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Epidemiology and risk factors for human papillomavirus infection in a diverse sample of low-income young women

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Abstract

Background—Two HPV vaccines prevent infection with HPV-16 and HPV-18, high-risk (cancer-associated) HPV types which together cause approximately 70% of cervical cancers; one vaccine also prevents HPV-6 and HPV-11, which together cause approximately 90% of anogenital warts. Defining type-specific HPV epidemiology in sexually experienced women will help estimate the potential clinical benefits of vaccinating this population.

Objectives—To examine HPV epidemiology in a diverse sample of sexually experienced women, and to determine factors associated with high-risk HPV and vaccine-type HPV (HPV-6, -11, -16, -18).

Study Design—Cross-sectional study of 13-26 year-old women (N=409) who completed a questionnaire and provided a cervicovaginal swab. Swabs were genotyped for HPV using PCR amplification. Logistic regression models were used to determine whether participant characteristics, knowledge, and behaviors were associated with high-risk and vaccine-type HPV.

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Dr. Rosenthal is a co-Principal Investigator on an investigator initiated grant funded by Merck and serves as a research consultant/collaborator on a Merck-sponsored research project and on a Merck advisory board.
Dr. Kahn is a co-Principal Investigator on an NIH-funded HPV vaccine clinical trial in HIV-infected adolescents, for which Merck, Inc., is providing HPV vaccine and immunogenicity testing.

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Results—Most women (68.4%) were positive for ≥ 1 HPV type, 59.5% were positive for ≥ 1 high-risk type, 33.1% were positive for ≥ 1 vaccine-type HPV, and 3.5% were positive for both HPV-16 and -18: none was positive for all four vaccine types. In adjusted logistic regression models, Black race (OR 2.03, 95% CI 1.21-3.41) and lifetime number of male sexual partners (OR 4.79, 95% CI 2.04-11.23 for ≥ 10 vs. ≤ 1 partner) were independently associated with high-risk HPV infection.

Conclusions—HPV prevalence was very high in this sample of sexually active young women, but < 5% were positive for both HPV-16 and HPV-18, suggesting that vaccination could be beneficial for many individual women who are sexually experienced.

Keywords

human papillomavirus; vaccination; cervical cancer; sexually transmitted infection; epidemiology

Background

Human papillomavirus (HPV) is a common sexually transmitted infection (STI) that may cause cervical cancer and other malignancies.[1, 2] Two HPV vaccines have recently been licensed and are recommended by national immunization programs in at least 15 countries.[3] Both vaccines prevent HPV-16 and HPV-18, high-risk (cancer-associated) types that together cause approximately 70% of cervical cancer, and one of the vaccines also prevents HPV-6 and HPV-11, which cause approximately 90% of anogenital warts.[4, 5] Thus, vaccination has the potential to substantially reduce the global burden of cervical cancer and other HPV-associated diseases.[6, 7]

Prophylactic HPV vaccines are most effective if given prior to HPV exposure;[8-10] therefore, there is consensus among national immunization programs that HPV vaccines should be targeted to girls who are < 15 years of age.[3] However, many countries also recommend "catch-up" vaccination of older women. In the U.S., national guidelines state that HPV vaccination should be targeted to 11-12 year-old girls, but that previously unvaccinated 13-26 year-old women should also receive the vaccine, regardless of sexual experience.[11] Because many sexually active women have already been exposed to HPV, the impact of widespread vaccination in this population is not well-defined. Therefore, understanding type-specific HPV epidemiology in sexually experienced women is important to help estimate the potential clinical benefit and cost-effectiveness of vaccinating this population. It is particularly important to understand the epidemiology of HPV in minority and low-income young women, who may be at higher risk for HPV infection or cervical cancer.[12-14]

Objectives

The aims of this study were to determine the prevalence of high-risk and vaccine-type HPV infection in a diverse sample of low-income women, and to examine whether participant sociodemographic characteristics, HPV knowledge, and behaviors are associated with high-risk and vaccine-type HPV infection.

