distribution of HPV genotypes varies by race/ethnicity (14–17). In particular, HPV 16 and 18 may be less prevalent among Hispanic and non-Hispanic black women compared to non-Hispanic white women (16, 17). However, it is unclear whether HPV 16 and 18 infections interact differently with hosts depending on their racial/ethnic backgrounds. Here we describe HPV 16/18 infection and integration status in a racially/ethnically diverse sample of women with a recent abnormal Pap test.

## Methods

Patients in this analysis represent a subset of those participating in a National Institutes of Health-supported clinical trial to evaluate emerging optical technologies for the diagnosis of cervical dysplasia. Recruitment and other study methods for the parent trial have been described in detail elsewhere (18–20). Briefly, trial participants were non-pregnant women, 18 years of age or older, with a history of an abnormal Pap test in the past 12 months. Thus participants may or may not have had a prevalent cytological abnormality at the time of participation, given that some cytological abnormalities may regress in that period of time. Patients were recruited at clinical sites in Houston, Texas, USA and Vancouver, British Columbia, Canada. As part of the parent trial, participants provided a cervical swab and a biopsy and answered an interviewer-administered questionnaire to ascertain demographic and behavioral risk factor information. The study protocol was reviewed and approved by the Institutional Review Boards of all participating institutions, and all study participants provided written informed consent.

Cervical swabs were initially tested for the presence of low- and high-risk HPV types using the Hybrid Capture II assay (Digene, Gaithersburg, MD) in a CLIA-certified commercial laboratory (LabCorp). In addition, specific typing for HPV 16 and HPV 18 was also conducting on a random sample of samples following the well-established protocol of Manos and Ting (21). Briefly, viral DNA was extracted from cervical cytobrush specimens using a commercially available kit (QIAamp DNA Mini Kit, Qiagen, Valencia, CA). We analyzed the samples for HPV DNA using MY9 and MY11, consensus HPV primers that amplify a 450 bp region of the L1 open reading frame of at least 28 different HPV types. PCR products were resolved by agarose gel electrophoresis, transferred to nylon membranes (Bio-Rad Laboratories, Hercules, CA), and hybridized to a $^{32}$P-labeled HPV consensus probe. Consensus probe-positive samples were then hybridized to $^{32}$P-labeled specific HPV 16 and HPV 18 probes on separate nylon membranes. Sample positivity was assessed by autoradiography following hybridization. Additionally, for samples collected later during the trial, the Linear Array HPV Genotyping Test (Roche Molecular Diagnostics, Pleasanton, CA) was completed according to manufacturer protocol to determine the presence of additional HPV types.

Type-specific HPV integration was determined for samples reported to be HPV 16-positive and/or HPV 18-positive using the method of Peitsaro et al.(22) with modifications described by Huang et al. (23). This real-time PCR method involved targeting the HPV E2 open reading frame (ORF), which is most often deleted during HPV integration, and the E6 ORF, which is not disrupted during integration. The primer sequences for E2 and E6 for HPV 16 and HPV 18 were previously reported by Ho et al. (24). If the HPV is episomal, both gene

targets should be equivalent. If the HPV is integrated, then the copy number of E2 should be less than E6 (i.e., E2/E6 ratio < 1.0). If E6 is detected but not E2, then the sample will be noted as having full HPV integration with no episomal forms present. This assay has been shown to be highly effective for determining integration status when various amounts of mixed episomal/integrated forms are present; as well as being one of the only methods able to detect only integrated HPV (25).

### Measures

Outcome variables in this analysis are: HPV 16 infection, HPV 18 infection, HPV 16 integration, and HPV 18 integration. Infection status was categorized as positive for HPV 16 and/or 18 or negative for both HPV 16/18. Integration status was dichotomized as fully integrated versus partially- or non-integrated due to small number of cases in which episomal HPV was present in HPV 16- and 18-positive samples, particularly among non-Hispanic whites. The primary exposure variable, race/ethnicity, was categorized as Hispanic (regardless of race reported), non-Hispanic white, non-Hispanic black, non-Hispanic Asian, or non-Hispanic other. Other variables of interest included age, country of birth, age of sexual debut, history of tobacco use, and number of lifetime sexual partners.

