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Impact of human papillomavirus vaccination on racial/ ethnic disparities in vaccine-type human papillomavirus prevalence among 14-26 year old females in the U.S.

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Abstract

Background: Low human papillomavirus (HPV) vaccination rates early after introduction, particularly among low income and minority adolescents, may have resulted in disparities in vaccine-type HPV prevalence (types 6, 11, 16, 18). The purpose of this study was to examine racial/ ethnic variations in HPV prevalence, and evaluate how HPV vaccination has affected vaccine-type HPV prevalence across time.

Methods: This study was a retrospective analysis of 6 cycles of the National Health and Nutrition Examination Survey (NHANES) data (2003–2014). Results on HPV status from vaginal samples of 14–26 year old females who responded about HPV vaccination were used to determine HPV

Conflicts of interest:
The authors have no conflicts of interest to report.

prevalence. Prevaccine HPV prevalence was compared to post-licensure prevalence. Racial/ ethnic comparisons were made across time, and models were developed to examine the role of HPV vaccination in observed variations in vaccine-type HPV prevalence.

Results: Among 4,080 females, 29.7% were black, 25.6% were Mexican American, 8.9% were Hispanic, and 35.8% were white. Compared to prevaccine years (2003–2006), vaccine-type HPV did not decrease until late post-licensure years (2011–2014; 14.2% vs. 5.2%, $p<0.001$). Most of the decrease occurred among white females between prevaccine and late post-licensure periods (15.2% vs. 4.1%, $p<0.001$). Although a decrease in prevalence was observed among black females during the same periods (16.9% vs. 9.8%, $p<0.05$), it was not as large as among white females. Prevalence decreased among Mexican Americans (8.2 vs. 4.0, $p>0.05$) during the same periods, but the difference was not significant. Interactions between race and time were significant ($p<0.001$), with uneven vaccination between black and white females contributing to the disparities observed.

Conclusions: HPV vaccination was low in among black and Mexican American females, which contributed to disparities in HPV prevalence. Increasing vaccination among all adolescents, particularly 11–12 year olds, is important because most children this age will not have been exposed.

Keywords

Racial/ ethnic disparities; human papillomavirus disparities; cervical cancer prevention; human papillomavirus vaccination

INTRODUCTION

Human papillomavirus (HPV) is a common infection that can cause anogenital cancers as well as oropharyngeal cancer. Racial/ ethnic disparities have been reported with cervical cancer and other HPV-related cancers disproportionately affecting blacks in the US [1–3]. Cervical cancer rates are also higher among Hispanics [3]. Many of the HPV types associated with these cancers can be prevented through vaccination, preferably before exposure to HPV. The most widely used HPV vaccine in the U.S. between 2006 and 2015 was a quadrivalent HPV vaccine (4vHPV), protecting against 2 high risk HPV types (16 and 18) responsible for 70% of cervical cancer cases and 2 low risk types responsible for 90% of genital warts (types 6 and 11) [4–7]. Since then, a 9-valent vaccine (9vHPV) has been introduced that is expected to reduce high risk HPV types more common among black and Hispanic women.

Recommendations for this vaccine include 2 doses among 11–12 year old females and males, with vaccination allowable as young as 9, and 3 doses for adolescents 15 years and older, up to 26 years [8]. Although the HPV vaccine has been demonstrated to be highly effective in preventing HPV infections and cervical dysplasia, vaccination rates remain low in the U.S. Low vaccination rates are due to a combination of factors, including a lack of strong health provider recommendation to parents of patients, lack of public knowledge or awareness of the HPV vaccine, and concerns with vaccine safety [9–12].

