Distribution of Vaccine-Type Human Papillomavirus Does Not Differ by Race or Ethnicity Among Unvaccinated Young Women

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Abstract

Background: Previous studies have demonstrated racial and ethnic differences in the distribution of human papillomavirus (HPV) types among adult women with cervical precancers. The aim of this study was to determine whether the distribution of vaccine-targeted HPV types varies by race/ethnicity among unvaccinated young women.

Materials and Methods: A secondary analysis was performed using data from four studies of sexually experienced, unvaccinated, 13–26-year-old women. Participants completed surveys and provided a cervicovaginal swab for HPV DNA testing. Multivariable logistic regression analyses were performed to examine whether race, ethnicity, and other factors were associated with type-specific HPV infection among the overall sample and among HPV-infected participants. Models controlled for age, HPV knowledge, sexual behaviors, substance use, and random study effect.

Results: The mean age of participants ($N=841$) was 19.3 years; 64.4% were black and 8.9% Hispanic. Black women were more likely than white women to be positive for ≥ 1 HPV type (odds ratio [OR] 1.83, 95% CI 1.30–2.58) and Hispanic women were less likely than non-Hispanic women to be positive for ≥ 1 HPV type (OR 0.47, 95% CI 0.24–0.92). However, among all young women and HPV-infected women, neither race nor ethnicity was associated with positivity for HPV types targeted by the following vaccines: 2-valent (HPV16 and/or 18), 4-valent (HPV6, 11, 16, and/or 18), or 9-valent (HPV6, 11, 16, 18, 31, 33, 45, 52, and/or 58).

Conclusion: The prevalence of HPV types targeted by the 2-valent, 4-valent, and 9-valent vaccines did not differ by race or ethnicity among all and among HPV-infected women in this sample.

Keywords: race, ethnicity, human papillomavirus, epidemiology, vaccine

Introduction

THE OVERALL PREVALENCE of human papillomavirus (HPV) is estimated to be 42.5% among reproductive-age women in the U.S.¹ Of the more than 190 types of HPV identified, ~13 are classified as high risk (cancer-associated).² Persistent infection with two high-risk types, HPV16 and 18, causes ~70% of cervical cancers and 50% of high-grade cervical lesions in women.^{3,4} Three HPV vaccines are currently licensed, all of which protect against HPV16 and HPV18. The quadrivalent vaccine also protects against HPV6 and HPV11, and the 9-

valent vaccine also protects against HPV6, HPV11, HPV31, HPV33, HPV45, HPV52, and HPV58. These vaccines have the potential to greatly reduce risk of HPV infection and mortality due to cervical cancer and other HPV-associated cancers.⁵

Black and Hispanic women have higher rates of HPV infection, cervical cancer incidence, and cervical cancer mortality compared with other racial and ethnic groups.^{1,6–10} The risk of death from cervical cancer for black women is twice that of white women in the U.S., and the 5-year survival rate for all stages of cervical cancer is 61% for black women compared with 72% for white women.⁹

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Primary prevention through vaccination offers the potential to reduce disparities in cervical cancer incidence and mortality, as long as access to vaccination, uptake of vaccines, vaccine immunogenicity, vaccine efficacy, and the prevalence of vaccine-type HPV in the cervix and in cervical cancers are similar across racial and ethnic groups. Previous studies have shown that HPV vaccines have comparable immunogenicity and efficacy in individuals of different race and ethnicity.^{11,12} However, a recent study demonstrated that the most common HPV types detected in African American women with cervical intraepithelial neoplasia (CIN) differed from those found in non-Hispanic white women.¹³ Among African American women, HPV types 16 and 18 were significantly less likely to be identified in CIN lesions. The most frequent high-risk HPV genotypes detected among European Americans were 16, 18, 56, 39, and 66, compared with types 33, 35, 45, 58, and 68 in African Americans. Another study demonstrated that black and Hispanic women have a lower proportion of HPV16/18-associated lesions in CIN2, CIN3, or adenocarcinoma *in situ* compared with non-Hispanic white women.¹⁴ Similarly, a multisite vaccine impact monitoring project demonstrated that HPV16/18-associated high-grade cervical lesions were less common in non-Hispanic blacks and Hispanics compared with non-Hispanic Whites.¹⁵

As the above studies enrolled adult women with abnormal cytology or CIN, who may or may not have been vaccinated, it is unknown whether there are racial differences in the distribution of HPV genotypes in unvaccinated adolescents and young adults who are in the age group targeted for vaccination. Therefore, we analyzed data from four studies of young women in the age group targeted for vaccination, with the following aim: to determine if race, ethnicity, and other factors were significantly associated with vaccine-type HPV infection among unvaccinated young women. Associations were examined among all women and among a subset of women who were HPV-infected.

