# Prevalence of Genital Human Papillomavirus Among Sexually Experienced Males and Females Aged 14-59 Years, United States, 2013-2014 

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#### Abstract

Background-Differences in human papillomavirus (HPV) prevalence among males and females have been reported. Using the 2013-2014 National Health and Nutrition Examination Survey, we evaluated sex differences in prevalence overall and by demographic and sexual behavior characteristics.

Methods-Self-collected penile and cervicovaginal swabs from participants aged 14-59 were tested for HPV DNA. Prevalences of any HPV and high-risk HPV (HR-HPV) were estimated for sexually experienced males and females. Overall and in models stratified by demographic characteristics and behaviors, prevalence was compared in males and females using prevalence ratios (PR).

Results-Overall, males had higher prevalence than females of any HPV (PR, 1.14; 95\% confidence interval [CI], 1.03-1.27) and HR-HPV (PR, 1.24; 95\% CI, 1.07-1.43). Prevalences were lower among males than females at ages 14-19 and higher at ages 40-49 and 50-59. Sex differences in models stratified by race/ethnicity, poverty, sexual behaviors, and smoking were observed. After adjusting for lifetime sex partners, most sex differences were attenuated, but males had lower prevalences at ages 14-19 and 20-24 and higher HR-HPV prevalence among nonHispanic blacks.


[^0]Conclusions—Any HPV and HR-HPV prevalences were significantly higher in males; sex differences varied by age group and race/ethnicity. Lifetime partners explained many of the differences by sex.

## Keywords

human papillomavirus; HPV prevalence; NHANES; sex differences


#### Abstract

Human papillomavirus (HPV) is the most common sexually transmitted infection [1]. In the United States, it has been estimated that at least $80 \%$ of people will acquire an HPV infection in their lifetime [2]. The majority of HPV infections are asymptomatic and become undetectable within 1 year [3]. However, persistent infection can lead to HPV-associated disease [4]. Among the $>40$ HPV types that infect the genital tract, 13-14 types are considered high risk due to their association with several types of cancer including cervical, vaginal, vulvar, penile, anal, and oropharyngeal cancers [4, 5].


In an effort to define HPV epidemiology and monitor early vaccine impact in the United States, cervicovaginal HPV DNA testing among females aged 14-59 years was incorporated into the National Health and Nutrition Examination Survey (NHANES) starting in 2003. Previous assessments of HPV prevalence among females in NHANES showed that prevalence of HPV varied significantly by age and race/ethnicity, with 20- to 24-year-olds and non-Hispanic black adults having the highest prevalences [6]. Assessments of NHANES have also shown that prevalence of the quadrivalent HPV vaccine types (ie, 6/11/16/18) declined after introduction of the vaccination program in 2006 [7, 8]. In 2013-2014, genital HPV DNA testing among males aged 14-59 years was incorporated into NHANES for the first time $[9,10]$.

The majority of research on the epidemiology of genital HPV in males and females has included studies limited to 1 sex; reviews have presented results from multiple studies and described differences between males and females [11, 12]. There have been few studies of prevalence in both males and females in the same population [13-15]. A previous report from NHANES showed that prevalence of genital HPV was higher in males than females overall and among non-Hispanic white adults, and prevalence of high-risk HPV was higher in males than females overall and among both non-Hispanic white and non-Hispanic black adults [15]. The influence of covariates on these associations is unclear. In this analysis, we explore these differences further by comparing genital HPV prevalence among males and females aged 14-59 years in the United States, overall and by race/ethnicity, and assess the influence of other demographic and sexual behavior characteristics on HPV prevalence differences between males and females.

## METHODS

## Data Source and Analytic Sample

NHANES is an ongoing cross-sectional survey administered by the National Center for Health Statistics (NCHS) at the Centers for Disease Control and Prevention (CDC) and is designed to be nationally representative of the noninstitutionalized, civilian population living
in the United States. Detailed survey methods have been described previously [16, 17]. Participants completed interviews both in the home and as part of a subsequent examination in the Mobile Examination Center (MEC). A physical examination, including biological specimen collection, was also completed in the MEC. The in-home interview included the collection of demographic information. More sensitive information (eg, sexual behavior information) was collected using audio-computer assisted self-interview in the MEC to ensure participants' privacy. Informed consent was obtained from all participants or their guardians. Data collection was approved by the NCHS Research Ethics Review Board.

Average response rates to the 2013-2014 NHANES cycle among males and females were $70.6 \%$ and $71.4 \%$ for the interview portion and $68.1 \%$ and $68.8 \%$ for the examination portion, respectively. A total of 2401 males and 2601 females were interviewed. This analysis was restricted to participants aged 14-59 years who were sexually experienced (defined as self-report of ever having had any vaginal, anal, or oral sex or at least 1 lifetime sex partner) and had adequate HPV DNA typing results (Figure 1).

