

Human Papillomavirus Vaccination and Infection in Young Sexual Minority Men: The P18 Cohort Study

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ABSTRACT

We examined the prevalence of infection with human papillomavirus (HPV) and HIV in a cohort of young gay, bisexual, and other men who have sex with men [sexual minority men (SMM)]. HPV vaccination uptake was assessed; HIV antibody testing was performed and genetic testing for oral and anal HPV infection was undertaken. We examined both HPV vaccination and infection in relation to key demographic and structural variables. Participants ($n=486$) were on average 23 years old; 70% identified as a member of a racial/ethnic minority group, and 7% identified as transgender females. Only 18.1% of the participants indicated having received the full dosage of HPV vaccination and 45.1% were unvaccinated. Slightly over half the participants (58.6%) were infected with HPV, with 58.1% testing positive for anal infection and 8.8% for oral infection. HIV seropositivity was associated with infection to oral HPV [adjusted odds ratio (AOR)=4.03] and vaccine-preventable HPV, whereas both neighborhood-level poverty (AOR=1.68) and HIV infection (AOR=31.13) were associated with anal infection to HPV (AOR=1.68). Prevalence of HPV infection is high among unvaccinated young SMM, despite the availability and eligibility for vaccination. HPV infection adds further health burden to these populations and is particularly concerning for those who are HIV positive as HIV infection increases the risk of developing HPV-related cancers. These findings underscore a missed prevention opportunity for an at-risk and underserved population and suggest the need for active strategies to increase HPV vaccination uptake in young SMM before the onset of sexual behavior.

Keywords: HPV, HIV, vaccination, cancer, gay and bisexual men, emerging adulthood

Introduction

HUMAN PAPILLOMAVIRUS (HPV) is the most common sexually transmitted infection among adults in the United States.¹ High-risk (oncogenic) types of HPV, such as HPV 16 and 18, account for most cases of anal, cervical, penile, and oropharyngeal cancers.^{2,3}

The quadrivalent (HPV 61, 11, 16, and 18) and nonavalent (6, 11, 16, 18, 31, 33, 45, 52, and 58) vaccines have been licensed for use in males since 2006 and 2014, respectively,³⁻⁵

and are most effective when the vaccination series is completed before sexual debut and potential exposure to the virus. Given the high prevalence of oncogenic HPV types and the risk for multiple types of cancers in men and women, the HPV vaccine has been recommended for men and women up to 26 years of age in the United States. In 2018, use of the vaccine was expanded to include those 27–45 years of age.⁶ Further, the recommended vaccination age for sexual minority men (SMM) who comprise gay, bisexual, other men who have sex with men, and transgender women is 22–26 years.⁷ Despite

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the availability of an effective vaccine, incidence and prevalence of HPV, particularly oncogenic types, remain high.^{8,9}

Infection with HPV is a population health burden in SMM for several reasons. First, the risk of HPV acquisition and transmission is greater among SMM. Therefore, both the incidence and prevalence of HPV are higher in SMM. In a study conducted by Meites et al.,⁷ of 834 young SMM, anal HPV was detected in 69.4% and oral HPV in 8.4% of the sample. However <10% reported getting the HPV vaccine to protect against cancers caused by HPV infection. Second, co-infection of HIV with HPV is also higher in SMM, thereby increasing the likelihood of the development of anal, penile, and oropharyngeal cancers among HIV-positive adults.^{10–13} Compared to penile and oropharyngeal HPV infections, anal HPV infections among SMM are more likely to be attributed to high-risk, oncogenic HPV types.^{10,14,15} Despite the increased health risks, there is also limited research on HPV vaccination uptake in SMM to explain why uptake is relatively low.^{16–19} Studies have reported that SMM, including those who were in their adolescence and vaccine eligible, had very low vaccine uptake, with only 5–13% of SMM initiating the vaccine series compared to 35% of all young men.^{9–14}

The objective of this study was to describe the baseline prevalence of HPV infection among young SMM. The aims of this study were as follows: (1) to document vaccination rates and examine the covariates associated with HPV vaccination in a racially/ethnically and socioeconomically diverse sample of young SMM in the New York metropolitan area, who entered adolescence after the availability of the vaccine; and (2) to describe the prevalence and covariates of anal and oral HPV infection, including both high-risk (oncogenic), low-risk (nononcogenic), and vaccine-preventable types, among young SMM.