Low vaccination rates among all females led to a series of initiatives and programs, including: health provider education and training on communication about the vaccine, increasing funding for programs offering universal vaccination, educating parents about the vaccine, and administration of HPV vaccines in schools [13–15]. Although these programs have been helpful at increasing HPV vaccination among all vaccine-eligible adolescents, there is some evidence that HPV vaccination was lower among black females early after the vaccine was introduced. HPV vaccine initiation rates were particularly low among black and Hispanic adolescents compared to white teenagers after the vaccine was introduced based on self-report data from the National Survey of Family Growth (NSFG) between 2006 and 2008 [16]. In addition, low rates of catch-up vaccination have been noted among black women and public insurance enrollees 19–26 years old [17]. Low rates of HPV vaccine series initiation combined with low completion of 3-doses of the HPV vaccine series among black females early after vaccine introduction may contribute to racial disparities in vaccine-type HPV prevalence [18–21]. By 2016, national data collected by the National Immunization Survey-Teen (NIS-Teen) indicated that initiation among black female adolescents (70% of 13–17 year olds) and Hispanic female adolescents (72% of 13–17 year olds) surpassed that of white female adolescents (60% of 13–17 year olds) [20, 22]. However, the early disparities in HPV vaccination rates may have had an effect on HPV prevalence by race/ethnicity. The purpose of this study is to examine variations in HPV prevalence by race/ethnicity among female adolescents and young adults, and evaluate how HPV vaccination has affected the prevalence of vaccine-type HPV (types 6, 11, 16, 18) across time.

METHODS

The NHANES survey is a complex, stratified, multistage probability sample that represents ongoing cross-sectional surveys of the civilian noninstitutionalized U.S. population that is nationally representative. Details about sampling and methodology can be found on the National Center for Health Statistics' (NCHS) website [23]. Briefly, it included a household interview followed by physical examinations in a mobile examination center (MEC). Our study used results from a self-collected cervicovaginal swab sample among 14–26 year old females. All self-collected cervicovaginal swab samples were tested for HPV DNA at the CDC [23]. The survey and MEC methods were approved by the NCHS/Centers for Disease Control and Prevention (CDC) research ethics board, and this secondary analysis of the data was exempted from review by the University of Texas Medical Branch Institutional Review Board.

Data from 2-year cycles between 2003 and 2014 were examined. The pre-vaccine years included 2003 to 2006 because the 4vHPV vaccine was approved by the Food and Drug Administration late in 2006, and the Advisory Committee on Immunization Practices recommended it in early 2007 [24]. Early post-licensure years included 2007–2010, and late post-licensure years included 2011–2014. The sample was restricted to female participants who answered either "yes" or "no" to a question about HPV vaccination during the post-licensure years, but was not restricted in prevaccine years. The number of HPV vaccine doses was also assessed among those that reported it.

Demographics, current smoking status, and sexual history were evaluated. Age was dichotomized as “14–17 years old” and “18–26 years old.” We dichotomized age to represent those who needed parental consent for vaccination or study participation and those who were old enough to consent themselves. Education was divided into 3 categories including those who had not graduated from high school (less than high school), those who had graduated or earned a GED, and those who had attended some college. Although education may be associated with age, we included it in the model, as education level is associated with health literacy [25]. Marital status was categorized as: single, living with a partner or “living together,” “married,” and “widowed/ divorced/ separated.” Age at first sex was divided into 4 categories including: “Never had sex,” “<14 years of age,” “14–18 years of age,” and “19+ years of age.” These groupings were developed because sexual initiation at younger ages, particularly before 14 years, is associated with higher risk of sexually transmitted infections and cervical cancer [26].

Linear Array HPV Genotyping Tests (Roche Diagnostics) were used to detect 37 HPV types (6, 11, 16, 18, 26, 31, 33, 35, 39, 40, 42, 45, 51, 52, 53, 54, 55, 56, 58, 59, 61, 62, 64, 66, 67, 68, 69, 70, 71, 72, 73, 81, 82, 83, 84, 89). HPV infections were categorized into 4 groups. Any HPV infection was defined as any positive value for one or more of the 37 HPV types. The other 3 categories included: vaccine-type HPV (types 6, 11, 16, 18), the 2 high-risk vaccine-types (16, 18), and nonvaccine-types (any of the 37 types, excluding types 6, 11, 16, 18). We included these 4 categories to determine whether any observed variations in prevalence were due to vaccination, or were due to natural variation in HPV prevalence. Women were included if they: 1) were 14 to 26 years of age, 2) participated in both the household interview and the MEC examination 3) had an adequate cervicovaginal swab sample, or tested positive for HPV DNA, and 4) either participated in the pre-vaccine cycle, or self-reported, “yes” or “no” to a question about HPV vaccination status for participants in the post-licensure cycles.