Self-reported race/ethnicity was categorized as non-Hispanic white, non-Hispanic black, Hispanic, and non-Hispanic Asian. Due to small sample sizes, males $(\mathrm{n}=65)$ and females ( n = 66) reporting "Other" race/ethnicity were excluded from the analysis, resulting in a final sample size of 1556 males and 1695 females. Poverty income ratio (PIR) was based on selfreported income, household size, and the state poverty level for the year of data collection; we classified PIR <1 as below poverty [18]. Number of lifetime sex partners included both same-sex and opposite-sex partners and was categorized as $1,2-5,6-9,10-15$, and $\geq 16$ lifetime sex partners. Number of partners in the past year was categorized as 0,1 , and $\geq 2$ partners. Age at sexual initiation was based on self-reported age at first sex and categorized as $<16$ years old and $\geq 16$ years old. Self-reported smoking status was categorized as ever smoker ( $\geq 100$ cigarettes in lifetime) and never smoker ( $<100$ cigarettes in lifetime).

## Specimen Collection and Laboratory Methods

Penile and cervicovaginal self-collected swabs were obtained from male and female participants aged 14-59 years as previously described [6, 19]. Specimens were shipped to and analyzed at CDC using Research Use Only Roche Linear Array Assay (Roche Diagnostics, Indianapolis, Indiana). This assay uses L1 consensus polymerase chain reaction (PCR) followed by type-specific hybridization for qualitative detection of 37 HPV types (6/11/16/18/26/31/33/35/39/40/42/45/51/XR(52)/5 3/54/55/56/58/59/61/62/64/66/67/68/69/70/71/72/73/81/82/83/84/IS39/89) and $\beta$-globin. A type-specific HPV-52 quantitative PCR was used to determine HPV-52 status of samples positive for XR and 33, 35, or 58 [19]. Samples negative for both HPV and $\beta$-globin were considered inadequate and excluded from analysis.

## Statistical Analysis

We estimated positivity to any of the 37 HPV types (any HPV) and any of 14 high-risk types (HR-HPV; HPV 16/18/31/33/35/39/45/51/XR(52)/56/58/59/66/68) by demographic characteristics, sexual behaviors, and smoking status for both males and females. All estimates were weighted using the examination sample weights to account for unequal
probability of selection and nonresponse, and variances were estimated using analyses for complex surveys [20]. Additionally, the number of HPV types for which each participant was positive was calculated; the mean and standard deviation of number of positive types were estimated. We estimated prevalences of any HPV and HR-HPV by age group and sex, overall and separately for non-Hispanic whites, non-Hispanic blacks, and Hispanics. Agespecific estimates for non-Hispanic Asians were not presented due to small sample sizes and unstable estimates; estimates with a relative standard error of $>30 \%$ were noted as statistically unstable.

Logistic regression was used to estimate unadjusted prevalence ratios (PR) using predicted marginal prevalence estimates comparing males to females overall and in models stratified by age group, race/ethnicity, PIR, number of lifetime sex partners, number of sex partners in the past year, age at sexual initiation, and smoking status. This was performed using the PREDMARG statement of the logistic regression procedure in SUDAAN. To determine the effects of covariates on sex differences in prevalence for each model, adjusted prevalence ratios (aPRs) were estimated using multivariable logistic regression. Covariates were included in a model if they significantly changed the PR by $\geq 10 \%$ for at least 1 level of the model-specific characteristic or behavior. Covariates were tested individually. If $>1$ covariate significantly changed at least 1 PR , the covariate that made the largest change was included. Once a covariate entered the model, each remaining variable was retested to assess whether its inclusion further changed the aPR estimates. This process was repeated until no further changes in aPRs were observed.

For all comparisons, a 2-tailed $P$ value was considered statistically significant if <.05. Data management and analysis were performed in SAS software version 9.3 (SAS Institute, Cary, North Carolina) and SAS-callable SUDAAN 11.0 (RTI International, Research Triangle Park, North Carolina).

## RESULTS

In 2013-2014 among sexually experienced 14- to 59-year-olds, prevalence of any HPV was $45.8 \%(95 \%$ confidence interval [CI], $41.9 \%-49.7 \%$ ) in males and $40.1 \% ~(95 \%$ CI, $36.1 \%-$ $44.2 \%$ ) in females. Prevalence of HR-HPV was $25.7 \%$ ( $95 \%$ CI, $23.4 \%-28.1 \%$ ) in males and $20.7 \%$ ( $95 \%$ CI, $17.8 \%-23.9 \%$ ) in females. Among those positive for any HPV, the mean number of types among both males and females was 2.1 , and $50.9 \%$ of males and $47.8 \%$ of females tested positive for exactly 1 HPV type. Additionally, among those positive for HPV, percentages of males and females testing positive for 2-3 types (males: $34.7 \%$; females: $38.2 \%$ ) and $\geq 4$ types (males: $14.5 \%$; females: $14.0 \%$ ) were also similar by sex.