Study Design

Young women 13-26 years of age (N=409) who had had sexual contact were recruited between October 2006 and May 2007 from three primary care clinics: a hospital-based Teen Health Center (N=268), obstetrics and gynecology clinic in a community health center affiliated with the city's Health Department (N=126), and sexually transmitted disease clinic also affiliated with the Health Department (N=15).[15] Of 418 women who were approached by the research coordinators, 409 (98%) provided informed consent. All participants completed a self-

administered questionnaire that assessed demographic factors, HPV knowledge, gynecologic history, and behaviors.[15]

Cervicovaginal swabs were collected from each participant using the same methods at each recruitment site. If the participant had to undergo a speculum examination, then specimens were collected during the exam by the clinician (70% of swabs); otherwise specimens were collected as a blind cervicovaginal swab by either clinicians (25%) or participants (4%). Results were combined based on a previous study of the same clinical population which found reasonable concordance between clinician- and self-testing for HPV DNA using the same method.[16] Samples were genotyped using the Roche Linear Array test, a PCR amplification technique that uses an L1 consensus primer system and a reverse-line blot detection strip to identify 37 different HPV genotypes (Roche Molecular Systems, Alameda, CA).[17] High- and low-level beta-globin controls were positive in 100% of the samples, indicating sufficient DNA and PCR amplification. This assay utilizes one capture probe that cross-reacts with HPV-33, -35, -52, and -58. A sample is considered positive for HPV-52 when it reacts with the HPV-52-cross-reactive probe but not with the remaining three type-specific individual probes.[18] Therefore, reported values for HPV-52 include detection of HPV-52 DNA only and detection of HPV-52 with a cross-reacting type.

The main outcome variables were infection with ≥ 1 high-risk HPV type and infection with ≥ 1 vaccine-type HPV (HPV-6, HPV-11, HPV-16, or HPV-18). Participant data from each site were combined for univariate and multivariable analyses because participants from each recruitment site did not differ in terms of the two outcome variables. We used unadjusted logistic regression modeling to determine whether the following factors were associated with each of the two outcome variables (infection with ≥ 1 high-risk HPV type and infection with ≥ 1 vaccine-type HPV): 1) participant sociodemographic characteristics, 2) HPV knowledge, 3) gynecologic history (history of STI, abnormal Pap test, colposcopy, family history of cervical cancer, number of times pregnant), and 4) behaviors. Behaviors included age of sexual initiation, number of male sexual partners (lifetime, previous three months), condom use with main sexual partner (previous three months, at last sexual intercourse), and smoking in the previous 30 days. Those variables associated with the outcomes at $p < .10$ in univariable analyses were entered into two separate multivariable logistic regression models to identify factors associated independently with high-risk and vaccine-type HPV infection. In the multivariable models, a p value $< .05$ was considered statistically significant.

Results

The mean age of participants was 17.6 years, 62% reported that they were Black, 29% that they were White, and 9% that they were another race or multiracial.[15] Six percent were Appalachian and 6% Latino. Participants were predominantly low-income: just under half (48%) had Medicaid insurance, 38% had no insurance or were unsure, and 14% had private insurance. Forty-nine percent reported a history of an STI, 35% an abnormal Pap test, and 43% a previous pregnancy. Age of first sexual intercourse was ≤ 14 years for 40% of participants and ≤ 18 years for 92% of participants. Eighteen percent reported ≤ 1 lifetime male sexual partners, 15% reported 2 partners, 14% reported 3 partners, 14% reported 4 partners, 25.3% reported 5-9 partners, and 14% reported ≥ 10 partners.

Most women (68.4%) were positive for ≥ 1 HPV type, 59.5% were positive for ≥ 1 high-risk type, and 33.1% were positive for ≥ 1 vaccine-type HPV (Table 1). The most common HPV types detected (prevalence ≥ 8%) were high risk types HPV-52, HPV-16, HPV-18, HPV-59, CP610, HPV-66, HPV-51, HPV-58, and low risk type HPV-6. The percentage of participants positive for the HPV types in the quadrivalent HPV vaccine were as follows: HPV-6 (9.4%),

HPV-11 (2.5%), HPV-16 (17.3%), HPV-18 (11.6%). Although no participant was positive for all four vaccine types, 3.5% were positive for both HPV-16 and HPV-18.