### Statistical analyses

Statistical analyses were performed in Stata version 13.1 (College Station, TX: StataCorp LP). Frequencies and proportions were used to describe demographic/risk behavior characteristics and the prevalence of HPV 16 and 18 infection and integration status in the sample. Unconditional logistic regression was used to evaluate the associations between self-reported race/ethnicity and HPV-related outcomes of interest. First, we generated unadjusted odds ratios (OR) and 95% confidence intervals (CI) to evaluate the association between self-reported race/ethnicity (reference = non-Hispanic White) and the following outcomes: HPV 16-postivitiy (versus HPV 16- and 18-negativity); HPV 18-positivity (versus HPV 16- and 18-negativity); full HPV 16 or 18 integration (versus partial or no HPV integration); and level of cervical dysplasia (≥CIN2 versus ≤CIN1). For each outcome, we then calculated an adjusted OR (aOR) including other independent variables identified as important predictors of HPV risk in the literature. These covariates included: age (continuous), age at sexual debut (continuous); number of lifetime sexual partners (reference = 5–9), and history of tobacco use (reference = No).

## Results

Of the initial 1,850 women enrolled in the parent clinical trial, HPV 16/18 DNA and integration data were available for 640 (35%) participants. Of these, 59% were non-Hispanic white, 18% Hispanic, 15% non-Hispanic black, 6% non-Hispanic Asian, and 2% non-Hispanic other (Table 1). Additional demographic, risk behavior, and clinical characteristics are described in Table 1. Most participants were 30 years of age or older (76%), born in the U.S. or Canada (79%), and did not have history of tobacco use (60%). The median age of sexual debut was 17 years, and 50% had five or more lifetime sexual partners. HPV 16 DNA was detected in 62% of samples (45% alone and 17% with HPV 18 DNA), and HPV 18

DNA was detected in 28%. Almost half of participants (46%) had low grade dysplasia (≤CIN1) and 21% had high grade dysplasia (≥CIN2).

There were racial/ethnic differences in the prevalence of HPV 16 and 18 DNA. Specifically, Hispanic and non-Hispanic black women had half the odds of prevalent HPV 16 DNA compared to non-Hispanic white women (OR = 0.50; 95% CI: 0.32–0.79, and OR = 0.51; 95% CI: 0.30–0.85, respectively, Table 2). Similar patterns were observed after controlling for age, age of sexual debut, number of lifetime sexual partners, and history of tobacco use for both groups ($aOR_{Hispanic}$= 0.43; 95% CI: 0.26–0.72; $aOR_{non\text{-}Hispanic\ black}$ = 0.45; 95% CI: 0.25–0.78). In regard to prevalent HPV 18 DNA (Table 3), the prevalence odds was less than half among Hispanic compared to non-Hispanic white women (aOR = 0.48; 95% CI: 0.26–0.88), but no statistically significant differences were observed between non-Hispanic black and non-Hispanic white women (aOR = 0.72; 95% CI: 0.38–1.37).

The HPV integration data (Table 4) indicated that HPV18 was more likely than HPV 16 to be in a fully integrated versus partially/unintegrated state (range of fully integrated DNA across racial ethnic groups: 42–57% for HPV 18 versus 10–33% for HPV 16). Interesting integration patterns were observed across racial/ethnic groups. Among women with prevalent HPV 16 DNA (n=396), the odds of having fully integrated viral DNA was 82% higher among Hispanic versus non-Hispanic white women, both before and after adjusting for age and behavioral risk factors (aOR = 1.93; 95% CI: 0.96–3.89, Table 4a), although this difference was not statistically significant. A statistically significant greater odds of fully integrated HPV 16 were observed among non-Hispanic black women, who had over a two-fold greater odds of having fully integrated HPV 16 DNA (aOR = 2.78; 95% CI: 1.29–5.98). No racial/ethnic differences were observed in regard to HPV 18 DNA integration (Table 4b).

Patterns of cervical dysplasia status by race/ethnicity are described in Table 5. The odds of high grade dysplasia (≥CIN2) were lower among Hispanic versus non-Hispanic white women after adjusting for age and behavioral risk factors (aOR = 0.48; 95% CI: 0.25–0.91). There were no statistically significant differences in the odds of prevalent ≥CIN2 between non-Hispanic black and non-Hispanic white women.