The pattern of age-specific prevalence differed by sex (Figure 2A). Among females, the prevalences of both any HPV and HR-HPV were highest in 20- to 24-year-olds ( $62.8 \%$ and $39.2 \%$ ), and prevalences were significantly lower in all older age groups. Among males, the prevalences of any HPV and HR-HPV were highest among 25- to 29-year-olds ( $51.9 \%$ and $33.0 \%$ ). Compared to 25 - to 29 -year-old males, prevalence of any HPV was not significantly different in older male age groups, but prevalence of HR-HPV was significantly lower in 40-
to 49-year-olds. Age-specific patterns in prevalence varied by race/ethnicity (Figure 2B-D and Supplementary Table).

Sex-specific prevalences, PRs, and aPRs are presented in Table 1 for each level of each demographic and behavioral characteristic. Overall, males had significantly higher prevalence of both any HPV (PR, 1.14; 95\% CI, 1.03-1.27) and HR-HPV (PR, 1.24; 95\% CI, 1.07-1.43) compared to females (Table 1). Among 14- to 19-year-olds, males had significantly lower prevalences of both any HPV and HR-HPV than females. In 20- to 24-year-olds, the difference by sex remained significant for any HPV but not for HR-HPV prevalence. HR-HPV prevalence among males was higher than among females in the age groups 30-39, 40-49, and 50-59 years. Additionally, in the oldest 2 age groups, males also had a significantly higher any HPV prevalence compared to females.

Sex differences in prevalence varied by race/ethnicity (Table 1). Among non-Hispanic whites, males had significantly higher prevalences of any HPV and HR-HPV compared to females. Among non-Hispanic blacks, males had a higher prevalence of HR-HPV than females (PR, $1.37 ; 95 \%$ CI, 1.14-1.65) but not of any HPV (PR, $0.95 ; 95 \%$ CI, $.86-1.05$ ). No significant prevalence differences by sex were observed among Hispanics or nonHispanic Asians.

There were also differences in HPV prevalence between males and females at levels of PIR, some sexual behaviors, and smoking status. In individuals at or above the poverty level (PIR $\geq 1$ ), prevalences of both any HPV and HR-HPV were significantly higher among males than females. For those below the poverty level (PIR < 1), prevalence of HR-HPV, but not any HPV, was significantly higher among males than females. While no differences were observed between males and females in lifetime sex partner categories of $<16$ partners, in those reporting $\geq 16$ lifetime sex partners, males had a higher prevalence of HR-HPV compared with females ( $\mathrm{PR}, 1.55 ; 95 \% \mathrm{CI}, 1.18-2.04$ ). In those reporting 1 partner in the past year and ever smokers, males had higher prevalences than females of both any HPV and HR-HPV. Among persons who reported sexual initiation at $\geq 16$ years of age, males had a significantly higher HR-HPV prevalence than females.

For all models, lifetime sex partners changed the PR for at least 1 level of the model-specific variable by $\geq 10 \%$; therefore, it was included in all model adjustments. For most models, lifetime sex partners was the only covariate that was included in the model (Table 1). After adjusting for number of lifetime sex partners and any other covariates in each model, most sex differences were attenuated. In the unstratified adjusted model, sex was not associated with overall prevalence of any HPV (aPR, $0.99 ; 95 \% \mathrm{CI}, .89-1.10$ ) or HR-HPV (aPR, 1.03; $95 \% \mathrm{CI}, .88-1.21$ ). In the youngest 2 age groups, adjusted prevalences of any HPV and HRHPV were significantly lower among males than females. Among non-Hispanic blacks, males had a lower adjusted any HPV prevalence and higher adjusted HR-HPV prevalence compared to females. Among persons reporting $\geq 16$ lifetime sex partners, adjusted HR-HPV prevalence among males was higher than females; this finding was likely because, within this category, males had more lifetime sex partners than females (median, 29.1 vs 24.7; mean, 61.1 vs 32.3 ). Among persons reporting $\geq 2$ sex partners in the past year, males had significantly lower adjusted prevalence of any HPV than females.

## DISCUSSION

In this comparison of genital HPV prevalence in sexually experienced males and females aged 14-59 years in a nationally representative United States sample, we observed differences in HPV prevalence by sex. Overall, any HPV and HR-HPV prevalences were significantly higher in males, but sex differences varied by age group with any HPV and HR-HPV prevalences being lower in males than females in 14- to 19-year-olds. Many associations seen across demographic groups were attenuated after adjusting for number of lifetime sex partners, suggesting that number of lifetime sex partners explained many of the prevalence differences observed between males and females. However, differences by sex remained significant in younger age groups, non-Hispanic blacks, those reporting $\geq 16$ lifetime sex partners, and those reporting $\geq 2$ sex partners in the past year. Interestingly, while prevalence of HR-HPV remained higher in males than females among non-Hispanic blacks, prevalence of any HPV was lower.