Materials and Methods

Participants were recruited for these studies from a hospital-based primary care clinic, a sexually transmitted disease clinic, and a health department clinic within the metropolitan area of a Midwest city. Three of the studies recruited from all three sites and one of the studies recruited only from the primary care clinic (Table 1). The four parent studies and this secondary analysis were approved by the hospital's Institutional Review Board.

Young women who were enrolled in each of the studies completed a self-administered survey assessing demographic factors, HPV knowledge, gynecological history and behaviors; items used for analysis were identically worded in the four studies.^{16–18} Race was defined as black, white, or other, and ethnicity was defined as Hispanic and non-Hispanic. Each woman also provided a cervicovaginal swab for HPV DNA testing, which was performed using the Roche Linear Array Assay, a PCR amplification/detection system that uses pooled L1 primers and a reverse-line blot detection step to identify 36 different HPV genotypes (Roche Molecular Systems, Alameda, CA).¹⁹ The HPV types detected included: 6, 11, 16, 18, 26, 31, 33, 34, 35, 39, 40, 42, 44, 45, 51, 52, 53, 54, 56, 58, 59, 61, 62, 66, 67, 68, 69, 70, 71, 72, 73, 81, 82, 83, 84, and 89. "Infection" was defined as detection of the specific HPV type in the Linear Array Assay. Details of survey administration and HPV DNA testing are provided in previous articles.^{16–18}

Across the four studies, a total of 1557 young women were recruited. Data from the participants who had had sexual contact and had not received an HPV vaccine ($n=841$) were eligible for these analyses. Vaccinated girls were not included in this study; however, vaccination rates increased from 0% at the beginning of the study conducted in 2006–2007 (before the vaccine was widely available) to 71.3% in the study conducted in 2013–2014. Survey and HPV DNA data from the four studies were merged. The three primary outcome variables were infection with at least one of the HPV types found in the 2-valent vaccine (HPV16, 18), the 4-valent vaccine (HPV6, 11, 16, 18), and the 9-valent vaccine (HPV6, 11, 16, 18, 31, 33, 45, 52, 58). Additional outcome variables were infection with at least one HPV type and at least one high-risk HPV type included in one of the three HPV vaccines; the latter was not included in multivariable analyses.

We used chi square tests, Wilcoxon rank-sum tests, and *t*-tests to determine whether the primary predictor variables, race, and ethnicity, were associated with the following outcomes: overall (≥ 1 type) and type-specific (2-valent, 4-valent, and 9-valent) HPV infection. We used the same univariable methods to determine whether other variables that were potential risk factors for HPV (HPV knowledge, gynecological history, sexual behaviors, and smoking) were associated with overall and type-specific HPV infection. We performed multivariable logistic regression to determine whether race and ethnicity were associated with (a) overall (≥ 1 type) and type-specific (2-valent, 4-valent, and 9-valent) HPV infection among all participants, and (b) type-specific HPV infection among HPV-infected participants, controlling for the covariates that

TABLE 1. CHARACTERISTICS OF POPULATION SAMPLES IN FOUR STUDIES OF THE DISTRIBUTION OF HPV TYPES

Study	Data collection (time period, years)	Participant age range (years)	Total recruited (N)	Eligible for secondary analysis (N) ^a	Eligible participants who were HPV-infected (N)
1	2006–2007	13–26	409	360	245
2	2009–2010	13–26	409	167	122
3	2008–2010	13–21	339	199	135
4	2013–2014	13–26	400	115	78
Total			1557	841	580

^aEligible for inclusion in the secondary data analysis were those young women who were sexually experienced and had not received an HPV vaccine.

were significantly associated with HPV outcomes in univariable analyses. Odds ratios (OR) and 95% confidence intervals (CI) were calculated. Random effects modeling was also conducted to account for possible heterogeneity among the four studies; the multivariable logistic regression models included a random effect study if the effect was significant.

Results

Among the 841 participants who were unvaccinated and thus eligible for the analyses, the mean age was 19.2 years (range 13–26), 60.8% reported their race as black, 8.7% reported their ethnicity as Hispanic, 53% reported Medicaid insurance, 19.4% reported first age of sexual contact as ≤ 13 years of age, 81.9% reported at least two lifetime sexual partners, and 5.8% reported at least two partners in the past 3 months. More detailed results from the individual studies, including differences between vaccinated and unvaccinated women, can be found in previous articles.^{16–18,20,21}

HPV results were as follows: 69.0% were positive for at least one HPV type, 56.1% for at least one high-risk type, 21.2% for at least one 2-valent vaccine type (15.8% for HPV16 and 8.0% for HPV18), 27.2% for at least one 4-valent type, and 41.5% for at least one 9-valent vaccine type (Table 2). Among HPV-infected women, 81.4% were positive for at least one high-risk type, 30.7% for at least one 2-valent type (22.9% for HPV16 and 11.6% for HPV18), 39.5% for at least one 4-valent type, and 60.2% for at least one 9-valent type.