Statistical analyses

Demographic characteristics were compared for the overall sample and by race/ ethnicity. The prevalence of all types of all HPV types and vaccine-types were determined for each cycle, and charted for white and black females. Cell sizes were too small for Mexican American and Hispanic women to be included in the detailed examination of HPV prevalence by survey cycle due to low prevalence of vaccine-type HPV in these groups. Bivariate comparisons were done using Rao-Scott Chi-square tests for comparisons of weighted data. All analyses were weighted with MEC weights provided in the dataset, using methods described in detail elsewhere [23].

Multivariable binary logistic regression models using weighted data were built to examine the effects of time and HPV vaccination on vaccine-type HPV prevalence. Prevalence adjusted odds ratios (PaORs) were used as the measure of effect. First, we evaluated the effects of time and HPV vaccination on the association between race/ ethnicity and vaccine-type HPV in models unadjusted for demographic characteristics to examine whether the associations of interest existed for all time periods examined. Three models were run, with the 1st model showing the unadjusted association between race/ ethnicity and vaccine-type

HPV. The 2nd model added a binary time variable, with prevaccine and post-licensure cycles represented. Finally, the 3rd model evaluated the effect of HPV vaccination on the unadjusted association between race/ ethnicity and HPV vaccination. Interactions between race and time were tested for vaccine-type HPV.

We examined 4 models restricted to the sample that was taken in the post-licensure years in order to determine the effect of time and HPV vaccination on racial/ ethnic variations in vaccine-type HPV prevalence, as well as the effects of HPV vaccination on significant associations between time and vaccine-type HPV. The first model controlled for race/ ethnicity, age, education, marital status, smoking status, age at first sex, and history of sexually transmitted infection to evaluate the effect of these variables on vaccine-type HPV prevalence. In model 2, we added the time variable to assess the effects of time on significant associations between race/ ethnicity and vaccine-type HPV. For model 3, we excluded time, and included HPV vaccination to observe its effect on associations between race/ ethnicity and vaccine-type HPV. Finally, in Model 4, time and HPV vaccination were included to examine effects of HPV vaccination on associations between time and vaccine-type HPV prevalence. Significance was determined at $\alpha < 0.05$. Analyses were carried out using SAS® statistical software (SAS Institute, Inc., Cary, NC).

Results

In this sample of young women, close to one-third were less than 18 years old. A high proportion (>40%) of the sample had less than a high school education, but many of the participants (14–18 year olds) would have currently been enrolled in high school (Table 1). The prevalence of current smokers was highest among white women. Age at sexual initiation and history of sexually transmitted infections (STIs) varied significantly by race/ ethnicity. HPV vaccination was lowest among black followed by Mexican American women. HPV vaccination was low overall, with 32.8% having received 1 or more vaccines, but it should be noted that this sample includes data from older females, who had lower rates of catch-up vaccination, and also includes data from early after HPV vaccine introduction when vaccination rates were low in general.

Differences in HPV prevalence were observed by racial/ ethnic group (eTable 1). Racial/ ethnic differences in vaccine-type HPV were marginally significant (p=0.03) in the prevaccine period and post-licensure periods (p<0.001). No differences in the prevalence of high-risk vaccine-type HPV was observed in pre-licensure years, but varied by race/ ethnicity (p=0.03) in post-licensure years.

There was a significant difference for vaccine-type HPV and high risk vaccine-type HPV between prevaccine and late post-licensure periods (Table 2). In analyses stratified by race/ ethnicity, differences in vaccine-type HPV prevalence were not observed between prevaccine and early post-licensure for any racial/ ethnic group. However, differences in vaccine-type HPV prevalence were observed among white women between prevaccine and late post-licensure periods (p<0.001). In addition, a decrease (p=0.03) was observed among young black women during the same period, but not among Hispanic women. Cell sizes for Mexican American women were too small for analysis for high-risk vaccine type in the late

post-licensure years. No variations in “any type HPV” or in nonvaccine type HPV were observed between time periods for any of the racial/ ethnic groups.