In our study, males and females showed different patterns of prevalence across age groups, consistent with sex-specific patterns found in other studies [21-24]. In males, any HPV prevalence has been found to be highest at later ages compared to females, with little to no decline in older age groups [21, 22]. In North American females, HPV prevalence has been reported to be highest between ages 20 and 25 years and lower at older ages [23, 24]. Some variation in prevalence patterns by geographical region has been noted. In South and Central America, regions of Europe, and regions of Africa, a U-shaped prevalence pattern has been seen, with the highest prevalence in females aged $\Sigma 25$ years and a later peak in women aged $>55$ years. This pattern is similar to the pattern seen in our study among non-Hispanic black and Hispanic females. Non-Hispanic white females, however, exhibit a pattern more similar to that of females from North America and Northern and Eastern Europe, where no increase in older age groups has been observed [24].

Other data comparing prevalence in males and females from the same population are available from an evaluation using urine sampling in a nationally representative survey in Britain [14] and for oral HPV from NHANES [13]. In both studies, overall HPV prevalence and age trends also varied by sex; however, there were differences compared to our study. In Britain, HPV prevalence among 16- to 44-year-olds was evaluated using urine, which is less sensitive for detection of genital HPV in males compared with females [14]. HR-HPV prevalence in females ranged from 1.7 to 7.4 times that of males in age groups through 34 years, but in 35- to 44-year-olds, HR-HPV prevalence was similar in males and females [14]. In contrast, our study found HR-HPV prevalence in females was 1.9 and 1.4 times that in males among those aged 14-19 and 20-24 years but similar or lower in older age groups. The sample used for genital HPV detection likely contributed to the difference in findings compared with our study. In the report of oral HPV prevalence among 14- to 69-year-olds, males had higher oral any HPV prevalence than females; even after accounting for risk behaviors, the overall oral HPV prevalence among males remained $>2$ times the prevalence among females [13]. Differences in risk factors and the natural history of oral and genital HPV have been reported [12].

We found that detection of multiple genital HPV types, among those with any HPV detected, was similar for males and females. Few estimates of prevalence of multiple-type HPV in males and females have been published, and studies often are not comparable due to differences in collection and genotyping methods. Estimates from our study are similar to previous research reporting that approximately $20 \%$ of both females and males have >1 HPV type detected [25, 26]. Concurrent infection with multiple HPV types has been associated with several negative health outcomes in females, including abnormal cytology [27], treatment failure for invasive cervical cancer [28], cervical intraepithelial neoplasia (CIN) severity [29], and cytological diagnoses of CIN grade 2 or worse and high-grade squamous intraepithelial lesions or worse [30]. Less is known about the importance of multiple-type infections in males.

The differences in patterns of genital HPV prevalence by sex we and others observed could be attributable to a combination of biological and behavioral differences. Knowledge about the differences in natural history of HPV between males and females has increased substantially in recent years [12,22]. Due to differing cell characteristics at the anatomic site of infection (keratinized vs mucosal epithelium), it is hypothesized that a weaker immune response is elicited after genital HPV infection in males compared with females. This biological difference may explain the lower rate of seroconversion in males, despite males having a higher HPV prevalence, and could result in less acquired immunity after infection and continued risk of acquiring new infections [31]. Sex differences in behaviors, such as differences in the number of sex partners throughout the life course [32], likely also contribute to sex differences in patterns of HPV prevalence.

When we controlled for number of lifetime sex partners and other covariates, male sex was no longer associated with higher prevalence overall or in most subgroups. Of note, most longitudinal heterosexual transmission studies among initially HPV-discordant couples have found that male-to-female transmission of HPV was lower than female-to-male transmission [12]. This suggests that prevalence in males would remain higher than females even after controlling for number of partners. In our study, this was observed for HR-HPV among nonHispanic blacks but not for other racial/ethnic groups or for any HPV. The higher incidence in males could be counterbalanced by shorter duration of infection [11]. Furthermore, differences in both duration and patterns of prevalence across age in males by country have been reported and suggest variation across population groups [22]. The higher probabilities for female-to-male transmission, the difference in naturally acquired immunity between males and females, and sexual behavior probably all contribute to the different patterns in prevalence across the life span by sex.

These data were collected 7-8 years after HPV vaccination was incorporated into the routine immunization schedule for females in the United States in 2006 and 2-3 years after the routine recommendation for males in 2011. Although coverage of at least 1 dose in 13- to 17 -year-olds has only reached $65 \%$ in females and $56 \%$ in males as of 2016 [33], vaccination has led to decreases in vaccine-type HPV prevalence in females aged 14-19 and 20-24 years in the United States [7, 8]. However, non-vaccine-type HPV prevalence has not declined significantly in these age groups [7, 8]. We found that prevalences of any HPV and HR-HPV among females were higher than among males for these age groups, despite higher
vaccination coverage and decreases in vaccine type prevalence in females. Therefore, we assume that in the absence of vaccination, sex differences in the youngest age groups would be similar, and potentially even more pronounced, than found in our study.