Methods

Study sample

The P18 Cohort Study, a prospective study informed by a syndemic conceptual model,²⁰ sought to examine patterns of health-seeking and health risk behaviors in emerging adult SMM. In the Fall of 2015, participants took part in an audio-computer-assisted cross-sectional survey to provide information on sociodemographic characteristics, health-related behaviors, HPV vaccination uptake,²¹ and testing for oral and anal HPV. Detailed recruitment and enrollment procedures have been described elsewhere.^{22–27}

At the time of the addition of HPV testing, study participants were partaking in either the month 12 or 18 assessment of the cohort study. A total of $n = 486$ (73.1%) participated in this subcomponent and gave informed consent. There was no differential attrition detected along key demographic variables between the baseline cohort sample ($n = 665$) and analytic sample ($n = 486$).

HPV vaccination status

The survey asked the following: “Have you ever received an HPV vaccination? (It is a shot)” and respondents were given the options of answering: “Yes,” “No,” or “Don’t know.” Those who indicated receiving the HPV vaccine were then asked the following: “How many times did you receive an HPV vaccination(s)?” with options of one, two, or

three (complete series) vaccinations or doses. At that time, both quadrivalent and nonavalent⁵ vaccine were available, although the quadrivalent vaccine was removed from the market in the United States in 2017.²⁸

Multi-site HPV specimen and HIV antibody collection procedures

HPV was assayed from oral mouthwash samples and anal swabs. Public health research assistants trained in the data collection of human biospecimens provided detailed instructions to study participants on how to self-administer the self-sampling by Dacron anal swabs. Testing of anal specimens was conducted using Gynasure HPV16/18/HR using Roche Cobas[®] 4800 method and additionally genotyping for all risk associations using multiplex polymerase chain reaction (PCR) and automated microcapillary electrophoresis. The oral HPV test was collected and prepared following the HIM Study protocols.^{29,30} The Oral HPV test is a noninvasive, easy-to-use screening tool where participants were asked to wash their oral cavity, including the throat, by swishing a prepared mouthwash solution in their mouth vigorously for 30 sec. Participants were instructed to swish the mouthwash solution such that the mouthwash covered as many surfaces inside the mouth as possible. Mouthwash samples were collected and sent to a contract laboratory for processing and testing by OraRisk HPV test (OralDNA Labs[®]), a PCR test used to detect HPV types. Based on these testing algorithms, we were able to collect data on 52 distinct HPV types. In addition, confirmed HIV status was available for each participant based on testing and reporting as part of the cohort study, through the implementation of the Alere Determine HIV-1/2 Ag/Ab Combo HIV-1/2 ABS with reflex confirmation.

Demographic and structural covariates

Participants reported date of birth, which was used to determine the age at the time of assessment, and identified race/ethnicity by two items, which were categorized as follows: black Non-Hispanic, white Non-Hispanic, Other Non-Hispanic, and Hispanic regardless of race. Self-reported personal annual income was measured on a categorical scale and was transformed into a binary variable for those selecting categories at or below the poverty level and above poverty level, aligning with the poverty statistics of the region. Level of educational attainment was collapsed into three categories: high school or less, some college, and college or graduate degree. Participants also identified as either cisgender male or transgender female; sexual orientation was assessed using the Kinsey scale³¹ and recoded to exclusively homosexual or not exclusively homosexual.