The results of univariable analyses examining HPV prevalence by race and ethnicity are shown for all participants and for HPV-infected participants in Table 2. Among all participants, HPV prevalence was higher among black compared with white women and women of other races: ≥ 1 HPV type was detected in 73.0% of black, 62.5% of white, and 63.0% of other women ($p=0.007$). High-risk HPV prevalence was also significantly higher among black compared with white women: 60.7% of black, 49.2% of white, and 48.0% of other

women ($p=0.004$). Although HPV16/18 was detected in a higher proportion of black than white women, the difference was not statistically significant: 22.1% of black, 18.8% of white, and 21.9% of other women ($p=0.55$). Rates of HPV were also significantly higher in black compared with white women for several individual HPV types, including HPV56, 68, 82, and 89, but numbers were small (data not shown).

Among all participants, prevalence of any HPV type did not differ between Hispanic and non-Hispanic women: ≥ 1 HPV type was detected in 69.9% of Hispanic and 68.7% of non-Hispanic women ($p=0.83$). Although HPV16/18 was detected in a slightly lower proportion of Hispanic than non-Hispanic women, the difference was not statistically significant. Among HPV-infected participants, prevalence of any HPV type did not differ significantly in black compared with white young women or in Hispanic compared with non-Hispanic women.

In multivariable models (Table 3), among all participants, black race was associated with a higher odds of infection with at least one HPV type (OR 2.30, 95% CI 1.25–4.24), and Hispanic ethnicity was associated with a lower odds of infection with at least one HPV type (OR 0.47, 95% CI 0.24–0.92). However, among all participants as well as HPV-infected participants, race and ethnicity were not associated with infection with a 2-valent, 4-valent, or 9-valent vaccine-targeted type, and no covariates were significantly associated with HPV outcomes in the final models. There was evidence of a random study effect for all models, except one, in which the outcome variable was ≥ 1 HPV type among all women.

Discussion

A high proportion of unvaccinated young women 13–26 years of age in this study sample were infected with HPV types that could have been prevented by vaccination: 21.2% with at least one 2-valent type, 27.2% with at least one 4-valent type, and 41.5% with at least one 9-valent type. These findings underscore the importance of vaccinating women at the recommended age

TABLE 2. HPV PREVALENCE AMONG ALL YOUNG WOMEN ($N=841$), AND AMONG HPV-INFECTED YOUNG WOMEN ($N=580$), 13–26 YEARS OF AGE BY RACE AND ETHNICITY

	<i>All</i>		<i>Black</i>		<i>White</i>		<i>Other</i>			<i>Hispanic</i>		<i>Non-Hispanic</i>		
<i>Variable</i>	N ^a	%	N	%	N	%	N	%	p ^b	N	%	N	%	p ^b
HPV prevalence among all young women (<i>N</i> =841)														
Any HPV	580	69.0	373	73.0	160	62.5	46	63.0	0.007	51	69.9	524	68.7	0.83
High-risk HPV ^c	472	56.1	310	60.7	126	49.2	35	48.0	0.004	39	53.4	428	56.1	0.66
2-valent vaccine-type HPV ^d	178	21.2	113	22.1	48	18.8	16	21.9	0.55	13	17.8	161	21.1	0.51
4-valent vaccine-type HPV ^e	229	27.2	151	29.6	59	23.1	18	24.7	0.14	16	21.9	209	27.4	0.31
9-valent vaccine-type HPV ^f	349	41.5	227	44.4	93	36.3	28	38.4	0.09	30	41.1	314	41.2	0.99
HPV prevalence among HPV-infected young women (<i>N</i> =540)														
Any HPV	580	100	373	100	160	100	46	100	N/A	51	100	524	100	N/A
High-risk HPV ^c	472	81.4	310	83.1	126	78.8	35	76.1	0.31	39	76.5	428	81.7	0.36
2-valent vaccine-type HPV ^d	178	30.7	113	30.3	48	30.0	16	34.8	0.81	13	25.5	161	30.7	0.44
4-valent vaccine-type HPV ^e	229	39.5	151	40.5	59	36.9	18	39.1	0.74	16	31.4	209	39.9	0.23
9-valent vaccine-type HPV ^f	349	60.2	227	60.9	93	58.1	28	60.9	0.84	30	58.8	314	59.9	0.88

^aTotal N : numbers in columns to the right may not add up to the total due to missing data on race and ethnicity.