This analysis has several limitations. First, some prevalence estimates were statistically unstable. We presented prevalence estimates stratified by race, sex, and age that suggested the possibility of a 3-way interaction, but we were unable to formally test this due to the limited number of degrees of freedom $(\sim 15)$ permitted with data from 1 cycle of NHANES. The NHANES analytic guidelines recommend combining adjacent cycles of data to improve precision and reduce sampling error, especially for subdomain analyses (eg, race/ethnicity). To date, only 1 cycle of NHANES data has been released that includes both males and females; future analyses should combine cycles when available to achieve more stable estimates and perform refined analyses. Second, HPV detection is dependent on anatomic site sampled. We compared genital HPV detection using penile swabs from males and cervicovaginal swabs from females. Differences in the anatomic sites sampled, collection methods, and laboratory processing could affect prevalence estimates. This limitation applies equally to all age groups and levels of other characteristics, and would be unlikely to affect the internal validity of this analysis. Additionally, the DNA extraction method was optimized for each sample type, and genotyping was performed in the same laboratory using the same method to mitigate any differences due to sampling. Third, NHANES is a cross-sectional survey, so it is possible that observed sex differences by age group may be due to cohort effects. Fourth, time since last sexual activity was not assessed using NHANES. It is possible that recent sexual activity might impact detection of HPV DNA that does not represent infection [34].

In conclusion, we report differences in any HPV and HR-HPV prevalences by sex in the United States and explore associations with demographic characteristics and sexual behaviors. In females, the highest prevalence of HPV was observed in women in their early 20s, while in males, the highest prevalence was observed in men in their late 20s. Overall, any HPV and HR-HPV prevalences were significantly higher in males than females, but sex differences varied by age group. Number of lifetime sex partners appeared to be the most important contributing factor to the observed sex differences in prevalence. Overall, there were no significant sex differences in HPV prevalence following adjustment for the number of lifetime sex partners. Differences were observed by race/ethnicity, with males having a higher HR-HPV prevalence than females only among non-Hispanic blacks. Our data contribute to the understanding of HPV epidemiology among both males and females. Future studies may be needed to further explore the sex differences observed in HPV prevalence by age and race/ethnicity in the United States.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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## References