Young black women had a higher prevalence of nonvaccine type HPV across time compared to white women (eFigure 1). During the prevaccine cycles, vaccine-type HPV was similar between black and white women, but vaccine-type HPV prevalence remained steady among young black women in early post-licensure cycles while decreasing among young white women. In late post-licensure cycles, vaccine-type HPV prevalence rates decreased among black women, but were twice that of vaccine-type HPV rates among white women in the last survey cycle. Interactions between time and race were significant between prevaccine and post-licensure years ($p<0.001$), as well as in the post-licensure period ($p<0.001$), but were not significant in the prevaccine ($p=0.54$) period (results not shown). These race and time interactions were significant in the prevaccine period to the late post-licensure period ($p<0.001$), as well as in the prevaccine and early post-licensure period ($p=0.04$).

To investigate observed racial/ ethnic differences, we examined the association between race/ ethnicity and vaccine-type HPV in an unadjusted model (eTable 2). Young black women had an elevated prevalence of vaccine-type HPV in this model, which remained significantly greater among black women after including the vaccine period. After including HPV vaccination in the unadjusted model, the association between race and vaccine-type HPV was smaller, but not completely attenuated. This indicates that some of the racial differences in vaccine-type HPV may have been attributable to differences in HPV vaccination rates.

Finally, we modeled the effects of time and HPV vaccination in the post-licensure sample ($n=2,244$) only in order to evaluate how time and vaccination status affected the association between race/ ethnicity and vaccine-type HPV. We observed increased prevalence of vaccine-type HPV among young black women after controlling for demographics and behavioral variables in Model 1 (Table 3). In Model 2, we added the study cycle for NHANES data collection. Although later cycles are associated with a lower prevalence of vaccine-type HPV, young black women continued to have an increased prevalence of vaccine-type HPV compared to young white women. After removing time and including HPV vaccination (Model 3), the association between race and vaccine-type HPV prevalence was reduced and no longer significant, suggesting that the differences in vaccine-type HPV prevalence between black women and white women was attributable to differences in HPV vaccination, and not time. Finally, Model 4 included both time and HPV vaccination. Including HPV vaccination in the model made the association between time and vaccine-type HPV non-significant, suggesting that the decrease in vaccine-type HPV was attributable to HPV vaccination. In a sensitivity analysis, we found that including both age and education in the models did not change the observed associations from models which excluded the education variable.

Discussion

Our results suggest that the lower rates of HPV vaccination by black female adolescents observed early after the vaccine was available in the US contributed to disparities in vaccine-

type HPV prevalence in post-licensure years (2007–2014) that may not have existed during prevaccine years (2003–2006). Our study indicated that vaccination was low among young black females compared to white females. Similar to our findings, a population-based survey collected between 2007 and 2008 indicated HPV vaccine uptake was lower among adolescents of black mothers compared to adolescents of white mothers, even when they had heard of the vaccine [16]. Black and Hispanic females also reported lower rates of healthcare provider recommendation for the HPV vaccine between 2008 and 2013 compared to white females [27]. Recently, efforts to increase vaccination among minority groups, including mitigating cost, education campaigns, and efforts to improve provider recommendation quality, appear to be successful, with vaccine initiation rates no longer differing by race/ ethnicity, although completion continues to remain low among minorities [13–15, 22]. Although vaccination is improving in these groups, it is important to also consider when the vaccine is administered, as our data indicate that young black females may be initiating sex at an earlier age. More than 7% of the NHANES sample that we evaluated had initiated sex before age 14, with young black and Hispanic females initiating sex at an early age. Although the HPV vaccine has been increasingly administered to girls before 13 years of age, almost half of vaccinated females received it after 13 years old in 2012 [28]. Further, 43% of females reported that they had engaged in sexual activity before or during the same year they had received the HPV vaccine [29]. Therefore, vaccination at the recommended age is very important to reducing HPV-related cancer rates and disparities, particularly among black adolescents, who are more likely than whites to initiate vaginal sex between ages 10 to 14 compared to their white counterparts [30].

The results from our study also indicate that much of the decrease in vaccine-type HPV [31] demonstrated in the US could be due mainly to decreases among young white women. We observed that the prevalence of high-risk vaccine-type HPV did not vary significantly by race/ ethnicity during the prevaccine years, similar to evidence reported among unvaccinated young women [32–34]. Although young black women experienced a modest decrease in prevalence of vaccine-type HPV, the decrease did not occur until late post-licensure years (2011–2014). Although these results indicate that HPV-related cervical dysplasia and cervical cancer may decrease in the future, they also suggest that racial/ ethnic disparities will be present among these cohorts.