## Discussion

Our data suggest that while HPV 16 and 18 infections are less prevalent among Hispanic and non-Hispanic black women compared to non-Hispanic whites, HPV 16 DNA is more likely to be in a fully integrated state among women of these racial/ethnic backgrounds, particularly non-Hispanic black women, despite a lower prevalence of ≥CIN2.

Findings on racial/ethnic differences in prevalent HPV 16 and 18 genotypes have been previously reported by our group (17) and by Vidal et al (16). In our prior analysis (17), non-Hispanic black and Hispanic women were about half as likely to be infected with HPV 16/18 compared to non-Hispanic white women (15% and 20% among blacks and Hispanics versus 38% among whites, $p<0.001$). Non-Hispanic black women were significantly more likely than non-Hispanic white women to have prevalent non-16/18 high-risk genotypes covered by the new nonavalent vaccine (most commonly HPV 52). However, both non-

Hispanic black and Hispanic women were more likely to have high-risk genotypes not covered by any of the licensed vaccines. Vidal et al. similarly reported that non-Hispanic black women were half as likely as non-Hispanic white women to harbor high-risk HPV 16/18 genotypes. As in our study, these racial/ethnic differences persisted when the analyses were restricted to women with histologically confirmed ≥CIN2.

The current study extends our previous work by describing racial/ethnic differences in viral integration status. Our data suggest that while HPV 16 infections are less common among Hispanic and non-Hispanic black women, when women of these race/ethnicities are infected, the viral genome is more likely to be present in a fully integrated state. Specifically, our data suggest a marginally significant 93% higher prevalence of fully-integrated HPV 16 DNA among Hispanics and a 2.8-fold higher prevalence among non-Hispanic black women. Interestingly, similar patterns were not observed for HPV 18 DNA. While HPV 18 DNA was less likely to be detected among Hispanic versus non-Hispanic white women, the integration status did not appear to vary by race/ethnicity.

Viral integration is believed to be a key process in the carcinogenesis of HPV. The recombination and integration of the viral and host genome is hypothesized to drive the transformation and immortalization of the host cells, which in turn is believed to confer a selective growth advantage over non-transformed cells (9). If this is the case, higher HPV 16 integration rates could contribute to the higher rates of cytological abnormalities and cervical dysplasia observed among non-Hispanic black (4) and Hispanic women (26) in the larger population, despite potentially lower rates of HPV 16/18 infection.

Contrary to larger population-level trends, we found that the non-Hispanic black and Hispanic women in our sample were less likely to have prevalent ≥CIN2 compared to non-Hispanic whites. We speculate that the lower prevalence of ≥CIN2 among these groups despite their higher prevalence of HPV16 integration could be explained in part by differences in the location of HPV integration into the host genome. While HPV integration can occur over the entire genome, there are certain accessible regions that are preferentially targeted for HPV integration (particularly transcriptionally active regions and regions with open chromatin) (28). Differences in the location of HPV integration would have implications for the affected pathways in the human genome and the subsequent likelihood of carcinogenesis (28). However, we are not aware of any studies that have evaluated differences in regions of HPV integration by race/ethnicity. Additionally, it is important to note that HPV 16-related cervical cancer in the absence of integration has been reported, as there are several other processes known to occur during tumorigenesis (27). Finally, the synergistic effects of HPV infection and other cofactors for the development of cervical cancer, particularly tobacco smoke (29, 30) could also partly explain the observed racial/ethnic trends in cervical dysplasia. While the high rates of cigarette smoking among women in our sample was high overall (40% across all women in the study), it was particularly prevalent among non-Hispanic whites (51%) who had the highest prevalence of ≥CIN2.

Our study has several strengths, including the racial/ethnic diversity of participants, the extensive biological data available (i.e., HPV DNA, integration status, and histology), and the inclusion of women with and without prevalent cytological abnormalities. Our findings