HIV prevalence as per participant zip code/neighborhood was extracted and recorded as the number of persons living with diagnosed HIV per 100,000 people in the residential zip code.³² These numbers were further isolated into categories of heightened prevalence (2% or higher) and lesser prevalence (<2%). As an indicator of neighborhood poverty, data on public assistance were retrieved from New York City’s Department of Youth and Community Development based on 2018 data.³³ The public assistance variable dichotomized high poverty (i.e., 20% or higher of the population received

public assistance) and low poverty (i.e., <20% of the population received public assistance).

Statistical analyses

First, rates of HPV vaccination were computed. Associations of vaccination status with the demographic and structural covariates were assessed through chi-square tests of independence. We then computed rates of HPV: (1) oral infection (high-risk cancer type, low-risk cancer type, unknown risk cancer, and any type), (2) anal infection (high-risk cancer type, low-risk cancer type, unknown-risk cancer type, and any type); (3) vaccine-preventable infection, and (4) any HPV infection. Specifically, we computed the HPV vaccine-preventable exposure based on the nonavalent vaccine for HPV 6, 11, 16, 18, 31, 33, 45, 52, and 58.³⁴ Each variable was examined in reference to the following demographic factors (race/ethnicity, confirmed HIV status, educational attainment, and poverty level) and structural factors (residential neighborhood HIV prevalence and poverty level) using chi-square tests of independence. In addition, associations between the four main exposure variables were computed by tetrachoric correlations. Based on the bivariable analyses, we examined the combined effects of the significant factors on each of the four exposure variables in relation to binary logistic regression models.

TABLE 1. CHARACTERISTICS OF STUDY PARTICIPANTS (N=486)

	% (n)
Race/ethnicity	
White	25.1 (122)
Hispanic/Latino	33.1 (161)
Black	25.5 (124)
Other	16.3 (79)
Educational attainment	
High school/GED or less	29.2 (142)
Some college	31.7 (154)
Bachelor's or graduate degree	38.9 (189)
Missing	0.2 (1)
Personal income	
Near/below poverty level	73.9 (359)
Above poverty level	21.4 (104)
Missing	4.7 (23)
Gender	
Male	93.2 (453)
Transgender	6.8 (33)
Confirmed HIV status	
Negative	93.2 (453)
Positive	6.8 (33)
Neighborhood HIV prevalence	
Low HIV prevalence	16.5 (80)
High HIV prevalence	27.2 (132)
Missing	56.4 (274)
Neighborhood poverty	
Low poverty	48.4 (235)
High poverty	31.5 (153)
Missing	20.2 (98)

Results

Participant characteristics

Age of the 486 cohort participants who took part in the HPV substudy ranged from 22 to 25 years old ($M=23.72$, $SD=0.70$). As shown in Table 1, >70% of participants were young men of color, with 33.1% ($n=161$) identifying as Hispanic (regardless of race) and 25.5% ($n=124$) identifying as non-Hispanic or black. In addition, 7.0% of participants identified as transgender, and 6.8% ($n=33$) of participants were HIV seropositive.

Over half (62.3%, $n=132$) of participants indicated residing in a zip code with an HIV prevalence of greater than 2%. Residing in a high HIV prevalence neighborhood was found to be associated with an HIV-positive status ($p=0.04$), a finding consistent with the literature.³⁵ In addition, 39.4% ($n=153$) resided in the neighborhood of high poverty, as indicated by rates of public assistance. Moreover, higher HIV prevalence was detected in neighborhoods with higher levels of public assistance [$\chi^2(1)=20.79$, $p=0.001$], with 80.5% ($n=70$) of those living in a high poverty neighborhood also living in a neighborhood with HIV prevalence of 2% or higher.

Uptake of HPV vaccination

In terms of HPV vaccination, only 18.1% ($n=88$) of participants indicated being fully vaccinated for HPV (i.e., reported receiving all three doses, the complete series) and 24.3% ($n=118$) reported receiving less than three doses (i.e., an incomplete series). The majority of those who did not receive the full series (64.4%, $n=76$) reported receiving only one dose of the vaccine. Importantly, 45.1% ($n=219$) had received no dose of HPV vaccination, despite coming of age when both the quadrivalent and nonavalent vaccines were available. As shown in Table 2, vaccination uptake is not associated with either race/ethnicity or HIV status. Vaccination is also not associated with age, educational attainment, sexual orientation, gender identity, or poverty level. Table 3 demonstrates that HPV vaccination is not associated with either of the structural covariates, namely residential neighborhood poverty and HIV prevalence.