No significant changes in vaccine-type HPV prevalence were observed between prevaccine and post-licensure years among Mexican Americans or Hispanics. However, both Mexican Americans and Hispanics had relatively low vaccine-type HPV prevalence in the cycles that were examined, and estimates were too low for comparisons to be made in the last cycle of data. Mexican Americans have been shown to have low HPV rates, and US-born Hispanics have been found to have higher rates than non-Hispanic whites, but lower than non-Hispanic black women, similar to our findings for any HPV types [35]. Additional information about how HPV vaccination is affecting HPV prevalence among Hispanics is needed, as cervical cancer incidence is elevated for this population in the US compared to non-Hispanic whites [3].

HPV vaccine series completion was lowest among racial/ ethnic minorities, which could explain why HPV prevalence did not decrease as quickly among young black women as

among white women. Although the HPV vaccine has been shown to have non-inferior immunogenicity when only 2 doses are administered in female adolescents compared to 3 doses among older women, immunogenicity for HPV type 18 may not be non-inferior when comparing 2 doses to 3 doses among younger girls after 2 years [36]. Since girls often receive the vaccine later than the recommended age of 11–12 years old, and the vaccine is not effective against established infections, it is possible that it will take several decades for disparities in HPV-related cancers to disappear, even as cervical cancer incidence decreases [28, 37].

This study demonstrates that the HPV vaccine, not time, is responsible for the observed decreases in vaccine-type HPV prevalence. Reductions in HPV prevalence, cervical abnormalities, and genital warts following introduction of HPV vaccination has been documented both nationally and internationally [38–41]. Our study adds to the literature by demonstrating the direct effect of HPV vaccination on population level vaccine-type HPV prevalence. Further, our study indicated decreases in vaccine-type HPV were a result of HPV vaccination and not due to incidental decreases over time. Although some herd immunity has been demonstrated with the current HPV vaccination rates [33], high HPV vaccination rates are needed to provide direct protection, and to improve herd immunity to reduce the burden of HPV-related disease in the US.

The primary strength of this study is the utilization of repeated cross-sectional survey data representative of white, black, and Mexican Americans living in the U.S. Pooling data to observe prevaccine and post-licensure years allowed us to examine variations in HPV prevalence between racial/ ethnic groups across time. There were also some limitations. HPV vaccination was limited to self-report, which is subject to recall bias. It has previously been found that accuracy of adolescent HPV vaccination reports by parents may vary by race/ ethnicity, with blacks and Hispanic vaccination reports less likely to agree with their providers' reports.[42] This may have contributed to our finding that HPV vaccination, particularly the receipt of 3 doses, differed by race/ ethnicity. As a result, our analyses likely resulted in a conservative estimate of the effect of vaccination on the association between race/ ethnicity and vaccine-type HPV infection. We also restricted the post-licensure sample to only those who responded to the question about vaccination, while the prevaccine years group was only restricted to those who had adequate cervicovaginal swab samples. This may have introduced some response bias in post-licensure years as it is unknown whether those who did not respond would have been vaccinated similarly to those who did respond.

Although we observed racial disparities in vaccine-type HPV prevalence, it is likely that increasing efforts to vaccinate all eligible adolescents will reduce these disparities. As the 9vHPV vaccine protects against 5 additional types of HPV (types 31, 33, 45, 52, and 58) shown to contribute to a higher proportion of cervical cancer cases among black and Hispanic women, [43] it continues to be important to monitor HPV vaccination uptake and completion among racial/ ethnic minorities in the effort to reduce disparities in HPV-related disease. Healthcare providers should continue to provide strong recommendations to all patients. It is particularly important to recommend to both females and males 11–12 years of age so that they are unlikely to have been exposed before vaccination.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- Lower vaccination in black females contributed to disparities in vaccine-type HPV
- Much of the national decrease in vaccine-type HPV occurred mainly among white women
- HPV vaccination resulted in the observed national decrease in vaccine-type HPV

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Table 1.