should nonetheless be interpreted in the context of certain limitations. The sample size is small for certain racial/ethnic groups, particularly Asians and Others, precluding our ability to examine HPV 16/18 infection and integration status among these groups. Also due to sample size constraints, we were limited in our ability to conduct sub-analyses describing racial/ethnic differences in integration status across histological categories. The small number of Hispanic, non-Hispanic black, and Asian women with episomal HPV 16/18 also restricted our ability to confirm whether consistent racial/ethnic viral integration patterns are observed when evaluating integration status as non-integrated (i.e., fully episomal) versus integrated (fully or partially). Finally, the lack of a probability-based sample limits the generalizability of our findings. Despite these limitations, the current analyses provide valuable data regarding differences in HPV 16/18 infection and integration status among racial/ethnic groups. While many studies have reported on racial/ethnic variation in HPV genotypes, we are aware of only one published study to examine racial/ethnic differences in HPV integration status (31). Han et al. (31) investigated the role of HPV 16 DNA integration in cervical lesions of Chinese women of Han and Uygur ethnicity, two populations with a high burden of cervical cancer for whom the prevalence of high-risk HPV varies greatly. No significant differences in viral integration were found across the two ethnic groups. Other studies have found racial/ethnic disparities in HPV persistence, specifically finding that high-risk HPV may persist longer among non-Hispanic black versus non-Hispanic white women (4). Further studies are needed to examine potential differences in the natural life history and carcinogenesis of HPV across women of different race/ethnicities.

In conclusion, our data corroborate findings that HPV 16 and 18 infections may be less prevalent among Hispanic and non-Hispanic black women compared to non-Hispanic whites. However, integration of HPV 16 DNA within the host genome may be more likely among non-Hispanic black, and to a lesser extent Hispanic women. This could potentially contribute to the higher rates of abnormal cytology and cervical dysplasia observed in these racial/ethnic groups.

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## Highlights

- HPV16 was less prevalent among Hispanic and black women versus white women.
- HPV16 DNA was more likely to be fully integrated among black women.
- HPV18 was less prevalent among black versus white women.
- HPV18 DNA integration status did not vary by race/ethnicity.

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**Table 1**

Demographic and HPV risk behavior characteristics of women with a history of an abnormal Pap test in the past 12 months participating in a clinical trial (n=640).

| Characteristic | Total | White | Hispanic | Asian | Black | Other |
|---|---|---|---|---|---|---|
| Age (years), *n* (%) | | | | | | |
| 18–24 | 68 (10.63) | 40 (10.61) | 13 (11.30) | 1 (2.44) | 12 (12.77) | 2 (15.38) |
| 25–29 | 88 (13.75) | 55 (14.59) | 14 (12.17) | 5 (12.20) | 12 (12.77) | 2 (15.38) |
| 30–34 | 109 (17.03) | 58 (15.38) | 26 (22.61) | 8 (19.51) | 15 (15.96) | 2 (15.38) |
| 35–39 | 94 (14.69) | 49 (13.00) | 22 (19.13) | 5 (12.20) | 16 (17.02) | 2 (15.38) |
| 40–44 | 83 (12.97) | 46 (12.20) | 11 (9.57) | 7 (17.07) | 17 (18.09) | 2 (15.38) |
| 45–49 | 83 (12.97) | 48 (12.73) | 16 (13.91) | 6 (14.63) | 12 (12.77) | 1 (7.69) |
| 50–54 | 53 (8.28) | 32 (8.49) | 7 (6.09) | 7 (17.07) | 6 (6.38) | 1 (7.69) |
| $55^{+}$ | 62 (9.69) | 49 (13.00) | 6 (5.22) | 2 (4.88) | 4 (4.26) | 1 (7.69) |
| Self-reported Race/Ethnicity, *n* (%) | | | | | | |
| White | 377 (58.91) | | | | | |
| Hispanic | 115 (17.97) | | | | | |
| Asian | 41 (6.41) | | | | | |
| Black | 94 (14.69) | | | | | |
| Other/Mixed | 13 (2.03) | | | | | |
| Birthplace | | | | | | |
| United States | 410 (64.06) | 260 (68.97) | 57 (49.57) | 2 (4.88) | 86 (91.49) | 5 (38.46) |
| Canada | 94 (14.69) | 91 (24.14) | 0 (0.00) | 0 (0.00) | 0 (0.00) | 3 (23.08) |
| Mexico | 31 (4.84) | 0 (0.00) | 31 (26.96) | 0 (0.00) | 0 (0.00) | 0 (0.00) |
| Central/South America | 29 (4.53) | 2 (0.53) | 27 (23.48) | 0 (0.00) | 0 (0.00) | 0 (0.00) |
| East Asia | 11 (1.72) | 0 (0.00) | 0 (0.00) | 11 (26.83) | 0 (0.00) | 0 (0.00) |
| Other | 63 (9.84) | 22 (5.84) | 0 (0.00) | 28 (68.29) | 8 (8.51) | 5 (38.46) |
| Unknown | 2 (0.31) | 2 (0.53) | 0 (0.00) | 0 (0.00) | 0 (0.00) | 0 (0.00) |
| History of Tobacco Use, *n* (%) | | | | | | |
| Yes | 255 (39.84) | 193 (51.19) | 32 (27.83) | 4 (9.76) | 18 (19.15) | 8 (61.54) |
| No | 384 (60.00) | 183 (48.54) | 83 (72.17) | 37 (90.24) | 76 (80.85) | 5 (38.46) |
| Unknown | 1 (0.16) | 1 (0.27) | 0 (0.00) | 0 (0.00) | 0 (0.00) | 0 (0.00) |