1. Satterwhite CL, Torrone E, Meites E, et al. Sexually transmitted infections among US women and men: prevalence and incidence estimates, 2008. Sex Transm Dis. 2013; 40:187-93. [PubMed: 23403598]
2. Chesson HW, Dunne EF, Hariri S, Markowitz LE. The estimated lifetime probability of acquiring human papilloma-virus in the United States. Sex Transm Dis. 2014; 41:660-4. [PubMed: 25299412]
3. Ho GY, Bierman R, Beardsley L, Chang CJ, Burk RD. Natural history of cervicovaginal papillomavirus infection in young women. N Engl J Med. 1998; 338:423-8. [PubMed: 9459645]
4. Doorbar J, Quint W, Banks L, et al. The biology and life-cycle of human papillomaviruses. Vaccine. 2012; 30(Suppl 5):F55-70. [PubMed: 23199966]
5. IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. Biological agents. Vol 100 B. A review of human carcinogens. IARC Monogr Eval Carcinog Risks Hum. 2012; 100:1441.
6. Hariri S, Unger ER, Sternberg M, et al. Prevalence of genital human papillomavirus among females in the United States, the National Health And Nutrition Examination Survey, 2003-2006. J Infect Dis. 2011; 204:566-73. [PubMed: 21791659]
7. Markowitz LE, Liu G, Hariri S, Steinau M, Dunne EF, Unger ER. Prevalence of HPV after introduction of the vaccination program in the United States. Pediatrics. 2016; 137:e20151968. [PubMed: 26908697]
8. Oliver SE, Unger ER, Lewis R, et al. Prevalence of human papillomavirus among females after vaccine introduction - National Health and Nutrition Examination Survey, United States, 20032014. J Infect Dis. 2017; 216:594-603. [PubMed: 28931217]
9. Gargano JW, Unger ER, Liu G, et al. Prevalence of genital human papillomavirus in males, United States, 2013-2014. J Infect Dis. 2017; 215:1070-9. [PubMed: 28170037]
10. Centers for Disease Control and Prevention. National Health and Nutrition Examination Survey 1999-2016: content brochure. Hyattsville, MD: National Center for Health Statistics; 2015.
11. Taylor S, Bunge E, Bakker M, Castellsagué X. The incidence, clearance and persistence of noncervical human papillomavirus infections: a systematic review of the literature. BMC Infect Dis. 2016; 16:293. [PubMed: 27301867]
12. Giuliano AR, Nyitray AG, Kreimer AR, et al. EUROGIN 2014 roadmap: differences in human papillomavirus infection natural history, transmission and human papillomavirus-related cancer incidence by gender and anatomic site of infection. Int J Cancer. 2015; 136:2752-60. [PubMed: 25043222]
13. Gillison ML, Broutian T, Pickard RK, et al. Prevalence of oral HPV infection in the United States, 2009-2010. JAMA. 2012; 307:693-703. [PubMed: 22282321]
14. Sonnenberg P, Clifton S, Beddows S, et al. Prevalence, risk factors, and uptake of interventions for sexually transmitted infections in Britain: findings from the National Surveys of Sexual Attitudes and Lifestyles (Natsal). Lancet. 2013; 382:1795-806. [PubMed: 24286785]
15. McQuillan G, Kruszon-Moran D, Markowitz LE, Unger ER, Paulose-Ram R. Prevalence of HPV in adults aged 18-69: United States, 2011-2014. NCHS Data Brief. 2017:1-8.
16. Centers for Disease Control and Prevention. National Health and Nutrition Examination Survey: interviewer procedures manual. Hyattsville, MD: National Center for Health Statistics; 2013.
17. Centers for Disease Control and Prevention. National Health and Nutrition Examination Survey (NHANES): MEC interviewers procedures manual. Hyattsville, MD: National Center for Health Statistics; 2013.
18. National Center for Health Statistics. National Health and Nutrition Examination Survey: 2013-2014 data documentation, codebook, and frequencies, demographic variables and sample weights. Hyattsville, MD: National Center for Health Statistics; 2015.
19. Onyekwuluje JM, Steinau M, Swan DC, Unger ER. A real-time PCR assay for HPV52 detection and viral load quantification. Clin Lab. 2012; 58:61-6. [PubMed: 22372346]
20. Johnson, CL., Paulose-Ram, R., Ogden, CL., et al. Vital and Health Statistics. Vol. 2. Hyattsville, MD: National Center for Health Statistics; 2013. National Health and Nutrition Examination Survey: analytic guidelines, 1999-2010.
21. Smith JS, Gilbert PA, Melendy A, Rana RK, Pimenta JM. Age-specific prevalence of human papillomavirus infection in males: a global review. J Adolesc Health. 2011; 48:540-52. [PubMed: 21575812]
22. Sudenga SL, Torres BN, Silva R, et al. Comparison of the natural history of genital HPV infection among men by country: Brazil, Mexico, and the United States. Cancer Epidemiol Biomarkers Prev. 2017; 26:1043-52. [PubMed: 28446543]
23. Smith JS, Melendy A, Rana RK, Pimenta JM. Age-specific prevalence of infection with human papillomavirus in females: a global review. J Adolesc Health. 2008; 43:S5-25. S.e1-41. [PubMed: 18809145]
24. Bruni L, Diaz M, Castellsagué X, Ferrer E, Bosch FX, de Sanjosé S. Cervical human papillomavirus prevalence in 5 continents: meta-analysis of 1 million women with normal cytological findings. J Infect Dis. 2010; 202:1789-99. [PubMed: 21067372]
25. Nielson CM, Harris RB, Flores R, et al. Multiple-type human papillomavirus infection in male anogenital sites: prevalence and associated factors. Cancer Epidemiol Biomarkers Prev. 2009; 18:1077-83. [PubMed: 19318438]
26. Mollers M, Vriend HJ, van der Sande MA, et al. Population- and type-specific clustering of multiple HPV types across diverse risk populations in the Netherlands. Am J Epidemiol. 2014; 179:1236-46. [PubMed: 24714726]
27. Dickson EL, Vogel RI, Geller MA, Downs LS Jr. Cervical cytology and multiple type HPV infection: a study of 8182 women ages 31-65. Gynecol Oncol. 2014; 133:405-8. [PubMed: 24657488]
28. Munagala R, Donà MG, Rai SN, et al. Significance of multiple HPV infection in cervical cancer patients and its impact on treatment response. Int J Oncol. 2009; 34:263-71. [PubMed: 19082497]
29. Bello BD, Spinillo A, Alberizzi P, et al. Cervical infections by multiple human papillomavirus (HPV) genotypes: prevalence and impact on the risk of precancerous epithelial lesions. J Med Virol. 2009; 81:703-12. [PubMed: 19235847]
30. Chaturvedi AK, Katki HA, Hildesheim A, et al. CVT Group. Human papillomavirus infection with multiple types: pattern of coinfection and risk of cervical disease. J Infect Dis. 2011; 203:910-20. [PubMed: 21402543]
31. Giuliano AR, Lee JH, Fulp W, et al. Incidence and clearance of genital human papillomavirus infection in men (HIM): a cohort study. Lancet. 2011; 377:932-40. [PubMed: 21367446]
32. Liu G, Hariri S, Bradley H, Gottlieb SL, Leichliter JS, Markowitz LE. Trends and patterns of sexual behaviors among adolescents and adults aged 14 to 59 years, United States. Sex Transm Dis. 2015; 42:20-6. [PubMed: 25504296]
33. Walker TY, Elam-Evans LD, Singleton JA, et al. National, regional, state, and selected local area vaccination coverage among adolescents aged 13-17 years-United States, 2016. MMWR Morb Mortal Wkly Rep. 2017; 66:874-82. [PubMed: 28837546]
34. Burchell AN, Coutlée F, Tellier PP, Hanley J, Franco EL. Genital transmission of human papillomavirus in recently formed heterosexual couples. J Infect Dis. 2011; 204:1723-9. [PubMed: 21984739]