Characteristics among women aged 14–26 years with human papillomavirus (HPV) results, NHANES 2003–2014

	Weighted % (95% Confidence Interval)					P-value
	Overall (N=4,080)	White (n=1,459)	Black (n=1,213)	Mexican American (n=1,046)	Other Hispanic (n=362)	
Age						
14–17 years old	32.1 (29.6–34.6)	33.4 (30.2–36.6)	29.9 (26.2–33.7)	31.8 (28.3–35.3)	25.4 (20.7–30.2)	0.02
18–26 years old	67.9 (65.4–70.4)	66.6 (63.4–69.8)	70.0 (66.3–73.8)	68.2 (64.7–71.7)	74.6 (69.8–79.3)	
Education						
Less than high school	43.5 (40.8–46.1)	41.2 (37.9–44.5)	42.3 (37.6–47.1)	56.24 (51.6–60.9)	45.1 (39.3–51.0)	<0.001
High school graduate/GED	16.7 (15.0–18.4)	16.4 (14.2–18.6)	17.5 (14.2–18.6)	16.2 (13.2–19.2)	19.0 (14.6–23.4)	
Some college or above	39.8 (36.8–42.8)	42.4 (38.8–46.1)	40.1 (34.9–45.4)	27.6 (22.3–32.8)	35.9 (29.2–42.6)	
Marital Status						
Never married	72.3 (69.7–74.8)	71.5 (68.0–74.8)	86.4 (83.7–89.1)	61.3 (57.2–65.4)	67.2 (60.3–74.1)	<0.001
Living together	11.3 (9.8–12.8)	11.0 (9.0–13.0)	8.4 (5.9–10.8)	14.8 (11.5–18.0)	14.6 (10.2–18.9)	
Married	14.4 (12.4–16.3)	15.4 (12.7–18.1)	4.4 (2.6–6.1)	21.1 (17.9–24.2)	15.7 (10.3–21.1)	
Widowed/ divorced/ separated	2.1 (1.5–2.7)	2.2 (1.2–3.2)	0.8 (0.1–1.6)	2.9 (1.7–4.1)	2.5 (0.5–4.5)	
Smoking status						
Currently smoking	20.7 (18.8–22.5)	24.8 (22.2–27.5)	13.2 (11.0–15.3)	11.0 (8.4–13.7)	15.3 (10.7–19.8)	<0.001
History of sexual intercourse						
Age at first sex						
Never	26.6 (24.7–28.4)	27.6 (25.1–30.2)	22.9 (19.9–25.9)	29.7 (26.2-33.2)	19.3 (15.4-23.2)	<0.001
<14 years old	7.5 (6.3–8.6)	6.7 (5.2–8.2)	12.1 (9.8–14.5)	5.0 (3.3–6.8)	8.6 (5.2–12.0)	
14–18 years old	55.5 (53.4–57.6)	55.3 (52.3–58.2)	57.6 (53.8–61.4)	52.8 (49.0–56.5)	58.0 (52.4–63.6)	
≥19 years old	10.4 (9.0–11.9)	10.4 (8.4–12.4)	7.3 (5.0–9.6)	12.5 (10.1–14.9)	14.1 (9.3–18.9)	
Current/past STI (genital herpes, gonorrhea, chlamydia) – data from questionnaire + lab	12.6 (11.0–14.2)	8.7 (6.8–10.5)	29.8 (26.3–33.2)	9.6 (6.8–12.4)	16.1 (10.0–22.2)	<0.001
HPV vaccination history						
HPV vaccine (≥1 dose) (Post-licensure only, years 2007–2014)	33.5 (30.4–36.7)	36.4 (32.1–40.7)	27.8 (23.3–32.2)	26.4 (20.5–32.3)	32.5 (25.1–39.9)	0.003
HPV vaccine dose number (Post-licensure only, years 2007–2014)						

	Weighted % (95% Confidence Interval)					P-value
	Overall (N=4,080)	White (n=1,459)	Black (n=1,213)	Mexican American (n=1,046)	Other Hispanic (n=362)	
0 dose	67.2 (64.1–70.4)	64.3 (60.0–68.6)	73.0 (68.6–77.3)	74.1 (68.5–79.8)	68.4 (60.9–76.0)	<0.001
1 dose	5.1 (4.3–6.0)	3.9 (2.8–5.0)	7.2 (5.0–9.4)	8.1 (5.1–11.1)	6.0 (4.0–8.0)	
2 dose	6.6 (5.2–8.0)	6.8 (4.9–8.8)	5.3 (3.4–7.2)	6.3 (3.8–8.8)	7.5 (3.3–11.7)	
3 dose	21.1 (18.2–24.0)	24.9 (20.8–28.9)	14.5 (10.5–18.5)	11.4 (7.5–15.3)	18.0 (12.8–23.2)	

* Data on HPV vaccination was included in survey years 2007–2014.