^bChi square test used to examine associations between HPV and race or ethnicity. Bold values indicate $p < .05$.

^cHigh-risk defined as HPV-16, 18, 26, 31, 33, 35, 39, 45, 51, 52, 53, 56, 58, 59, 67, 68, 70, 73, 82, and/or 89.

^dHPV-16 and/or 18.

^eHPV-6, 11, 16, and/or 18.

^fHPV-6, 11, 16, 18, 31, 33, 45, 52, and/or 58.

TABLE 3. ASSOCIATIONS BETWEEN RACE, ETHNICITY, AND HPV: RESULTS OF MULTIVARIABLE LOGISTIC REGRESSION MODELS THAT CONTROL FOR RANDOM STUDY EFFECT (IF EVIDENCE FOR A RANDOM EFFECT) AND OTHER COVARIATES ASSOCIATED WITH EACH OUTCOME VARIABLE IN UNIVARIABLE ANALYSES

Outcome variable		OR	95% CI	p ^a	Model controls for the following variables ^b
All participants ≥1 HPV	Race				Insurance plan ($p=0.04$) No. of male sexual partners, lifetime ($p<0.0001$)
	Black vs. Other	2.30	1.25–4.24	0.008	
	Black vs. White	1.83	1.30–2.58	0.0006	
	Other vs. White	0.79	0.43–1.46	0.46	
	Ethnicity				
	Hispanic vs. Non-Hispanic	0.47	0.24–0.92	0.03	
2v ^c	Race				No. of male sexual partners, past 3 months ($p=0.027$)
	Black vs. Other	0.91	0.46–1.83	0.80	
	Black vs. White	1.11	0.75–1.66	0.60	
	Other vs. White	1.22	0.60–2.50	0.59	
	Ethnicity				
	Hispanic vs. Non-Hispanic	1.09	0.53–2.24	0.82	
4v ^d	Race				No. of male sexual partners, past 3 months ($p=0.04$)
	Black vs. Other	1.19	0.61–2.30	0.61	
	Black vs. White	1.24	0.86–1.80	0.25	
	Other vs. White	1.05	0.53–2.07	0.90	
	Ethnicity				
	Hispanic vs. Non-Hispanic	1.03	0.53–2.01	0.92	
9v ^e	Race				No. of male sexual partners, lifetime ($p=0.0003$)
	Black vs. Other	1.46	0.81–2.61	0.21	
	Black vs. White	1.37	0.98–1.91	0.07	
	Other vs. White	0.94	0.52–1.71	0.83	
	Ethnicity				
	Hispanic vs. Non-Hispanic	0.61	0.34–1.09	0.09	
HPV-infected participants					
2v ^c	Race				None
	Black vs. Other	0.66	0.31–1.43	0.29	
	Black vs. White	0.86	0.55–1.33	0.49	
	Other vs. White	1.29	0.59–2.86	0.52	
	Ethnicity				
	Hispanic vs. Non-Hispanic	1.40	0.65–3.02	0.39	
4v ^d	Race				Lifetime smoking history ($p=0.02$)
	Black vs. Other	0.79	0.37–1.67	0.53	
	Black vs. White	0.74	0.46–1.17	0.20	
	Other vs. White	0.94	0.44–2.01	0.87	
	Ethnicity				
	Hispanic vs. Non-Hispanic	1.58	0.76–3.28	0.22	
9v ^e	Race				None
	Black vs. Other	0.93	0.45–1.92	0.84	
	Black vs. White	0.98	0.65–1.47	0.91	
	Other vs. White	1.05	0.50–2.22	0.89	
	Ethnicity				
	Hispanic vs. Non-Hispanic	0.99	0.51–1.93	0.97	

Evidence of random study effect was found for all models, except for the first model, in which the outcome variable was ≥1 HPV type among all women.

^aBold values indicate $p<.05$.

^bVariables that were associated with outcome variable in univariable analyses: p -value indicated is the p -value from the univariable analysis (conducted using chi square tests, Wilcoxon rank-sum tests, and t -tests), but none of the variables in this column were significantly associated with outcome variables in the final multivariable models.

^c2-valent vaccine-type HPV (HPV-16 and/or -18).

^d4-valent vaccine-type HPV (HPV-6, -11, -16, and/or -18).