Prevalence of HPV infection

More than half of the participants tested positive for any HPV infection (58.6%, $n=285$). Anal HPV infection was high, with 56.0% ($n=272$) of the sample testing positive, while 8.8% ($n=43$) tested positive for oral infection. Also, 30.9% ($n=150$) tested positive for a nonavalent vaccine-preventable HPV infection, irrespective of site. Multi-site HPV infections were detected: specifically, co-infection with anal and oral HPV ($p<0.05$); anal vaccine-preventable HPV ($p<0.01$); and oral vaccine-preventable HPV ($p<0.01$).

Table 2 provides a summary of HPV infection overall by race/ethnicity and HIV serostatus. As noted, no differences emerged regarding race/ethnicity. In addition, there were no statistically significant relationships between any of the HPV infection variables with educational status, gender identity, income, sexual orientation, and age. However, HIV infection was significantly associated with HPV infection [OR=5.13, 95% confidence interval (CI)=1.77–14.83]. Specifically,

TABLE 2. HUMAN PAPILLOMAVIRUS VACCINATION AND INFECTION STRATIFIED BY RACE/ETHNICITY AND CONFIRMED HIV STATUS

	<i>Race/ethnicity</i>					<i>Confirmed HIV status</i>	
	<i>Total % (n)</i>	<i>Latino % (n)</i>	<i>White % (n)</i>	<i>Black % (n)</i>	<i>Other % (n)</i>	<i>HIV+ % (n)</i>	<i>HIV- % (n)</i>
Vaccination status							
Fully vaccinated	18.1 (88)	19.2 (30)	20.7 (25)	16.5 (20)	16.9 (13)	27.3 (9)	17.9 (79)
Partially vaccinated	24.3 (118)	30.1 (47)	17.4 (21)	26.4 (32)	23.4 (18)	33.1 (11)	24.2 (107)
Not vaccinated	45.1 (219)	40.4 (63)	53.7 (65)	47.1 (57)	44.2 (34)	33.3 (11)	47.1 (208)
Unsure	10.3 (50)	10.3 (16)	8.3 (10)	9.9 (12)	15.6 (12)	6.1 (2)	10.9 (48)
Anal HPV							
High risk	34.6 (162)	34.2 (54)	33.6 (39)	35.0 (41)	36.4 (28)	43.8 (14)	33.9 (148)
Low risk [†]	30.1 (141)	25.9 (41)	27.6 (32)	34.2 (40)	36.4 (28)	56.3 (18)	28.2 (123)
Unknown risk*	7.3 (34)	5.1 (8)	12.9 (15)	3.4 (4)	9.1 (7)	3.1 (1)	7.6 (33)
Any anal	58.1 (272)	56.3 (89)	58.6 (68)	55.6 (65)	64.9 (50)	78.1 (25)	56.7 (247)
Oral HPV							
High risk* [†]	4.7 (23)	1.9 (3)	5.7 (7)	4.0 (5)	10.1 (8)	12.1 (4)	4.2 (19)
Low risk [†]	3.1 (15)	1.9 (3)	2.5 (3)	4.8 (6)	3.8 (3)	9.1 (3)	2.6 (12)
Unknown risk	1.0 (5)	1.2 (2)	0.0 (0)	2.4 (3)	0.0 (0)	3.0 (1)	0.9 (4)
Any oral [†]	8.8 (43)	5.0 (8)	8.2 (10)	11.3 (14)	13.9 (11)	24.2 (8)	7.7 (35)
Vaccine-preventable HPV[†]	32.1 (150)	31.0 (49)	28.4 (33)	33.3 (39)	37.7 (29)	53.1 (17)	30.5 (133)
Any HPV[†]	60.6 (285)	58.2 (92)	60.3 (70)	58.5 (69)	69.2 (54)	87.9 (29)	58.6 (256)

*Significant differences by race/ethnicity, $p < 0.05$.