Figure 1.
Participation and sample collection among participants in the National Health and Nutrition Examination Survey, 2013-2014. ${ }^{\text {a Unweighted percentage sexually experienced among }}$ male participants by age group: $44.1 \%, 90.0 \%, 94.0 \%, 98.0 \%, 98.6 \%$, and $97.2 \%$ among ages $14-19,20-24,25-29,30-39,40-49$, and 50-59 years, respectively. ${ }^{\text {b }}$ Unweighted percentage sexually experienced among female participants by age group: $46.4 \%, 92.6 \%$, $96.1 \%, 98.8 \%, 98.1 \%$, and $97.3 \%$ among ages $14-19,20-24,25-29,30-39,40-49$, and $50-$ 59 years, respectively. Abbreviation: MEC, mobile examination center.


Figure 2.
Prevalences of any human papillomavirus (HPV) and high-risk HPV (HR-HPV) by sex, age, and race/ethnicity among sexually experienced 14- to 59-year-olds, United States, 20132014. $A$, Overall. $B$, Non-Hispanic whites. $C$, Non-Hispanic blacks. $D$, Hispanics. Relative standard error $>30 \%$ : any HPV, Hispanic males aged $14-19$ years; HR-HPV, non-Hispanic black females aged 25-29 and 40-49 years, Hispanic males aged 14-19 years.