Table 2.

HPV prevalence in women (14–26 years) in pre- and post-vaccine years, NHANES 2003–2014

	Prevaccine years (2003–2006)	Early post-licensure (2007–2010)		Late post-licensure (2011–2014)	
	% (95% CI)	% (95% CI)	*P*-value*[a]	% (95% CI)	*P*-value*[b]
Overall (n=4,080)					
Any HPV types	42.3 (39.2–45.5)	43.9 (39.8–47.9)	0.55	41.3 (36.2–46.4)	0.72
Vaccine-type (6, 11, 16, 18)	14.2 (12.5–16.0)	12.2 (9.8–14.5)	0.18	5.2 (2.8–7.6)	<0.001
High-risk vaccine-type (16, 18)	10.2 (8.4–12.0)	9.6 (7.3–1.8)	0.64	4.0 (2.1–6.0)	<0.001
Nonvaccine-type (non-6, 11, 16, 18)	40.0 (37.0–43.0)	42.3 (38.2–46.5)	0.37	40.6 (35.5–45.7)	0.83
White (n=1,459)					
Any HPV types	38.8 (35.2–42.3)	41.4 (35.7–47.1)	0.43	36.7 (30.8–42.3)	0.55
Vaccine-type (6, 11, 16, 18)	15.2 (12.4–18.0)	11.0 (7.7–14.3)	0.06	4.1 (1.6–6.5)	<0.001
High-risk vaccine-type (16, 18)	11.0 (8.2–13.9)	9.4 (6.1–12.6)	0.44	3.4 (1.0–5.7)	<0.001
Nonvaccine-type (non-6, 11, 16, 18)	36.5 (33.2–39.8)	40.3 (34.5–46.1)	0.24	36.5 (30.7–42.3)	0.99
Black (n=1,213)					
Any HPV types	58.8 (53.3–64.3)	56.6 (51.3–61.9)	0.56	61.2 (54.5–67.9)	0.57
Vaccine-type (6, 11, 16, 18)	16.9 (12.8–20.9)	19.9 (14.1–25.7)	0.38	9.8 (5.6–14.0)	0.03
High-risk vaccine-type (16, 18)	12.2 (8.7–15.8)	13.6 (8.5–18.8)	0.64	7.0 (4.5–9.4)	0.02
Nonvaccine-type (non-6, 11, 16, 18)	56.0 (51.2–60.9)	53.7 (48.4–59.0)	0.51	59.7 (53.2–66.1)	0.36
Mexican American (n=1,046)					
Any HPV types	39.0 (31.3–46.7)	37.9 (31.1–44.6)	0.82	34.8 (27.4–42.2)	0.44
Vaccine-type (6, 11, 16, 18)	8.2 (6.0–10.3)	8.1 (3.2–13.1)	0.99	4.0 (0.0–8.3)	0.18
High-risk vaccine-type (16, 18)	4.9 (3.7–6.1)	6.2 (2.5–9.9)	0.46	*c*	
Hispanic (n=362)					
Any HPV types	47.3 (36.2–58.5)	49.5 (43.6–55.4)	0.73	46.7 (36.3–57.2)	0.94
Vaccine-type (6, 11, 16, 18)	6.2 (0.85–11.6)	13.1 (8.8–17.3)	0.10	6.3 (2.8–9.8)	0.99
High-risk vaccine-type (16, 18)	5.2 (0.0–10.6)	8.6 (5.0–12.2)	0.36	6.3 (2.8–9.8)	0.74
Nonvaccine-type (non-6, 11, 16, 18)	46.3 (35.4–57.2)	47.0 (39.4–54.6)	0.91	45.4 (35.6–55.2)	0.90

95% CI=95% confidence interval

[*] Rao-Scott's chi-square test comparisons between time periods among non-Hispanic whites, non-Hispanic blacks, Mexican Americans, and Hispanics

[a] P-values for comparisons between prevaccine and early post-licensure period.