[†]Significant differences by HIV status, $p < 0.05$.

HPV, human papillomavirus.

87.9% of HIV-positive men tested positive for an HPV infection, irrespective of site, compared to 58.6% of HIV-negative men.

In addition, we examined anal, oral, vaccine-preventable, and any HPV infection in relation to neighborhood-level HIV prevalence and neighborhood-level poverty as indicated by the proportion of the population receiving public assistance as shown in Table 3. Higher neighborhood-level poverty was associated with any HPV infection (OR = 1.81, 95% CI = 1.16–2.83) as well as anal HPV infection (OR = 1.74, 95% CI = 1.12–2.71).

Multi-variable modeling

Based on the results of the bivariate analyses, we conducted multi-variable models to test the combined effects of neighborhood poverty and HIV status with HPV infection utilizing hierarchical entry. These findings, as shown in Table 4, indicate a statistically significant relationship between HIV seropositivity with oral HPV infection ($p < 0.05$) and vaccine-preventable HPV infection ($p = 0.01$), as well as the combined effects of HIV-positive serostatus and neighborhood poverty for anal HPV infection ($p = 0.01$) and any HPV infection ($p < 0.001$).

TABLE 3. HUMAN PAPILLOMAVIRUS VACCINATION AND EXPOSURE STRATIFIED BY RESIDENTIAL NEIGHBORHOOD-LEVEL POVERTY AND HIV PREVALENCE

	<i>Neighborhood poverty</i>		<i>Neighborhood HIV prevalence</i>	
	<i>Low poverty % (n)</i>	<i>High poverty % (n)</i>	<i>Low prevalence % (n)</i>	<i>High prevalence % (n)</i>
Vaccination status				
Unvaccinated	44.8 (104)	44.9 (66)	50.0 (40)	38.4 (48)
Partially vaccinated	23.3 (54)	26.5 (39)	20.0 (16)	26.4 (33)
Fully vaccinated	20.7 (48)	16.3 (24)	20.0 (16)	20.0 (25)
Unsure	11.2 (26)	12.2 (28)	10.0 (8)	15.2 (19)
Anal HPV infection*	56.0 (126)	68.5 (100)	54.7 (41)	60.3 (76)
Oral HPV infection	8.9 (21)	8.5 (13)	7.5 (6)	6.1 (14)
Vaccine-preventable HPV infection^a	30.7 (69)	39.0 (57)	33.3 (25)	34.9 (44)
Any HPV infection[†]	57.7 (131)	71.2 (104)	54.7 (41)	61.4 (78)

No significant associations between HPV type and HIV prevalence.

* $p < 0.05$.

[†] $p < 0.01$.

^aNonavalent vaccine (HPV 6, 11, 16, 18, 31, 33, 45, 52, and 58).

TABLE 4. MULTI-VARIABLE MODELING OF HUMAN PAPILLOMAVIRUS INFECTION

	OR (95% CI)	AOR (95% CI)
Anal HPV infection		
Poverty	1.71 (1.10–2.65)*	1.68 (1.08–2.62)*
HIV status	—	3.13 (1.04–9.46)*
Oral HPV infection		
Poverty	0.95 (0.46–1.95)	0.91 (0.44–1.90)
HIV status	—	4.03 (1.48–10.98) [†]
Vaccine-preventable HPV infection		
Poverty	1.45 (0.94–2.24)	1.42 (0.91–2.21)
HIV status	—	3.21 (1.34–7.65) [†]
Any HPV infection		
Poverty	1.82 (1.16–2.83) [†]	1.80 (1.15–2.82)*
HIV status	—	6.91 (1.59–29.98) [†]

* $p < 0.05$.[†] $p < 0.01$.

AOR, adjusted odds ratio; CI, confidence interval.