| Characteristic | Sample Size |  | Any HPV ${ }^{\text {a }}$ |  |  |  | High-Risk HPV ${ }^{\text {b }}$ |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  |  | Weighted Prevalence \% (95\% CI) |  | PR (95\% CI) | aPR (95\% CI) | Weighted Prevalence \% (95\% CI) |  | PR (95\% CI) | aPR (95\% CI) |
|  | Males | Females | Males | Females |  |  | Males | Females |  |  |
| Total | 1556 | 1695 | 45.8 (41.9-49.7) | 40.1 (36.1-44.2) | 1.14 (1.03-1.27)* | $0.99(.89-1.10)^{c}$ | 25.7 (23.4-28.1) | 20.7 (17.8-23.9) | 1.24 (1.07-1.43)* | $1.03(.88-1.21)^{c}$ |
| Age, y |  |  |  |  |  |  |  |  |  |  |
| 14-19 | 174 | 187 | 22.5 (17.9-27.9) | 45.5 (38.9-52.2) | 0.49 (.37-0.66) * | 0.47 I.35-.64) ${ }^{\text {c, * }}$ | 15.5 (11.3-20.8) | 29.4 (23.5-36.1) | 0.53 (.37-75) * | $0.51(.36-71)^{\text {c, * }}$ |
| 20-24 | 173 | 179 | 41.8 (34.9-49.1) | 62.8 (53.7-71.1) | 0.67 (.55-0.81)* | $0.63(.54-.74)^{c}$, * | 28.2 (23.2-33.7) | 39.2 (28.2-51.6) | 0.72 (.49-1.05) | 0.67 (.47-95) ${ }^{\text {c,* }}{ }^{\text {* }}$ |
| 25-29 | 158 | 166 | 51.9 (42.9-60.8) | 44.8 (36.5-53.5) | 1.16 (.91-1.47) | $1.06(.84-1.34)^{c}$ | 33.0 (25.7-41.2) | 25.3 (19.3-32.4) | 1.30 (.88-1.94) | $1.21(.82-1.79)^{c}$ |
| 30-39 | 371 | 383 | 45.3 (39.4-51.3) | 38.9 (30.9-475) | 1.16 (.96-1.41) | $0.98(.81-1.19)^{c}$ | 25.2 (22.1-28.6) | 19.2 (15.4-23.6) | 1.31 (1.03-1.68)* | $1.04(.84-1.30)^{c}$ |
| 40-19 | 336 | 401 | 47.0 (40.6-53.4) | 35.1 (30.2-40.3) | 1.34 (1.13-1.58)* | 1.13 (.96-1.33) ${ }^{c}$ | 22.6 (17.2-29.1) | 15.4 (11.2-20.8) | 1.47 (1.11-1.94)* | 1.13 (.86-1.49) ${ }^{\text {c }}$ |
| 50-59 | 344 | 379 | 50.2 (41.1-59.3) | 32.0 (25.2-39.6) | 1.57 (1.15-2.15)* | $1.27(.90-1.79)^{c}$ | 26.9 (21.8-32.5) | 14.3 (10.6-19.1) | 1.88 (1.32-2.66)* | 1.43 (.91-2.24) ${ }^{\text {c }}$ |
| Race/ethnicity |  |  |  |  |  |  |  |  |  |  |
| Non-Hispanic white | 662 | 702 | 44.2 (38.7-49.9) | 36.7 (31.7-42.1) | 1.20 (1.04-1.39)* | 1.06 (.91-1.23) ${ }^{\text {c }}$ | 25.1 (21.9-28.7) | 19.4 (15.5-24.0) | $1.29(1.04-1.61)^{*}$ | $1.08(.86-1.36){ }^{d}$ |
| Non-Hispanic black | 353 | 359 | 62.2 (56.9-67.3) | 65.4 (59.3-71.1) | 0.95 (.86-1.05) | $0.87(.81-.94)^{\text {c }}$,* ${ }^{*}$ | 38.5 (33.9-43.4) | 28.1 (23.8-32.8) | 1.37 (1.14-1.65)* | $1.32(1.10-1.57)^{d, *}$ |
| Hispanic | 384 | 462 | 44.9 (37.3-52.7) | 37.9 (33.3-42.7) | 1.18 (.95-1.48) | $0.95(.76-1.18){ }^{c}$ | 21.9 (17.7-26.8) | 21.7 (17.7-26.4) | 1.01 (.73-1.39) | $0.81(.58-1.12)^{d}$ |
| Non-Hispanic Asian | 157 | 172 | 27.5 (19.9-36.8) | 26.4 (19.6-34.4) | 1.04 (.71-1.53) | $0.97(.66-1.42)^{c}$ | 13.5 (8.4-21.0) | 14.4 (9.5-21.3) | 0.94 (.52-1.69) | $0.71(.35-1.43){ }^{d}$ |
| PIR |  |  |  |  |  |  |  |  |  |  |
| <1 | 349 | 414 | 54.8 (47.8-61.6) | 50.8 (46.3-55.3) | 1.08 (.92-1.27) | $0.92(.79-1.08)^{c}$ | 36.6 (30.7-42.9) | 26.3 (22.7-30.3) | 1.39 (1.12-1.73)* | $1.18(.94-1.48)^{c}$ |
| $\geq 1$ | 1097 | 1170 | 44.9 (40.1-49.7) | 38.0 (34.0-42.2) | $1.18(1.03-1.35)^{*}$ | $1.03(.90-1.18)^{c}$ | 24.2 (21.3-27.4) | 19.6 (16.5-23.1) | 1.23 (1.01-1.51)* | $1.04(.84-1.29)^{c}$ |
| Total lifetime sex partners. No. |  |  |  |  |  |  |  |  |  |  |
| 1 | 213 | 341 | 10.2 (6.2-16.3) | 12.6 (8.9-17.6) | 0.81 (.44-1.48) | 1.05 (.62-1.76) ${ }^{e}$ | $3.9(1.9-8.1)^{f}$ | 6.9 (4.4-10.7) | 0.57 (.22-1.48) | $0.58(.20-1.72){ }^{g}$ |
| 2-5 | 445 | 671 | 31.1 (25.3-37.6) | 36.1 (31.0-41.6) | 0.86 (.71-1.04) | $0.91(.75-1.12){ }^{e}$ | 14.9 (10.7-20.4) | 18.7 (15.0-23.0) | 0.80 (.59-1.08) | $0.78(.56-1.10){ }^{g}$ |
| 6-9 | 250 | 273 | 50.6 (42.0-59.2) | 50.0 (43.5-56.6) | 1.01 (.83-1.23) | $1.00(.82-1.22)^{e}$ | 24.5 (17.8-32.8) | 27.1 (21.5-33.4) | 0.91 (.61-1.35) | $0.88(.61-1.28){ }^{g}$ |

All unadjusted and adjusted PRs used females as the reference group.
Abbreviations: aPR, adjusted prevalence ratio; CI , confidence interval; HPV , human papillomavirus; PIR, poverty income ratio; PR , prevalence ratio.

${ }^{b}$ High-risk HPV includes types 16/18/31/33/35/39/45/51/XR(52)/56/58/59/66/68
${ }^{c}$ Adjusted for lifetime sex partners.
${ }^{d}$ Adjusted for lifetime sex partners and smoking status.
Adjusted for race/ethnicity and PIR.
$f$ Relative standard error $>30 \%$
${ }^{g}$ Adjusted for PIR, age, race/ethnicity, and smoking status.
Adjusted for lifetime sex partners, age, and PIR.


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    ## Supplementary Data

    Supplementary materials are available at The Journal of Infectious Diseases online. Consisting of data provided by the authors to benefit the reader, the posted materials are not copyedited and are the sole responsibility of the authors, so questions or comments should be addressed to the corresponding author.
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