^e9-valent vaccine-type HPV (HPV-6, -11, -16, -18, -31, -33, -45, -52, and/or -58).

of vaccination, 11–12 years, which generally precedes first HPV exposure.²² Numerous studies have examined effective strategies for improving HPV vaccine uptake; strategies are summarized in recent reports by the Centers for Disease Control and Prevention, the President's Cancer Panel, and the National Vaccine Advisory Committee. These include reducing missed

clinical opportunities to administer HPV vaccines, educating parents and adolescents about HPV vaccination, vaccinating in alternative settings such as schools, increasing parents' and adolescents' acceptance of HPV vaccines, maximizing access to HPV vaccination services, and promoting global HPV vaccine uptake.^{23–25} Such strategies are especially important for low-

income, minority youth such as those enrolled in these studies, to reduce disparities in morbidity and mortality due to HPV-associated cancers. Furthermore, given that the licensed vaccines are highly effective in preventing HPV infection, and substantial decreases in vaccine-type HPV infections have been demonstrated to occur after vaccine introduction,¹⁸ these findings suggest that HPV vaccination with the currently licensed vaccines could have a substantial impact on the prevalence of HPV types that cause cervical cancers in similar populations.

Among all participants, infection with HPV overall, irrespective of type, was higher in black compared with white women in univariable and multivariable analyses, which is consistent with some previous studies.^{1,16} In one study, the higher rates of HPV infection in black women appeared to be due, in part, to racial differences in marital status and mean income, and the authors noted that other contributing factors may have included unmeasured differences in social and economic assets not reflected by income, such as wealth, community resources, and access to health services and education.⁶ Most importantly, however, in this study race and ethnicity were not associated in univariable or multivariable analyses with vaccine-type HPV infection (*i.e.*, types in the 2-, 4-, or 9-valent vaccines), among all participants and among HPV-infected participants. In addition, no covariates were significantly associated with HPV outcomes in multivariable models.

These findings suggest that women of different race and ethnicity should benefit similarly from vaccination with respect to prevention of the HPV types targeted by the currently available vaccines, although not necessarily with respect to prevention of other HPV types. As noted previously, some studies have demonstrated that the distribution of HPV types differs by race and ethnicity in women with cervical precancers.^{13–15} Our findings may have differed because previous studies enrolled adult women with abnormal cytology or CIN, who may have been vaccinated, whereas our study enrolled adolescent and young adult women who were unvaccinated. Additional research is warranted to examine whether susceptibility to the oncogenic potential of specific HPV types is affected by race or ethnicity.

This study had several limitations. The number of Hispanic participants was relatively low, limiting the power to detect differences by ethnicity in HPV types. The number of women positive for individual HPV genotypes was also low, limiting power to determine differences in individual types by race or ethnicity. The outcome measures are not independent as there is overlap between the women who were infected with HPV types found in the 2-valent, 4-valent, and 9-valent vaccines. Finally, this study was conducted in a population of predominantly low-income minority women, limiting the generalizability of the findings to other populations.

Conclusion

This study provides novel evidence that vaccine-type HPV prevalence does not differ by race or ethnicity in a sample of 13–26-year-old women who stand to benefit from HPV vaccination. Although larger, population-based studies are needed to confirm these findings and explore the prevalence of individual types by race and ethnicity (and similar studies are needed in male populations), the findings provide reassurance that vaccination should prevent HPV infection in similar proportions of women of different races and ethni-

cities. To decrease racial and ethnic disparities in HPV-related cancers, ensuring that all women have excellent access to vaccination and uptake of vaccines will be important.²⁶ Continued monitoring of the epidemiological changes in the prevalence of HPV infection, precancers, and cancers in diverse populations will also be important to assess the impact of vaccination and ensure that disparities in outcomes are narrowing.

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Author Disclosure Statement

Dr. Kahn has co-chaired two NIH-funded HPV vaccine clinical trials in HIV-infected individuals, for which Merck & Co., Inc., provided vaccine and immunogenicity titers. Dr. Kahn chaired a grant review committee for the Society for Adolescent Health and Medicine evaluating public health demonstration project proposals to improve adolescent vaccination; grant funding for this program was from Merck, Inc. Dr. Franco has served as occasional advisor to companies involved with HPV vaccination (Merck, GSK) and HPV and cervical cancer diagnostics (Roche, BD, Qiagen). His institution has received unconditional funding from Merck for investigator-initiated studies carried out in his unit. Dr. Brown has received honoraria and grant support from Merck, and his institution Indiana University and Merck have a confidential agreement that pays the University based on certain landmarks of vaccine development; Dr. Brown receives a portion of this money as income. For the remaining authors, no competing financial interests exist.

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