Discussion

The findings add to a growing literature that documents the burden of HPV infection in SMM.^{36,37} Our analyses expand our understanding regarding high cancer risk and low cancer risk HPV types and barriers to HPV prevention and treatment, through documenting the rates of both oncogenic and nononcogenic HPV infections affecting young SMM who entered adolescence after HPV vaccination was approved and available. The rates presented in this study are predicated on the fact that routine vaccination has been implemented in the last 5 years.

Even though HPV vaccination is highly effective in the prevention of HPV infections,^{38–40} vaccination rates in young SMM remain low.^{39,41} Less than a quarter of participants were partially vaccinated, and less than a fifth completed their vaccination series. While receiving only one or two of the three recommended doses may provide some protection, the efficacy and implications of incomplete vaccination are not well studied, and have not been researched among SMM.^{42,43} Our findings indicate a missed opportunity for HPV prevention in this sample, given the low rates of HPV vaccination uptake coupled with a high prevalence of HPV infection, particularly infection with vaccine-preventable HPV types at oral and anal sites.

The findings further indicate no significant difference in HPV vaccination and infection by race/ethnicity or income. These results run counter to the health disparities research in the United States that demonstrate differences across these strata, whereby black and Hispanic populations and lower levels of income are more likely to be burdened.^{44,45} Instead, the findings present a distribution of HPV infection along the continuum of race and income that closely resembles the infection distribution at the onset of the AIDS epidemic, in which there was little differentiation by socioeconomic status and race/ethnicity to HIV-related morbidity and mortality,⁴⁶ although these disparities have emerged over the last four decades. Moreover, we might expect such differences to emerge as noted in more widespread vaccinations such as influenza.⁴⁷ These results suggest that lack of HPV vaccination in young SMM is not presently directed by access.

Instead, a lack of education regarding HPV vaccination and infection may be the driver of this health challenge. For example, in Australian men who have sex with men, only 30% were aware that an HPV vaccine existed and 93% of those surveyed indicated that they would be open about their sexual orientation with health care providers to obtain the HPV vaccine for free.⁴⁸

With most of the sample testing positive for HPV, it seems that current HPV prevention measures and HPV education efforts have failed not only SMM but also men in general. A systematic review by Holman et al.⁴⁹ of HPV vaccination also suggests that male-specific HPV information and education are lacking, prompting many adolescent males and parents of young men to ignore the vaccine due to conflicting or no male-specific HPV information.¹ In addition, vaccination rates and HPV exposure remained consistent across all sociodemographic variables for SMM in our study, further indicating the systemic failings of male-specific HPV education and prevention that cut across races and income levels. These findings call for novel health promotion and education efforts that focus on increasing HPV information and vaccination in males, specifically in SMM.

These findings indicate high rates of co-infection of HIV with both oncogenic and nononcogenic types of HPV infection, a finding consistent with the literature.^{36,50} For example, Combes et al.⁵¹ found that HIV-positive men older than 35 years reported high rates of HPV 16. This cohort of older men also came of age at the time when HPV vaccination was not available and during a period when HIV transmission heavily burdened the population before the implementation of antiretroviral therapy (ART). This association is not surprising; these findings raise concern because this HPV and HIV co-infection are detected in a younger cohort of men, all of whom came of age after ART and in the era of HPV vaccination.

The high prevalence of HPV infection in young SMM, particularly those who are co-infected with HIV, has been well documented worldwide.^{52–54} In this study, 87.9% of HIV-positive young SMM were also infected by at least one HPV type, either of high or low oncogenic risk, a proportion comparable to the 92.6% reported in a systematic review and meta-analysis of HPV/HIV co-infected men who have sex with men.⁵⁵ Current evidence suggests that HPV and HIV infections may interact in multiple negative biological and immunological ways.^{56,57} For instance, the clearance of HPV infection is compromised by the presence of HIV infection,^{58–60} leading to the persistence of HPV and an increased risk of intraepithelial cervical and anal neoplasia. These findings suggest a further need to increase efforts to screen for and treat cases of anal dysplasia and carcinoma, particularly among SMM with HPV and HIV co-infection.⁶¹