In adjusted logistic regression models, Black race and lifetime number of partners were independently associated ($p < .05$) with high-risk HPV infection (Table 2). Participants who self-identified as Black had twice the odds of high-risk HPV infection compared to those who identified as White. Participants with 10 or more lifetime partners had almost five times the odds of high-risk HPV infection. Only one category of lifetime number of sexual partners (4 vs. ≤ 1) was independently associated with vaccine-type HPV infection.

We also examined univariable associations between all predictor variables and three individual high-risk HPV types - HPV-16, HPV-18, and HPV-52 - to further characterize demographic and behavioral risk factors for these HPV types. The only factor associated significantly with HPV-16 was Black race (OR 2.72, 95% CI 1.33-5.58). No factors were associated with HPV-18. Factors associated with HPV-52 (HPV-52 only, without cross-reacting types) were: number of sexual partners (OR 5.17, 95% CI 1.03-26.02 for 3 vs. ≤ 1 partners), having ever had anal sex (OR 3.01, 95% CI 1.43-6.34) and having had anal sex within the past three months (OR 3.83, 95% CI 1.71-8.56).

Discussion

In this study, we examined the epidemiology of HPV and risk factors associated with high-risk and vaccine-type HPV infection in a racially and ethnically diverse, predominantly low-income population of sexually experienced young women eligible for “catch-up” HPV vaccination. The cross-sectional prevalence of HPV in this sample was high at 68%: this likely underestimates the cumulative lifetime prevalence of HPV as most infections do not persist. Previous studies of predominantly low-income adolescents have demonstrated similarly high prevalence rates.[19, 20] In contrast, the prevalence of HPV in study samples that are more representative of the U.S. population is considerably lower,[21] supporting the growing evidence that poverty is a strong predictor of HPV infection.[14] The high rates of HPV infection in our study sample, combined with evidence that HPV is often acquired soon after sexual initiation,[22] suggest that vaccinating young women prior to sexual initiation must be a priority to maximize both the individual and population-level health benefits of vaccination.

Although almost 70% of participants were positive for HPV, fewer than 20% were positive for HPV-16 or HPV-18, < 5% were positive for both HPV-16 and HPV-18, and none was infected with all four vaccine types. HPV vaccines do not have clinical benefit in women who are infected with vaccine-type HPV at the time of vaccination; however, women who are uninfected with some of those types should derive partial benefit from vaccination.[23] Thus, even in this sample of women, who had a very high overall HPV prevalence rate, the majority would be expected to benefit at least partially from vaccination. The findings do not address the question of whether vaccination of sexually active women will be cost-effective or have population-level benefit, but they provide evidence that individual women are likely to benefit.

In addition to HPV-16 and HPV-18, one of the most commonly identified HPV types was HPV-52. Global epidemiologic studies suggest that HPV-52 prevalence varies across regions but is particularly common in Africa and Asia.[24-26] Although HPV-52 has substantially lower potential to cause cervical neoplasia in comparison to HPV-16 and HPV-18,[27, 28] these high prevalence rates are concerning as HPV-52 is not targeted by the two licensed vaccines. To our knowledge, previous studies have not examined risk factors for HPV-52. In this study, risk factors were similar for HPV-52 as for other high-risk types, with the exception of anal sex, which was associated only with HPV-52. Additional information is needed regarding the

epidemiology of HPV-52, its role in carcinogenesis, and what clinical significance it will have in the vaccination era.