[b] P-values for comparisons between prevaccine and late post-licensure period.

[c] Cell size was ≤5 for the outcome, and output could not be obtained, per Center for Disease Control and Prevention (CDC) and National Center for Health Statistics (NCHS) guidelines to protect confidentiality of study participants.

Table 3.

Multivariable logistic regression to examine effect of time, age at first sex, and vaccination on the association between race and vaccine type HPV (6, 11, 16, 18), NHANES 2007–2014 (N=2,244)

	Model 1 PaOR (95% CI) [a]	Model 2 PaOR (95% CI) [a]	Model 3 PaOR (95% CI) [a]	Model 4 PaOR (95% CI) [a]
Study cycle				
2007–2008		-		-
2009–2010		0.76 (0.48–1.22)		0.85 (0.52–1.40)
2011–2012		0.34 (0.16–0.72)		0.41 (0.18–0.92)
2013–2014		0.32 (0.14–0.74)		0.44 (0.18–1.04)
Race/ ethnicity				
White	-	-	-	-
Black	1.80 (1.07–3.03)	1.91 (1.12–3.26)	1.62 (0.98–2.68)	1.68 (1.00–2.82)
Mexican American	1.01 (0.48–2.13)	0.97 (0.47–1.99)	0.92 (0.45–1.88)	0.90 (0.45–1.80)
Hispanic	1.29 (0.69–2.39)	1.30 (0.69–2.47)	1.29 (0.69–2.42)	1.33 (0.69–2.53)
Age				
14–17 years	0.69 (0.33–1.42)	0.61 (0.30–1.27)	0.75 (0.36–1.60)	0.67 (0.31–1.46)
18–26 years	-	-	-	-
Education				
<high school	0.80 (0.42–1.54)	0.73 (0.38–1.40)	0.75 (0.39–1.44)	0.71 (0.37–1.37)
High school diploma or GED	1.39 (0.77–2.49)	1.28 (0.72–2.29)	1.18 (0.65–2.13)	1.13 (0.63–2.02)
Some college or college graduate	-	-	-	-
Marital status				
Married	-	-	-	-
Living with partner	1.82 (0.74–4.50)	1.93 (0.80–4.66)	1.98 (0.80–4.96)	2.06 (0.84–5.08)
Single	2.46 (1.20–5.03)	2.50 (1.27–4.92)	2.95 (1.45–6.00)	3.00 (1.50–6.01)
Separated/ divorced/ widowed	2.05 (0.62–6.82)	2.04 (0.66–6.28)	2.46 (0.73–8.27)	2.45 (0.78–7.73)
Smoking status				
Not current smoker	-	-	-	-
Current smoker	2.03 (1.16–3.53)	2.00 (1.17–3.41)	1.96 (1.15–3.34)	1.94 (1.15–3.28)

	Model 1 PaOR (95% CI) [a]	Model 2 PaOR (95% CI) [a]	Model 3 PaOR (95% CI) [a]	Model 4 PaOR (95% CI) [a]
Age at first sex				
Never had sex	-	-	-	-
<14 years of age	9.27 (3.38–25.41)	9.32 (3.38–25.71)	10.10 (3.73–27.35)	10.13 (3.70–27.74)
14–18 years of age	17.13 (6.35–46.21)	17.67 (6.63–47.10)	18.52 (6.90–49.70)	19.01 (7.08–51.03)
19+ years of age	10.89 (3.37–35.20)	10.53 (3.32–33.40)	10.60 (3.36–33.48)	10.50 (3.37–32.71)
History of sexually transmitted infection (STI)				
No history	-	-	-	-
At least 1 STI in past or current lab positive	1.70 (1.11–2.61)	1.63 (0.97–2.74)	1.65 (1.05–2.57)	1.63 (0.96–2.77)
HPV vaccine initiation				
0 doses			-	-
1+ doses			0.27 (0.15–0.48)	0.32 (0.17–0.60)

95% CI=95% confidence interval

[a] PaOR = prevalence adjusted odds ratio, odds ratios are weighted and adjusted for all variables shown in each column, with the exception of shaded out variables.

All models controlled for age, education, marital status, smoking status, age at first sex, and history of sexually transmitted infection.