| Characteristic | Total | White | Hispanic | Asian | Black | Other |
|---|---|---|---|---|---|---|
| Age of Sexual Debut (median years; range) | 17 (4–37) | 17 (4–37) | 18 (7–30) | 23 (15–34) | 17 (7–35) | 18 (13–21) |
| Lifetime Sexual Partners, *n* (%) | | | | | | |
| 0–2 | 163 (25.47) | 76 (20.16) | 46 (40.00) | 26 (63.41) | 12 (12.77) | 3 (23.08) |
| 3–4 | 115 (17.97) | 61 (16.18) | 29 (25.22) | 6 (14.63) | 18 (19.15) | 1 (7.69) |
| 5–9 | 178 (27.81) | 107 (28.38) | 28 (24.35) | 5 (12.20) | 34 (36.17) | 4 (30.77) |
| 10 or More | 149 (23.28) | 112 (29.71) | 9 (7.83) | 3 (7.32) | 21 (22.34) | 4 (30.77) |
| Unknown | 35 (5.47) | 21 (5.57) | 3 (2.61) | 1 (2.44) | 9 (9.57) | 1 (7.69) |
| HPV 16 & 18 DNA, *n* (%) | | | | | | |
| HPV $16^+$ | 290 (45.31) | 184 (48.81) | 42 (36.52) | 25 (60.98) | 32 (34.02) | 7 (53.85) |
| HPV $18^+$ | 74 (11.56) | 43 (11.41) | 9 (7.83) | 3 (7.32) | 17 (18.09) | 2 (15.38) |
| HPV $16^+$ and $18^+$ | 106 (16.56) | 65 (17.24) | 21 (18.26) | 4 (9.76) | 14 (14.89) | 2 (15.38) |
| Negative for HPV $16^+$ or $18^+$ | 170 (26.56) | 85 (22.55) | 43 (37.39) | 9 (21.95) | 31 (32.98) | 2 (15.38) |
| Cervical Dysplasia, *n* (%) | | | | | | |
| Negative for Dysplasia | 190 (29.69) | | | | | |
| ≤CIN1 | 292 (45.63) | | | | | |
| ≥CIN2 | 137 (21.41) | | | | | |
| No Diagnosis Possible | 13 (2.03) | | | | | |
| Unknown | 8 (1.25) | | | | | |

## Table 2

Self-Reported Race/Ethnicity and HPV $16^+$ (n = 566).

| | HPV $16^+$ (*n*)* | HPV 16 and 18 Negative (*n*) | OR (95% CI) | aOR (95% CI)[a] |
|---|---|---|---|---|
| Self-Reported Race/Ethnicity | | | | |
| Non-Hispanic White | 249 (75%) | 85 (25%) | Reference (1.00) | Reference (1.00) |
| Hispanic | 63 (59%) | 43 (41%) | 0.50 (0.32–0.79) | 0.43 (0.26–0.72) |
| Non-Hispanic Black | 46 (60%) | 31 (40%) | 0.51 (0.30–0.85) | 0.45 (0.25–0.78) |
| Non-Hispanic Asian | 29 (76%) | 9 (24%) | 1.10 (0.50–2.42) | 1.11 (0.45–2.71) |
| Non-Hispanic Other | 9 (82%) | 2 (18%) | 1.54 (0.33–7.25) | — |

aOR adjusted for age (continuous); age of sexual debut (continuous); number of lifetime sexual partners (reference = 5–9); history of tobacco use (reference = no).

* Includes those who are also HPV $18^+$.

— Adjusted estimate not shown due to cell counts less than five.