Identification of factors associated with high-risk and vaccine-type HPV infection may help to inform decisions about vaccine delivery and the design of interventions to prevent infection. Our finding that Black race and number of sexual partners were associated with high-risk HPV is consistent with previous studies.[14, 19, 29, 30] However, a previous analysis suggests that higher rates of HPV infection in Black compared to White women are explained in large measure by racial differences in marital status and income. In that study, Black women were more likely than White women to be unmarried and poor, and those socioeconomic factors explained racial differences in HPV infection.[14] Racial differences may also be driven by unmeasured disparities in social and economic assets related to income, such as wealth, community resources, and access to health services and education. The findings suggest that minority and low-income women must have enhanced access not only to HPV vaccines, but also to education and other preventive health services to prevent cervical cancer, including Pap screening. Culturally sensitive educational interventions about HPV vaccines are especially important for minority women, as mistrust of medical professionals and the government may underlie concerns about new vaccines among Black women.[31-34]

In this study sample, measures of recent sexual activity were not independently associated with high-risk HPV infection, whereas lifetime number of sexual partners was associated with infection. Our findings are similar to that of a study of sexually active urban adolescents.[19] Number of lifetime partners may be a more reliable and stable measure of exposure to HPV and other STI in adolescent and young adult women because number of recent partners may not be a reliable marker of sexual history; for example, among those with few recent partners, there may be substantial variation in lifetime number of partners. In contrast to the findings related to high-risk HPV infection, no variables were independently associated with vaccine-type HPV infection, supporting current recommendations for universal vaccination.

This study has several limitations. Because participants were predominantly minority and low-income, and because only sexually experienced women were included, the results are not generalizable to all young women eligible for catch-up HPV vaccination. Sexual and gynecologic history was self-reported, possibly limiting validity. We did not include income measures, limiting our ability to explore associations between poverty, race, and HPV infection. The HPV assay used in this study included several high-risk types that are not included in the HPV tests that are commercially available and commonly used in clinical practice; therefore, HPV prevalence in this study may be higher than if a routine clinical test were used. Despite these limitations, this study provides novel data about the prevalence and risk factors associated with type-specific HPV infection in a diverse sample of sexually experienced women, shortly after the quadrivalent HPV vaccine was licensed in the U.S. Our findings provide support for universal vaccination of sexually experienced 13-26 year-old young women in the U.S.

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Conflict of Interest

Funding: Charlotte R. Schmidlapp Women's Scholar Award, Fifth Third Bank, Cincinnati, OH (J. Kahn, Principal Investigator) R01 AI073713 (J. Kahn, Principal Investigator), and Medical School Training Grant T35 DK 060444 (J. Heubi, Principal Investigator).

Abbreviations

HPV, human papillomavirus; OR, odds ratio; CI, confidence interval; STI, sexually transmitted infection; PCR, polymerase chain reaction.

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Table 1

Type-specific human papillomavirus infection among participants recruited fromaTeen Health Center and Health Department sites