HPV infection also negatively affects an individual's susceptibility to contract and clear HIV. HPV has been found to increase HIV susceptibility by (1) affecting the epithelial barrier; (2) recruiting HIV target cells; and/or (3) generating pro-inflammatory conditions.⁵⁷ The presence of unusually high levels of HPV/HIV co-infection among men who have sex with men and their harmful symbioses, signals the need for concerted actions by researchers, health service providers, and policymakers to learn more about this ongoing public health issue to clarify the specific dynamics shaping the field.

The Federal Drug Administration (FDA) strongly recommends that all boys and girls should be offered the HPV

vaccine between 9 and 26 years of age, ideally before starting their sexual lives. The recommended vaccination age range was expanded in 2018 to include those 27–45 years of age.⁶ The value of administering the HPV vaccine to adult SMM, either HIV positive or not, who may have already been exposed to high-risk strains of HPV, is debatable. As suggested by Patel et al.,⁶² the use of the HPV vaccine in older HIV-positive adults warrants further evaluation. That being said, HPV vaccine use in such cases is currently considered an “off-label” usage,⁶³ despite evidence suggesting decreases in recurrence of high-grade anal intraepithelial neoplasia in individuals receiving vaccination.⁶⁴

Strengths and limitations

There are several limitations that we acknowledge in this study. With regard to the detection of HPV infection, self-sampling may have limited detection of HPV infection if not conducted properly.⁶⁵ Attempts were made to mitigate sampling errors by providing the participants with clear written instructions for oral and anal sampling procedures, including pictographic instructions for the anal swabs. The DNA PCR was used for rectal detection of HPV types. Another limitation is that nonavalent vaccines became the standard of care in the United States in 2017.^{5,34} It is possible that some of the participants may have been vaccinated before that time; we also did not distinguish between whether they received the quadrivalent versus the nonavalent vaccine. We determined HPV vaccine-preventable infection based on the nonavalent vaccine, but note that a positive exposure for a small subset of the participants may be due to lack of information on whether they were vaccinated when the quadrivalent vaccine was standard of care. Another limitation is that HPV vaccination data are based on self-report and are subject to recall bias and social desirability. However, given the low levels of vaccination uptake and completion, our estimates are not likely to overestimate vaccine uptake or completion in this sample. Further, as with all cross-sectional data, we cannot infer causality with these data.

Our results are also limited regarding how we assessed socioeconomic/neighborhood-level variables, HIV prevalence, and poverty estimates. Poverty estimates were assessed by (proxy through the proportion of the zip code population) the percent of individuals receiving public assistance in New York City, a calculation that could have been refined by using zip code tabulation area data from the 2000 US Census to quantify the neighborhood socioeconomic status.^{66,67} Moreover, these data were not available for those residing outside New York City. Finally, zip code level data are crude in their assessment of neighborhood characteristics.

Despite this limitation, our study design is robust in many ways; findings highlight the disparities in HPV vaccine uptake in a highly diverse sample of SMM, who, for all intents and purposes, should have been vaccinated for HPV. The findings generate knowledge about HPV detection and associated drivers of HPV infection in a new generation of SMM coming of age both after the implementation of ART to treat HIV in 1996 as well as after the implementation of the HPV vaccination, a decade later.

HPV infection is a significant health burden experienced by sexual minority boys and young men.^{68,69} A call to action for vaccinating all boys and men before sexual debut⁶⁹ has

increased salience for SMM who continue to be at heightened risk for HIV.^{70–72} The low vaccination of HPV uptake among boys and young men in the United States combined with nonroutinized testing for anal and oral cancers in the population of SMM, particularly those living with HIV, create an undue, yet preventable personal and community health burden.

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Ethical Statement

The study protocol was approved by the Institutional Review Board of both Rutgers University and New York University. Informed consent was obtained from all participants.

Author Disclosure Statement

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