## Table 3

Self-Reported Race/Ethnicity and HPV $18^+$ (n = 350).

| | HPV $18^+$ (*n*)* | HPV 16 and 18 Negative (*n*) | OR (95% CI) | aOR (95% CI)[a] |
|---|---|---|---|---|
| Self-Reported Race/Ethnicity | | | | |
| Non-Hispanic White | 108 (56%) | 85 (44%) | Reference (1.00) | Reference (1.00) |
| Hispanic | 30 (41%) | 43 (59%) | 0.55 (0.32–0.95) | 0.48 (0.26–0.88) |
| Non-Hispanic Black | 31 (50%) | 31 (50%) | 0.79 (0.44–1.40) | 0.72 (0.38–1.37) |
| Non-Hispanic Asian | 7 (44%) | 9 (56%) | 0.61 (0.22–1.71) | 0.49 (0.14–1.76) |
| Non-Hispanic Other | 4 (67%) | 2 (33%) | 1.57 (0.28–8.80) | — |

aOR adjusted for age (continuous); age of sexual debut (continuous); number of lifetime sexual partners (reference = 5–9); history of tobacco use (reference = no).

*Includes those who are also HPV $16^+$.

— Adjusted estimate not shown due to cell counts less than five.

## Table 4

Self-Reported Race/Ethnicity and a) HPV $16^+$ and b) and HPV $18^+$ Integration.

| | Fully/Partially Integrated (*n*) | Not Integrated (*n*) | OR (95% CI) | aOR (95% CI)[a] |
|---|---|---|---|---|
| a) HPV 16 Integration (n=396) | | | | |
| Self-Reported Race/Ethnicity | | | | |
| Non-Hispanic White | 42 (17%) | 207 (83%) | Reference (1.00) | Reference (1.00) |
| Hispanic | 17 (27%) | 46 (73%) | 1.82 (0.95–3.48) | 1.93 (0.96–3.89) |
| Non-Hispanic Black | 15 (33%) | 31 (67%) | 2.38 (1.18–4.80) | 2.78 (1.29–5.98) |
| Non-Hispanic Asian | 3 (10%) | 26 (90%) | 0.57 (0.16–1.97) | — |
| Non-Hispanic Other | 0 (0%) | 9 (100%) | *** | — |
| b) HPV 18 Integration (n=180) | | | | |
| Self-Reported Race/Ethnicity | | | | |
| Non-Hispanic White | 46 (43%) | 62 (57%) | Reference (1.00) | Reference (1.00) |
| Hispanic | 14 (47%) | 16 (53%) | 1.18 (0.52–2.66) | 1.01 (0.42–2.46) |
| Non-Hispanic Black | 13 (42%) | 18 (58%) | 0.97 (0.43–2.19) | 1.12 (0.43–2.91) |
| Non-Hispanic Asian | 4 (57%) | 3 (43%) | 1.80 (0.38–8.42) | — |
| Non-Hispanic Other | 2 (50%) | 2 (50%) | 1.35 (0.18–9.93) | — |

[a] aOR adjusted for age (continuous); age of sexual debut (continuous); number of lifetime sexual partners (reference = 5–9); history of tobacco use (reference = no).

*** Not able to calculate due to sample size.

— Adjusted estimate not shown due to cell counts less than five.

**Table 5**

Self-Reported Race/Ethnicity and Cervical Dysplasia (n = 640).

| | ≥CIN2 (*n*) | ≤CIN1 (*n*) | OR (95% CI) | aOR (95% CI)[a] |
|---|---|---|---|---|
| Self-Reported | | | | |
| Race/Ethnicity | | | | |
| Non-Hispanic White | 87 | 277 | Reference (1.00) | Reference (1.00) |
| Hispanic | 16 | 93 | 0.55 (0.31–0.98) | 0.48 (0.25–0.91) |
| Non-Hispanic Black | 16 | 77 | 0.66 (0.37–1.19) | 0.56 (0.29–1.09) |
| Non-Hispanic Asian | 11 | 29 | 1.21 (0.58–2.52) | 1.75 (0.74–4.15) |
| Non-Hispanic Other | 7 | 6 | 3.71 (1.22–11.35) | 3.63 (1.06–12.51) |

[a]OR adjusted for age (continuous); age at sexual debut (continuous); number of lifetime sexual partners (reference = 5–9); history of tobacco use (reference = no).