	N (%) positive[1]			P value[2]
	Teen Health Center (N=267)	Health Department (N=138)	All (N=405)	
≥ 1 HPV type	190 (71.2)	87 (63.0)	277 (68.4)	.10
≥ 1 high-risk HPV type	165 (61.8)	76 (55.1)	241 (59.5)	.19
≥ 1 vaccine-type HPV	88 (33.0)	46 (33.3)	134 (33.1)	.94
HPV-6	22 (8.2)	15 (10.9)	37 (9.4)	.75
HPV-11	8 (3.0)	2 (1.5)	10 (2.5)	.90
HPV-16	45 (16.9)	25 (18.1)	70 (17.3)	.75
HPV-18	32 (12.0)	15 (10.9)	47 (11.6)	.74
HPV-26	2 (0.8)	0	2 (.5)	.31
HPV-31	13 (4.9)	9 (6.5)	22 (5.4)	.49
HPV-33	2 (0.8)	1 (0.7)	3 (0.7)	.98
HPV-35	11 (4.1)	6 (4.4)	17 (4.2)	.91
HPV-39	15 (5.6)	7 (5.1)	22 (5.4)	.82
HPV-40	11 (4.1)	1 (0.7)	12 (3.0)	.06
HPV-42	21 (7.9)	5 (3.6)	26 (6.4)	.10
HPV-45	14 (5.2)	3 (2.2)	17 (4.2)	.14
HPV-51	23 (8.6)	10 (7.3)	33 (8.2)	.63
HPV-52				
HPV-52 plus cross reacting type	30 (11.2)	14 (10.1)	44 (10.9)	.74
HPV-52 only	24 (9.0)	9 (6.5)	33 (8.1)	.39
HPV-52 total	54 (20.2)	23 (16.7)	77 (19.0)	.39
HPV-53	18 (6.7)	12 (8.7)	30 (7.4)	.48
HPV-54	9 (3.4)	13 (9.4)	22 (5.4)	.01
HPV-55	9 (3.4)	3 (2.2)	12 (3.0)	.50
HPV-56	6 (2.3)	7 (5.1)	13 (3.2)	.13
HPV-58	20 (7.5)	13 (9.4)	33 (8.2)	.50
HPV-59	33 (12.4)	8 (5.8)	41 (10.1)	.04
HPV-61	17 (6.4)	6 (4.4)	23 (5.7)	.41
HPV-62	17 (6.4)	11 (8.0)	28 (6.9)	.55
HPV-64	2 (0.8)	0 (0)	2 (.5)	.31
HPV-66	24 (9.0)	10 (7.3)	34 (8.4)	.55
HPV-67	11 (4.1)	1 (0.7)	12 (3.0)	.06
HPV-68	19 (7.1)	6 (4.4)	25 (6.2)	.27
HPV-69	0	0	0	--
HPV-70	7 (2.6)	2 (1.5)	9 (2.2)	.45
HPV-71	0	0	0	--
HPV-72	2 (0.8)	3 (2.2)	5 (1.2)	.22
HPV-73	14 (5.2)	5 (3.6)	19 (4.7)	.46
HPV-81	13 (4.9)	1 (0.7)	14 (3.5)	.03
HPV-82	17 (6.4)	3 (2.2)	20 (4.9)	.06
HPV-83	10 (3.8)	4 (2.9)	14 (3.5)	.66
HPV-84	22 (8.2)	7 (5.1)	29 (7.2)	.24
HPV-IS39	0	0	0	--
HPV-CP610	29 (10.9)	12 (8.7)	41 (10.1)	.49

[1] A total of 409 subjects enrolled in the study from all sites. HPV DNA results were missing for a total of 4/409 subjects: 1/268 from the Teen Health Center and 3/141 from the Health Department sites. Results are shown for the 405 subjects (99% of the 409 who enrolled) for whom HPV DNA results were available.

Table 2

Variables associated with high-risk HPV (≥ 1 high-risk HPV type) and vaccine-type HPV (HPV-6, -11, -16, and/or -18) in participants recruited from a Teen Health Center and Health Department sites: results of unadjusted and adjusted logistic regression models

	Unadjusted odds ratios (95% confidence intervals)[1]		Adjusted odds ratios (95% confidence intervals)[2]	
	High-Risk HPV	**Vaccine-Type HPV**	**High-Risk HPV**	**Vaccine-Type HPV**
Race				
Black vs. white	**2.54 (1.57-3.90)**	**1.90 (1.15-3.13)**	**2.03 (1.21-3.41)**	1.19 (0.68-2.14)
Other vs. white	1.26 (0.60-2.65)	0.99 (0.42-2.34)	1.45 (0.62-3.73)	0.77 (0.30-1.99)
Ethnicity				
Appalachian	1.24 (0.531-2.87)	0.80 (0.32-1.96)		
Hispanic	0.73 (0.33-1.65)	0.80 (0.33-1.97)		
Insurance status				
Uninsured vs. insured	1.17 (0.72-1.91)	0.88 (0.53-1.47)		
Medicaid vs. private	1.31 (0.72-2.38)	1.37 (0.72-2.62)		
No insurance vs. private	1.63 (0.88-3.02)	1.17 (0.60-2.28)		
Marital status: unmarried vs. married	0.57 (0.264-1.24)	0.96 (0.42-2.19)		
Age	0.96 (0.90-1.03)	1.03 (0.96-1.101)		
History of STI	**2.15 (1.43-3.24)**	**1.78 (1.17-2.72)**	1.29 (0.78-2.13)	**1.71 (1.02-2.88)**
Pap screening: abnormal vs. normal Pap	1.22 (0.78-1.91)	1.31 (0.83-2.10)		
Colposcopy	1.42 (0.78-2.57)	1.18 (0.64-2.15)		
Family history of abnormal Pap	0.95 (0.56-1.61)	0.58 (0.32-1.05)		
Family history of cervical cancer	0.77 (0.44-1.38)	0.97 (0.52-1.82)		
Pregnancy history: number of pregnancies				
1 vs. 0	0.84 (0.52-1.37)	0.89 (0.54-1.47)	0.96 (0.56-1.64)	1.01 (0.59-1.73)
≥ 2 vs. 0	**0.51 (0.30-0.87)**	**0.53 (0.29-0.98)**	0.57 (0.31-1.05)	0.61 (0.32-1.19)
Knowledge about HPV	2.23 (0.875-5.68)	0.88 (0.34-2.31)		
Age of first sexual intercourse	0.90 (0.81-1.0)	1.1 (0.941-1.17)		
Lifetime number of partners				
2 vs. ≤ 1	1.89 (0.92-3.83)	1.44 (0.67-3.12)	1.81 (0.86-3.80)	1.31 (0.59-2.92)
3 vs. ≤ 1	**3.29 (1.56-6.95)**	1.04 (0.46-2.34)	**3.58 (1.61-7.96)**	0.99(0.42-2.32)
4 vs. ≤ 1	**3.81 (1.78-8.17**	**2.89 (1.35-6.20)**	**3.62 (1.61-8.12)**	**2.53 (1.13 -5.68)**
5-9 vs. ≤ 1	**3.18 (1.672-6.05)**	1.40 (0.70-2.78)	**2.95 (1.44-6.03)**	1.17 (0.54-2.52)
≥10 vs. ≤ 1	**4.71 (2.16-10.28)**	1.36 (0.62-3.00)	**4.79 (2.04-11.23)**	1.22 (0.51-2.92)
Male sexual partners: past 3 months				
1 vs. 0	1.075 (0.59-2.0)	1.1 (0.57-2.12)		
2 vs. 0	1.61 (0.72-3.58)	1.99 (0.88-4.46)		
≥3 vs. 0	1.31 (0.52-3.31)	1.2 (0.46-3.16)		
New male partners: past 3 months				
≥ 1 vs. 0	1.237 (0.81-1.88)	0.994 (0.64-1.54)		
Ever had anal sex with male partner	1.44 (0.89-2.32)	1.42 (0.88-2.29)		
Anal sex with male partner: past 3 months	1.47 (0.81-2.67)	1.32 (0.74-2.37)		
Condom use with main sexual partner				
Never vs. always	1.33 (0.75-2.35)	0.83 (0.45-1.53)		
Occasionally vs. always	1.38 (0.74-2.59)	0.94 (0.49-1.82)		
Mostly vs. always	1.94 (0.97-3.88)	1.42 (0.72-2.81)		
Condom use with main partner last sex	0.75 (0.48-1.16)	1.19 (0.75-1.87)		
Condom use with partner other than main sexual partner				
Never vs. always	0.71 (0.23-2.21)	0.89 (0.27-2.92)		
Occasionally vs. always	1.53 (0.43-5.43)	2.23 (0.70-7.07)		
Mostly vs. always	1.25 (0.46-3.39)	1.95 (0.76-5.02)		
Smoked≥1 cigarettes in past 30 days	0.84 (0.55-1.28)	**0.61 (0.38-.96)**		0.67 (0.39-1.15)

[1] Variables associated withhigh-risk HPV or vaccine-type HPV at $p < .05$ are shown in bold.

[2] Only those variables entered into multivariable models and associated with HPV high risk type or HPV vaccine type at $p < .05$ are shown in these columns.